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CONCEPTUAL UNDERSTANDING OF COMPLEX BIOMEDICAL CONCEPTS: CARDIAC OUTPUT AND ITS REGULATION

by

David R. Kaufman

A Thesis submitted to the Faculty of Graduate Studies and Research as Partial Fulfillment of the Requirements for the Degree of Doctor of Philosophy

Department of Educational Psychology
and
Centre For Medical Education, Faculty of Medicine
McGill University, Montréal.

October, 1992

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ABSTRACT

The application of scientific principles in diverse science domains is widely regarded as a hallmark of expertise. However, the role of basic science knowledge and principles in clinical medicine is the subject of considerable controversy. The study examined the understanding of complex biomedical concepts related to cardiovascular physiology. Subjects at various of levels of expertise were presented with questions and problems pertaining to the concepts of *cardiac output* and *venous return*. The experiment employed the combined methods of a cognitive science approach to problem-solving with a focused clinical interview approach common in science education research.

The results indicated a progression of conceptual models of the circulatory system as a function of expertise. This was evident in subjects' explanations and applications of these concepts. The study also characterized the etiology of significant misconceptions and biases, evident in subjects' reasoning at each level of expertise.

This research has implications for a theoretical model of conceptual understanding in complex science domains, as well as, implications for medical instruction. It is proposed that the development of a clinically-relevant epistemology of basic science knowledge could contribute substantially to a revised medical curriculum that could impart a more robust conceptual understanding of biomedical concepts to medical students.

Résumé

Une des caractéristiques essentielles de l'expert dans un domaine scientifique est l'utilisation appropriée de principes propres à ce domaine. Il existe toutefois une controverse quant au rôle des théories et principes en médecine clinique. Cette étude porte sur la compréhension de concepts relatives à la physiologie cardio-vasculaire. Des questions et problèmes portant sur le débit cardiaque et le retour veineux ont été présentés à des sujets choisis selon leurs différents niveaux d'expertise dans ce domaine. Les méthodes utilisées en sciences cognitives pour l'étude de la resolution de problèmes ont été combinées à une méthode d'interview clinique couramment utilisée dans les recherches sur l'enseignement des sciences.

Les résultats démontrent une progression des modèles conceptuels qu'ont les sujets du système circulatoire en fonction de leur expertise. Une analyse des explications des experts et de leur utilisation de ces concepts en a fourni plusieurs indications. De plus, l'analyse des raisonnements des sujets révèle l'étiologie d'erreurs de compréhension notables ainsi que de certains biais.

Les implications de cette recherche portent sur une théorie de la compétence conceptuelle dans les domaines scientifiques complexes, ainsi que sur l'éducation médicale. Il est suggéré que l'élaboration d'une épistémologie des connaissances scientifiques plus pertinente à la pratique clinique serait à considérer lors d'une révision d'un programme d'étude médicale, dans le but de permettre une meilleure compréhension conceptuelle des concepts médicaux.

PUBLICATIONS AND PRESENTATIONS

Parts of the work presented in this thesis have been reported in the following:

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Presentations

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CHAPTER ONE

INTRODUCTION

The present research is concerned with the issue of conceptual understanding of complex biomedical concepts and progressions in mental models as a function of expertise. The role of biomedical knowledge in clinical medicine is a subject of considerable debate. Biomedical knowledge is believed to provide a framework upon which clinical knowledge can be developed. However, its precise role in medical reasoning remains controversial (Patel, Evans, & Groen, 1990).

There are three interrelated issues in characterizing the role of basic science knowledge in clinical medicine. The first issue is concerned with how students and physicians understand biomedical concepts and how this understanding varies from an optimal model of understanding. The second issue pertains to the correspondences between basic science knowledge and clinical knowledge. The third concern is the functional utility of this knowledge in clinical practice. This research is concerned with the first issue, that is the conceptual understanding of biomedical concepts in relation to cardiovascular physiology.

The first three chapters constitute the review of the literature. The first chapter discusses the role of basic science knowledge in medicine. The first part of the chapter examines the evolving role of basic science in medical education and the ensuing difficulties that have arisen in recent years. The second section reviews psychological studies investigating the role of

biomedical knowledge in clinical reasoning, and the final part of the chapter considers epistemological issues related to the utility of biomedical knowledge in clinical medicine.

Chapter three presents a review of literature pertaining to the understanding of scientific concepts. There is an extensive body of literature documenting the formidable difficulties students have in mastering concepts in scientific domains. The primary emphasis of this review is on conceptual understanding and investigations of differences in knowledge structures as characterized by researchers in cognitive science and science education. This chapter covers a wide range of empirical research and theoretical frameworks related to the conceptual understanding of science concepts, particularly in the physical sciences.

Chapter Four discusses empirical and theoretical issues related to the understanding of biological and biomedical concepts. Research findings and theories from these investigations are then compared with the results of studies in the physical sciences with specific reference to the issues of expertise, problem representation, analogical transfer, and misconceptions.

The mechanics of cardiovascular and circulatory physiology comprises the content domain for this research. There are two superordinate concepts, cardiac output and venous return, which are the focal points of the study. *Cardiac output* is defined as the total amount of blood pumped from the heart per unit time. *Venous return* is the amount of blood returning to the heart per unit time. Chapter Five presents a review of some of the pertinent concepts in this domain.

The purpose of Chapter Six is to provide a theoretical and methodological rationale for the study and to synthesize the issues raised in prior chapters in the contexts of the goals of this research. The theoretical rationale is motivated by an attempt to develop a framework for conceptual understanding. The methodological section discusses the issues pertinent to implementing this framework for studying conceptual understanding in the content domain.

Chapter Seven describes the methodology used in the study. The contents include: a discussion of the selection of subjects, the stimulus materials used in the study; the procedure for the experiment; the methods of analysis; and the research hypotheses. The subjects range along the continuum of expertise, from an undergraduate student to medical students, at each level of training, to resident physicians, to expert cardiologists. The materials for study the consisted of 49 stimulus questions and problems presented on cue cards. The experiment combined the methods of a problem-solving approach with a focused clinical interview approach, common in science education.

Chapter Eight presents the results of the study together with a discussion of the findings. The chapter is divided into the four sections of the study. The results are first presented in a tabular form for groups of questions in each section, which reflects the coding of subjects' response for the correct answer, as well as, for original content. Following an evaluation of the overall pattern of responses, there is a discussion of the effects of expertise on conceptual understanding with regards to the specific content matter under consideration. This is followed by an evaluation of selected

individual's responses to a single question or to sets of questions. The purpose of the analyses is to characterize misconceptions, errors in problem representation or analysis, the spontaneous use of analogies, and strategies for synthesizing information.

Chapter Nine presents a general discussion and summary of the results within the context of the issues discussed in previous chapters. The chapter subsequently presents a discussion on the limitations of the study. The last two sections evaluate the potential for further related research and examine the instructional implications of this study.

CHAPTER TWO

THE ROLE OF BASIC SCIENCE KNOWLEDGE IN MEDICINE

The role of basic science knowledge is a subject of considerable debate in medical education. It is generally accepted that basic science or biomedical knowledge provides a foundation upon which clinical knowledge can be built. However, its precise role in medical reasoning is controversial (Clancey, 1988; Patel, Evans, & Groen, 1989a). Biomedical knowledge has undergone a dramatic transformation over the past couple of decades. This has presented unique and formidable challenges to medical education. There is presently considerable uncertainty concerning the relationship between basic science conceptual knowledge of subject matter and the practice of physicians (Dawson-Saunders, Feltovich, Coulson, & Steward, 1990).

The purpose of this chapter is to characterize the functional role of basic science knowledge in clinical contexts. The first section discusses the evolving role of basic science in medical education and the ensuing difficulties. Then I review research related to understanding the use of biomedical knowledge in clinical reasoning. The final part of the chapter deals with epistemological issues related to the functional role of biomedical knowledge from different perspectives.

Basic Science Learning in Medical Education

In the early part of this century, the basic structure of medical curricula took shape. Perhaps the most significant event in shaping the contemporary medical school was a document known as the Flexner Report, published in 1910 (Barzansky, 1992). In 1909, Abraham Flexner was commissioned by the American Medical Association and The Carnegie

Foundation for the Advancement of Teaching to study the state of medical schools in North America (Hudson, 1992). His report documented the grossly inadequate state of most medical schools in the United States and Canada.

The Flexner report included specific recommendations and objectives regarding the role of basic science education in medicine. The objectives included: 1) a partitioning of the curriculum into basic science and clinical science years of education. The basic science core subjects included anatomy, physiology, pathology, and bacteriology. In addition, Flexner recognized that these subjects were second level sciences and that students entering medical school should have backgrounds in biology, chemistry and physics (Hudson, 1992). The report further suggested that the basic sciences should be taught by basic scientists, individuals with Ph.D. degrees, rather than by physicians. The Flexner model emphasized the importance of training in the "scientific method" supported by active student learning and ample use of the laboratory in all disciplines (Barzansky, 1992). However, Flexner recognized the interdependence of the basic and clinical sciences and stressed that basic science teaching should be oriented towards the professional goals of medical practice (Neame, 1984).

The Flexner report was endorsed by the American Medical Association and its recommendations were widely adopted by most medical schools. While the specific contents of basic science courses have changed dramatically, the subjects taught have changed very little in most medical schools from the Flexner era until the present (Barzansky, 1992). The preclinical or basic science phase of the medical curricula is quite consistent between medical schools (Neame, 1984).

In recent years, there has been a dramatic increase in knowledge in cellular and molecular biology and the ensuing technologies have

transformed the practice of medicine (Friedman & Purcell, 1983). New diagnostic and therapeutic tools have enormously increased the ability of doctors to detect disease and change its course (Tosteson, 1990). There is every indication that the trend is accelerating and will require that physicians master an ever more specialized body of knowledge. An additional complicating factor is that medical schools have come to recognize the importance of teaching courses in the behavioural sciences and in bioethics (Barzansky, 1992). These courses, while undoubtedly of considerable importance, compete for time with traditional basic sciences courses during the two preclinical years of medical school.

Medical schools have typically responded by adding the new content to existing courses, increasing the number of classroom lectures and assigning more textbook readings (Stritter & Mattern, 1983). This has resulted in a dramatic decrease in laboratory time and small group teaching during the preclinical years. The basic science courses are increasingly taught by Ph.D. research scientists from diverse departments (e.g., anatomy) with minimal background in clinical medicine. There is also a lack of coordination between the different basic science departments affiliated with the medical schools.

There have been increasing expressions of dissatisfaction with basic science teaching in medicine. Neame (1984) suggests that substantial parts of the basic science medical courses are irrelevant to the future needs of practitioners, and the concepts are presented at a time when students are not prepared to grasp their significance. Furthermore, the presentation of information encourages passivity and rote learning. This inhibits the development of understanding. Neame argues that preclinical courses fail to achieve their objectives of imparting useful and relevant knowledge to the future clinical practitioner. In addition, the primary student evaluations are

multiple choice examinations that emphasize recall of factual information rather than conceptual understanding and integration of concepts.

Medical students have also expressed their antipathy towards the current methods of teaching basic science knowledge. Eichna (1983) claims that medical school fosters a negative attitude towards biological sciences. The courses taught in the preclinical years are of secondary importance and they are to be "endured" before getting to "real medicine", the clinical years. The focus is on amassing facts acquired from lectures and textbooks. There are very few opportunities for students to engage in any meaningful problem solving. This problem is exacerbated by the fact that, unlike other science textbooks, most biomedical textbooks do not include any problem-solving exercises at the end of chapters.

The state of basic science curriculum has led some to question its utility. Cavazos (1984) expresses this point of view very clearly:

> Medical education should not be designed to develop scientists nor students who are encyclopedias of scientific trivia, no matter how vital that trivia might be in pursuit of pure science. We have no need to teach medical students vast quantities of information which results in memorization when such information can be computer-stored and retrieved in seconds. We do have a need to graduate ethical and compassionate students with high level skills in data analysis and independent critical thinking (Cavazos, 1984, P. 763).

This quote underscores a particular cynicism towards the teaching of basic science knowledge and the view that the training of medical scientists and humane medical practitioners are competing goals. This radical proposal offered by Cavazos, which very likely captures the sentiment of many medical educators, demonstrates a significant lack of understanding about the nature of knowledge acquisition. The view expressed equates understanding with the accumulation of facts and since computers can store facts better than humans can, why not take advantage of it. He fails to appreciate that storing

information is not the same as structuring useful and accessible knowledge (Cruess, Patel, & Groen, 1984). Even if a practitioner could access information that effortlessly, it would be of relatively little value if he or she did not have some prior knowledge to interpret this information. It seems unlikely that ethical, compassionate, and highly skilled technicians would be a suitable replacement for today's clinical practitioners.

A more constructive response to the increase in information has been to increase the emphasis on teaching the skills of knowledge acquisition and self-directed learning (Dawson-Saunders et al., 1990). In fact, many medical schools have changed orientations from the traditional format towards a problem-based curriculum. Problem-based approaches attempt to resolve the basic science problem by developing fully integrated curricula (Patel, Evans, & Groen, 1989b). In this approach, students are encouraged to acquire all relevant knowledge while working on clinical problems (Barrows & Tamblyn, 1980). The premise is that the problem serves as a stimulus for learning. This allows the student to resolve the problem, and in the process learn the related facts, principles, or procedures in important areas of basic or clinical science. However, there is little evidence at present to support the contention that the problem-based learning approach has resulted in significant gains in clinical competency (Schmidt, Dauphinee, & Patel, 1990).

Research in Medical Problem-Solving

In this section, I review some of the pertinent research in medical problem solving. The focus is on research that addresses the role of basic science knowledge in clinical medicine. Studies in medical problem solving encompass different domains of knowledge (e.g., cardiology and radiology) and a wide range of performance tasks.

Lesgold, Rubinson, Feltovich, Glaser, Klopfer, and Wang (1988) investigated the abilities of radiologists, at different levels of training and expertise, to interpret chest x-ray pictures and provide a diagnosis. The results revealed that the experts were able to initially detect a general pattern of disease. This resulted in a gross anatomical localization and served to constrain the possible interpretations. Novices had greater difficulty focusing in on the important structures and were more likely to maintain inappropriate interpretations despite discrepant findings in the patient history. The authors concluded that the knowledge that underlies expertise in radiology includes the mental representation of anatomy, a theory of anatomical perturbation, and the constructive capacity to transform the visual image into a three-dimensional representation. The less expert subjects have greater difficulty in building and maintaining a rich anatomical representation of the patient.

Norman, Rosenthal, Brooks, and Muzzin (1989) compared subjects' performance at various levels of expertise in tasks that required them to diagnose and sort dermatologic slides according to the type of skin lesion present. The results indicated that experts were more accurate in their diagnoses and took significantly less time to respond than did novices. The two groups used different kinds of categories in sorting the slides. Expert dermatologists grouped the slides into superordinate categories, for example "viral infections", which reflected the underlying pathophysiological structure. Novices tended to classify lesions according to their surface features, for example "scaly lesions". The implication is that experts' knowledge is organized around domain principles, which facilitate the rapid recognition of significant problem features.

In medicine, the pathophysiological explanation task has been used to examine clinical reasoning (Feltovich & Barrows, 1984; Patel & Groen, 1986). Pathophysiology refers to the physiology of disordered function. This task requires subjects to explain the causal pattern underlying a set of clinical symptoms. Protocols from this task can be used to investigate the ability of clinicians to apply basic science concepts in diagnosing a clinical problem

Patel and members of her research team have conducted a series of studies investigating the role of basic science knowledge in clinical reasoning, using the pathophysiological explanation task. Patel and Groen (1986) investigated expert cardiologists' ability to diagnose and explain the underlying pathophysiology of a difficult patient problem, acute bacterial endocarditis. The results indicated that the pathophysiological protocols of the expert physicians who accurately diagnosed the case could be accounted for in terms of a forward-reasoning strategy that involved moving from propositions in the stimulus text to conditions that suggested a component of the diagnosis. The explanations consisted primarily of top-level clinical rules yielding a correct diagnosis. This is in contrast to the expert physicians who misdiagnosed the case; they tended to introduce many more intermediate-level, basic science inferences into their explanations.

Patel, Groen, and Scott (1988) presented medical students, at different levels of training, with basic science text material and subsequently asked them to integrate it into their explanations of the underlying pathophysiology of a clinical problem. The results indicated that the basic science information was used sparingly by most subjects. When basic science facts were introduced, they generally resulted in inappropriate clinical inferences.

In a related study, Patel, Evans, and Kaufman (1990) reversed the procedure described above by presenting the clinical case first and

subsequently presenting the basic science text materials. The results indicated a substantially greater use of basic science information than was demonstrated in the previous study. The results can be best characterized by a two-stage model of the diagnostic process. The first stage involves the induction of hypotheses from the data, and is referred to as *data-driven reasoning*. The second stage involves the generation of inferences driven by hypotheses, and is referred to as *predictive reasoning*. The results indicated that, with the exception of the final year students, the use of basic science information interfered with data-driven reasoning. However, it tended to facilitate the predictive reasoning of students across levels. The authors proposed that a sound knowledge of the taxonomic disease classification system is necessary before students can make accurate use of basic science information at both stages of the reasoning process during medical problem solving.

The overall results of the preceding studies indicate that an integrated clinical and basic science curriculum, such as that used in problem-based medical school curricula, may enhance the appropriate use of basic science information in clinical reasoning. Patel, Groen, and Norman (1992; in press) attempted to replicate these studies in a medical school that used a problem-based curriculum. The results indicated that the primary difference between the students from the conventional curriculum (those discussed in the previous two studies) and students from the problem-based curriculum is that the students from the problem-based school generated many more inferences from basic science information than did the students from the conventional curriculum. However, in doing so, these students produced many more errors in reasoning and this diminished their ability to accurately diagnose the problem.

Epistemological Issues

Medical knowledge consists of two types of knowledge: clinical knowledge, including knowledge of disease entities and associated findings; and basic science knowledge, incorporating subject matter such as biochemistry, anatomy, and physiology. Basic science or biomedical knowledge is supposed to provide a scientific foundation for clinical reasoning.

It has been widely accepted that biomedical and clinical knowledge could be seamlessly integrated into a coherent knowledge structure that supported all cognitive aspects of medical practice, such as diagnostic and therapeutic reasoning. This notion is exemplified in an influential theoretical paper by Feinstein (1973). He proposed an elaborate theory of clinical reasoning as a logical process. Diagnostic reasoning is described as a process of passing through a series of *explanatory stations* during which the input data of a patient's manifestations are converted to the output, a diagnosis of a particular disease. The sequence begins with the determination that the patient has a manifestation for which an explanation is to be sought. The manifestation is then referred to a *domain*. A clinical domain is a portion of the body that is the structural or functional source of the manifestation. A domain may refer to an organ, region, channel or physiological system of the body. The next step is to further refine the description of the symptomatology upon which a *disorder* can be identified. A disorder is defined as a gross abnormality in structure or function. Once a disorder is identified the search continues with the physician seeking confirmatory evidence and exploring further the exact etiology and underlying pathophysiology of the disease process.

From this perspective, clinical and biomedical knowledge becomes intricately intertwined, providing medical practice with a sound scientific basis. The goal of diagnosis, as stated by Feinstein, is to find the structural fault in the system. As discussed previously, these assumptions have been called into question by medical educators (Neame, 1984), researchers in medical problem-solving (Patel, Evans, & Groen, 1989a) and in medical artificial intelligence (Clancey, 1988). In the following section, we examine some of epistemic and pragmatic constraints that elucidate the inherent difficulty in viewing biomedical and clinical knowledge as a unitary structure.

The two primary purposes of medical problem solving are diagnosis and, therapeutics and patient management. Diagnosis is the aspect of clinical practice that has received the most attention in medical artificial intelligence (Clancey & Shortliffe, 1984). Medical problems can be characterized as ill-structured, in the sense that the initial states, the definite goal state, and the necessary constraints are unknown at the beginning of the problem-solving process. In a diagnostic situation, the problem space of potential findings and associated diagnoses is enormous. The problem space becomes defined through the imposition of a set of plausible constraints that facilitate the application of specific decision strategies (Pople, 1982). Plausible constraints are produced, for example, by narrowing the range of possible diagnostic solutions by evoking categories of disorders (e.g., cardiovascular problems) or through the elimination of classes of problems. Diagnostic reasoning has been characterized as a process of heuristic classification involving the instantiation of specific slots in a disease schema (Clancey, 1988). As expertise develops, the disease knowledge of a clinician becomes more dependent on clinical experience, clinical problem-

solving is increasingly guided by the use of exemplars and analogy, and less dependent on a functional understanding of the system in question.

Biomedical knowledge is of a qualitatively different nature, embodying elements of causal mechanisms and characterizing patterns of perturbation in function and structure. Schaffner (1986) characterizes biomedical science as a *series of overlapping interlevel temporal* models.[1] Temporal models include collection of entities that undergo a process of change and can be represented as a sequence of events. In the physical sciences, time is usually embodied in differential equations. The explicit temporal sequence is of considerably greater significance in biomedical theories (Schaffner, 1980). The term interlevel refers to the fact that entities grouped within a biomedical theory are at different levels of aggregation. An entity e_2 is at a higher level of aggregation than entity e_1, if e_2 has e_1 as some of its parts, and the defining properties of e_2 are not simple sums of e_1 but require additional organizing relations (Schaffner, 1980).

Blois (1990) discusses the practical implications of the interlevel structure of biomedical knowledge. He characterizes task of medical diagnosis as one involving vertical reasoning. Medicine draws upon different sources of knowledge from the biomedical and to a lesser degree the physical sciences. This knowledge can be arranged in a hierarchical schema of the scientific sources. At the bottom is atomic physics, where matter is described with reference to atoms and their constituent properties (Blois, 1990). At each higher level in the hierarchy, there are newly emergent properties not entirely predictable from lower levels. Each new level has different

[1] Not all biomedical disciplines can be characterized by having explicitly causal or temporal components. In particular, anatomy and histology are predominantly concerned with aspects of structure.

conceptual entities and a unique language of description. Higher levels introduce more uncertainty and a greater degree of inexactness in ascribing causality. At the clinical level, models of disease are commonly described in terms of associations between clinical findings and diagnoses.

The problem in medical reasoning arises from an uncertainty of how to combine all observational data (Blois, 1990). The very nature of the inference changes substantially at each level. This is illustrated by Wilson's disease (Table 1), which is a central nervous system disorder caused by a metabolic defect in which the body cannot properly eliminate copper from the blood. (Summarized from Blois, 1990, p849).

Table 1

Attributes of Wilson's Disease at Different Levels in the Hierarchy

Level	Disease Attribute	Field
Patient As A Whole	Malaise, bizarre behaviour, labile affect	Clinical Medicine
Physiologic Systems	Intention tremor, dysarthia, chorea	Physiology
Organs	Kayser-Fleischer ring, ascites	Physiology
Cells	Alzheimer type II cells, abnormal glycogen deposits, necrosis of neurons	Physiology, pathology
Biopolymers	Decreased serum ceruplasmin, increased alkaline phosphatase	Biochemistry
Molecules	Aminoaciduria	Chemistry
Atoms	Decreased Serum copper, increased urinary copper	Physics

The lower-level abnormalities are revealed by laboratory tests and the higher-level attributes come from patients' reports and physical examinations. This problem is rather atypical. Few diseases can be traced

across aggregate levels in this manner. However, biomedical research is increasingly building these vertical connections that provide medical science with a deeper understanding of biomedical disorders. The example serves to highlight the challenge of synthesizing information from different levels of aggregation.

A View from Medical Artificial Intelligence

Medical artificial intelligence (AI) is primarily concerned with the construction of AI programs that perform diagnostic or therapeutic tasks (Clancey & Shortliffe, 1984). Medical AI has been one of the most active domains of AI research. The purpose of this brief discussion is to examine some epistemological issues pertaining to the functional role of basic science knowledge in diagnostic reasoning from a medical AI perspective.

The first generation of medical expert systems, such as MYCIN (Buchanan & Shortliffe, 1984) and INTERNIST (Miller, Pople, & Myers, 1984) were based on empirical associations between manifestations and diseases (Ramoni, Stefanelli, Magnani, & Barossi, 1992). They did not explicitly represent any pathophysiological knowledge. MYCIN, an expert system designed to diagnose infectious diseases, is cited as the prototypical first generation expert system (Clancey, 1984). It was an extremely influential program that achieved a level of diagnostic performance comparable to an expert physician, when the problem under consideration was in the system's knowledge base (Buchanan & Shortliffe, 1984). However, it suffered from many of the limitations of first generation medical AI programs. MYCIN was implemented as a heterarchical rule-based production system. All knowledge, including control and strategic knowledge, was encoded as a series of condition-action rules (if-then). Biomedical

knowledge was seldom used and when it was, it was given equal status as clinical findings or diagnostic hypotheses in a rule as an antecedent or consequent.

The problem with most first generation systems was that they were inherently brittle in that they exhibit a sudden performance degradation when the problem at hand was near or beyond the limits of their domain knowledge (Ramoni, et al., in press). In addition, they lacked significant explanatory capabilities that limited their pedagogical utility (Clancey, 1983).

Clancey used the MYCIN knowledge base to develop NEOMYCIN, which had greatly enhanced instructional capabilities, so that it could be used as a front end for an Intelligent Tutoring System. NEOMYCIN was organized into multiple hierarchies, which distinguished findings from hypotheses and added layers of control and strategic knowledge (Clancey, 1988). Most significantly, the inference model or reasoning procedure is completely separate from medical knowledge. Diagnostic reasoning operates upon a "network of stereotypic knowledge of disorders, that is, knowledge derived from experience of diagnosing many cases, not a working model of the human body and how it can be faulted" (Clancey, 1988, p. 346). There is no explicit representation of the underlying pathophysiological knowledge and therefore basic science knowledge plays only an implicit supporting role.

Many medical expert systems have attempted to overcome the brittleness problem by explicitly incorporating knowledge of the underlying pathophysiological mechanisms. Biomedical knowledge can serve different functional roles depending on the goals of the system. Chandrasakeran and colleagues developed a framework for characterizing such systems (Chandrasakeran, Smith, & Sticklen, 1989). In particular they specify two goal types for diagnostic reasoning. The first type of diagnostic reasoning,

D1, has the goal of the identification of that aspect of the system can account for the disturbance in the form of a "malfunction hypothesis", given some observed problem in the behaviour of a system. Most cases of diagnostic reasoning could be subsumed by the first type. The second goal type, D2, has the further goal of identifying the structural fault that has led to the aberrant behaviour. To engage in D1-type reasoning, an agent needs knowledge of the space of possible malfunctions and knowledge that relates observations to malfunctions. To employ D2-type reasoning, in addition to the above knowledge, an understanding is required of how behaviour, structure and function interrelate.

There are several combinations of control and knowledge structures that could be used in diagnostic systems of type D1. Partial pattern matching is an approach used in systems like MYCIN and INTERNIST. This involves relating observations to intermediate hypotheses, which partition the problem space, and further associating intermediate hypotheses with diagnostic hypotheses. The knowledge-base would include only entities related to taxonomic classification; diagnostic hypotheses and clinical findings (Chandrasakeran, et al., 1989). A system such as NEOMYCIN augments this type of approach with an elaborate control structure that focuses problem-solving and establishes a top-down control. The content knowledge remains essentially the same, consisting of diagnoses and clinical findings.

Certain expert systems explicitly encode biomedical knowledge in a multi-level causal network. This approach is exemplified by ABEL, a consultation system for electrolyte and acid-base disorders (Patil, Szolovits & Schwartz, 1984). ABEL attempts to identify the disease process causing a patient's illness. Knowledge is encoded in a hierarchical semantic network

and can explain pathophysiological states in varying degrees of granularity, for example, from clinical levels to specific biochemical processes.

ABEL attempts to account for the clinical findings by developing a multi-level explanation of the problem, known as a patient specific model (Patil, et al., 1984). The program constructs this explanation by navigating between levels via processes such as aggregation (summarizes the description to the next more aggregate level), and elaboration (elaborates the description to the next more detailed level). The pathophysiological description provides the ability to solve complex clinical situations with multiple etiologies and organize large amounts of information into a coherent causal explanation (Patil, et al., 1984).

A system such as ABEL contains only stored compiled causal knowledge. This means that the knowledge can only be retrieved from memory. It cannot be generated and can only be used to solve D1 type diagnostic problems (Chandrasakeran, et al., 1989). This type of system is limited in the type of causal reasoning can exhibit because the causal knowledge does not contain explicit information about the structural and behavioural assumptions that underlie causal links.

Chandrasakeran (Chandrasakeran, et al., 1989) describes a type of system that is referred to as a deep system. This is similar to systems used in qualitative physics (Bobrow, 1985) and embody causal mental models. A system such as MDX-2 (Chandrasakeran, et al., 1989) or QSIM (Kuipers, 1987) has an explicit representation of structural components and their relations, the functions of these components (in essence their purpose), and its relationship to behavioural states. The causal and diagnostic knowledge can be generated by "'running" or simulating the system and qualitatively deriving behavioural sequences that can identify and explain the

malfunction. The knowledge is not precompiled as in the previously described systems, but can be generated in real-time to find fault in a system. This principled knowledge could theoretically be used to generate the widest range of possible diagnostic hypotheses and explain multi-system conditions that the program had never previously encountered.

Summary

This chapter dealt with a range of issues concerning the role of basic science knowledge in diagnostic reasoning. The first section discussed the evolving role of basic science teaching in medical school. The teaching of basic science subjects has changed relatively little in the past eighty years, in the sense that it is taught in the first two years of medical school and mostly in a didactic lecture format. The possible exception are the medical schools that have adapted a problem-based learning curricula. The content has changed dramatically as has the practice of clinical medicine. This has led to increasing expressions of concern of whether medical education provides the appropriate opportunities for students to engage in meaningful learning of biomedical knowledge. It is questionable whether students acquire knowledge that is accessible and useful to them in their future clinical practices.

Investigations of medical problem solving provide some evidence to support the contention that biomedical knowledge is not used optimally in clinical contexts. The research findings suggest that basic science is used differentially in different tasks and in different medical domains; experts and novices differ in their use of basic science and that, in many instances, basic science knowledge may actually interfere with clinical problem-solving. The evidence also suggests that students possess substantial inert knowledge that

frustrates their ability to apply specific biomedical concepts to clinical problem-solving tasks. In addition, the problems appear to be at least equally pervasive in problem-based medical schools with integrated curricula. The results also suggest that when used appropriately biomedical knowledge can facilitate explanation. It may also be possible that in certain domains, particularly those that emphasize perceptual skills (e.g., radiology), basic science knowledge may play a role in the initial stages of problem representation or hypothesis formulation. In other domains, biomedical knowledge may be used most effectively to distinguish between competing hypotheses in the latter stages of the diagnostic reasoning process.

The final section of the chapter addresses epistemological issues related to the functional role of basic science knowledge in clinical medicine. At a point in time there seemed to be substantial agreement that basic science knowledge and clinical knowledge could be integrated into a single body of knowledge that a practicing physician could access in the course of diagnostic or therapeutic reasoning. This assertion is not supported by empirical evidence. More recent conceptualizations emphasize the hierarchical multi-level nature of biomedical knowledge. Each level consists of a unique ontology, having different conceptual entities[2] and relationships. This would suggest that biomedical knowledge is fundamentally qualitatively different from clinical knowledge.

Medical AI represents a discipline in which particular epistemological positions are expressed in working implementations. While this may not conform to a complete psychological theory, it does provide an opportunity to

[2]Following Greeno (1983), a conceptual entity refers to a cognitive object that the system can reason about in a direct way. That is if the object can be taken as argument in a proposition. This distinguishes entities from attributes and relations.

consider the different functions of biomedical knowledge in diagnostic reasoning as expressed in these systems.

There are three major perspectives on the functional role of basic science knowledge. Each of the perspectives is associated with a particular implementation approach. In the first approach, diagnostic reasoning is viewed as a process of classification, in which clinical findings are explained via their association with particular diagnostic hypotheses. Basic science knowledge is used implicitly or plays a relatively minor support role. This type of reasoning could be characteristic of diagnostic reasoning in most routine situations.

The second approach incorporates an explicit encoding of multilevel causal biomedical knowledge. This knowledge is stored, precompiled knowledge that could be easily accessed when the situation demands a more detailed causal explanation. This is analogous to a physician having access to a complex hierarchical network of cause and effect relationships in their knowledge base. The physician could retrieve information at different levels of aggregation depending on the complexity of the problem and the nature of the evidence under consideration (laboratory findings may require a different level of explanation than would a clinical finding). This type of knowledge can be used to resolve impasses by abstracting from potentially unobservable states to observable states and provide causal explanations (Chandrasakeran, 1989). However, if a correspondence between an observed state and the stored causal knowledge could not be established, the individual would have no way of generating a diagnostic hypotheses.

The third approach incorporates an explicit representation of structure, function and behaviour. A physician could use this knowledge to explain a patient's condition by running the model and envisioning the

consequences in terms of different behavioural outcomes. This model embodies the most powerful diagnostic reasoning approach, as well as the most computationally demanding. The completeness and coherency of such a mental model would likely be a function of expertise.

A psychological theory of clinical reasoning may need to embody each of these three models. The role of biomedical knowledge in a model of diagnostic reasoning would likely be determined by three factors: 1) the complexity of a problem; 2) the extent to which a domain can be characterized as a dynamical system; and 3) expertise. A routine problem would not necessitate the use of explicit biomedical knowledge, while a complex multi-system problem would very likely engage such knowledge. Certain domains, such as radiology and dermatology would be more amenable to pattern recognition approach, while others such as nephrology and cardiology may need the support of "deeper" biomedical models. Which model would be most exemplary of subjects at different levels of expertise would probably be a function of the two factors. For example, productive and efficient performance, typified by expert subjects, in a routine problem would involve superior recognition and classification skills rather than deeper models of biomedical knowledge. This discussion is somewhat speculative. Further empirical and computational research is needed to determine the role or roles of biomedical knowledge in clinical reasoning.

CHAPTER THREE

UNDERSTANDING SCIENCE CONCEPTS

There has been a great deal of research into science concept understanding and acquisition over the past fifteen years (Eylon & Linn, 1988). There is an extensive body of literature documenting the formidable difficulties students have in mastering concepts in various domains of science. The primary emphasis of this review is on conceptual understanding and characterizations of differences in knowledge structures rather than on problem solving or reasoning strategies. This chapter covers a wide range of empirical research and theoretical frameworks related to the conceptual understanding of science concepts. The breadth of this review suggests the complexity and multi-dimensional nature of the topic, and its increasing importance in cognitive research. However, it also reflects the fragmentation and lack of communication between research communities that may share common fundamental interests. This chapter includes concept research dealing with subject matter in the physical and to a lesser extent mathematical sciences. The next chapter deals with biological and biomedical concept research.

Cognitive Science and Science Education

In general, there are two communities of researchers who have addressed the issue of science concepts; cognitive scientists and science education researchers. These groups approach the subject matter from somewhat different perspectives.

Cognitive science research has predominantly focused on contrasting the behaviour of expert and novice subjects in problem-solving tasks and

developing models of competent performance (e.g., Larkin, McDermott, Simon, & Simon, 1980). This research has emerged from the information-processing tradition (Newell & Simon, 1972). The emphasis of these investigations is on characterizing the sequence of overt and cognitive behaviours used by an individual in proceeding from an initial problem state to a goal state. Early investigations focused predominantly on studies of experimentally contrived tasks (e.g., cryptarithmetic) and other "knowledge lean" environments (Greeno & Simon, 1988). The research shifted focus to semantically complex (involving the use of natural language), and knowledge-rich (content domains with an elaborate knowledge-base) domains in the late 1970s. At this point, studies of problem-solving in scientific domains became prominent and the characterization of subjects' knowledge became a focal point in many of these investigations.

The principle focus of science education research has been on the content and structure of students' knowledge prior to and following instruction (e.g., McCloskey, Caramazza, & Green, 1980). Particular emphasis is placed on the students' initial understanding of scientific concepts and how particular patterns of misunderstanding can impede learning.

Research into the acquisition of science concepts is informed by a *constructivist epistemology* (Millar, 1989). This research owes a great intellectual debt to Piaget, in terms of methodology and theoretical models. According to the constructivist position, the learning and growth of understanding involves a learner constructing his or her own private understanding of large bodies of public knowledge. Public knowledge is reflected in the consensually agreed upon bodies of scientific knowledge imparted to students (Pines & West, 1985). Learning entails integrating

new knowledge with pre-existing schemata, and the reconstruction of meaning rather than the mere accumulation of new facts (Millar, 1989).

Science education research has also been strongly influenced by contemporary *post-positivist* philosophers of science (e.g., Kuhn, 1970). In particular, many of the philosophical views were judged to be consistent with the claim that students' conceptions relied on a configuration of beliefs, commitments and expectations in a manner analogous to a community of scientists (Confrey, 1991).

There are methodological differences between the cognitive science and science education approaches. In cognitive science studies of problem solving, the primary method of data acquisition is the "think-aloud protocol" (Ericsson & Simon, 1984). In these studies, subjects are instructed to think-aloud as they perform a particular task. A typical task in studies of scientific problem-solving involves asking subjects to solve a set of textbook problems (e.g., Larkin et al., 1980). The think-aloud protocols can be used to construct computer simulations which provide a measure of sufficiency for a particular theory. The measure of sufficiency requires that an investigator demonstrate that a simulation of subjects' reasoning processes and represented states can, at minimum, produce the same behavioural outcome and reproduce the same pattern of errors (Simon, 1978).

Research in science education has typically focused on investigating dimensions of cognitive structure (White, 1985). These investigations use a wide range of tasks to study subjects' understanding of concepts. One of the most widely used measures is the clinical interview. The clinical interview derives largely from the clinical method of Piaget. This method was used to study children's level of cognitive competence on a wide range of tasks (Ginsburg, Kossan, Schwartz, & Swanson, 1983). Science education research

uses a variation of this approach in which the interviewer presents problems and questions to a subject in a flexible manner. The questions posed may be contingent on the subject's response and the interviewer may attempt to elicit and verify subjects' beliefs about particular relationships. The procedure is much more open-ended than the think-aloud protocol, in which the role of the experimenter is rather circumscribed and dialogue is to be kept at a minimum.

There are a number of other tasks that are used in these types of experiments, many of which have their origins in Piagetian methods. An experimental task that is also used as an instructional device is sometimes referred to as the DOE (demonstrate, observe, explain) technique (Champagne, Gunstone, & Klopfer, 1985). Students are presented with a physical apparatus (e.g., a pendulum) and are provided with an explanation. They are then asked to predict the outcome of the demonstration and to explain the reasons for their predictions.

In recent years, there has been a convergence in empirical approaches and theoretical models, to the point where the distinction between the two research groups is not as conspicuous (Glaser & Bassok, 1989).

Research in Science Concept Understanding

Expertise, Knowledge and Problem-solving Abilities

The study of expertise is one of the principal paradigms in problem-solving research. Comparing experts to novices provides us with the opportunity to explore the aspects of performance that undergo change and result in increased problem-solving skill (Lesgold, 1984). It also permits investigators to develop domain-specific models of competence.

Knowledge-based differences impact on the problem representation and determine the strategies a subject uses to solve a problem. In a series of studies, Larkin, McDermott, Simon, and Simon (1980) compared novice students with expert physicists in solving textbook physics problems. The results indicated that experts solved the problems in one quarter of the time and with fewer errors than novices. The novices solved most of the problems by working *backward* from the unknown problem solution to the givens of the problem statement. The novices seemed to require goals and subgoals to generate a solution strategy. The management of goals and subgoals undoubtedly places a considerable burden on short memory and may occupy considerable time (Larkin, et al., 1980). Experts tended to work *forward* from the givens to solve the necessary equations and to determine the specific quantities they were asked to solve for. This was particularly evident in the easier problems in which the experts could recognize the problem situation from experience and proceed to solve the equations without any deliberate planning (Larkin, et al., 1980).

A consistent theme across studies of the development of expertise has been the role that the evolution of knowledge structures have in facilitating the recognition of significant objects within a problem and enhancing one's ability to recognize typical situations. Many of the differences in the problem-solving performance of experts and novices can be related to the use of qualitatively different problem representations. A problem representation is a cognitive structure associated with a problem, constructed by an individual on the basis of his or her domain-related knowledge and it's organization (Chi, Glaser, & Rees, 1982). A representation may include elements from the problem statement, such as the initial state and the goal

state, and knowledge of legal problem-solving operators and plausible inferences.

Chi, Feltovich, and Glaser (1981) hypothesized that cognitive structures could be examined by asking subjects to sort a set of textbook physics problems into categories that reflect the common properties of the problems. The novice subjects, who had completed one semester of mechanics, tended to group problems according to similarity of surface structure (frequently, the literal physical terms mentioned in the problem), such as "spring" or "inclined plane" problems. In contrast, expert physicists categorized the problems by virtue of underlying principles or fundamental laws, such as "Newton's Second Law of Motion".

A general finding is that experts engage in a qualitative analysis of the problem prior to working with the appropriate equations (Larkin, 1983). Chi and colleagues suggest that this phase involves the early activation of appropriate principle-oriented knowledge structures or schemata. This is supported by the fact that experts took longer than the novices to sort the physics problems. When the schema is tested for appropriateness and confirmed (e.g., the principle was correct), the knowledge in the schema provides the general form that the specific equations to be solved will take (Chi, Glaser & Rees, 1982). The contents of the schemata of experts may not differ dramatically in terms of information content. The expert's schemata are organized hierarchically which facilitates the appropriate problem abstractions. The novice's knowledge tends to be more heterarchical, with different features not organized in such a way for supporting abstracted solution methods.

There have been numerous studies in diverse domains of science that have documented the qualitative differences between expert and novice

subjects in categorizing problem types. These studies have tended to replicate the result that experts classify problems according to domain principles and novices tend to rely on surface structures. Thibodeau, Hardiman, Dufresne, and Mestre (1989) attempted to elucidate the effect of problem categorization criteria on problem-solving ability. In one experiment, subjects were presented with a model problem and two potential matching problems. Expert physicists and novice students were asked to choose which of the comparison problems would be solved most similarly to the model problem. The problems presented to the subjects matched either in the deep structure, the surface structure, both the surface and deep structure, or neither surface nor deep structure. The deep structure referred to the underlying principle and surface structure would include a similar equation type or a literal similarity between objects in the problem statements.

As would be expected experts more frequently chose the comparison problem that matched the deep structure of the model problem more often than the did novices. Nonetheless, the surface features affected even the categorization process of experts. When pairs of problems were presented in which one problem matched the surface structure and the other matched the deep structure, the experts tended to experience some difficulty in making the correct choice.

In a second experiment, novice students who had completed a single mechanics course were asked to determine whether two problems could be solved similarly and, if so, to explain why (Thibodeau, et al., 1989). The students were classified on the basis of their response according to the type of reasoning they most frequently employed, surface feature, principle, or mixed. They were subsequently asked to solve a set of problems. The results

indicated that the students who most frequently used principle as a basis for comparison performed significantly better than the other students. The *mixed students* scored significantly higher than the *surface feature students.* The correlation between categorization and problem-solving ability was highly significant even when mathematics proficiency and other measures of scientific achievement were partialed out. The authors concluded that principles play a fundamental role in the organization of conceptual and procedural knowledge for good problem solvers at all levels.

The Thibodeau study illustrates the way in which themes that emerge from expert-novice studies can be extended to study differences between students of differential abilities. Chi, Bassok, Lewis, Glaser, & Reiman (1989) compared the explanations of good and poor students while attempting to learn from previously worked out textbook physics problems. The results indicated that good students generated almost twice as many self-explanations. The number of physics explanations were correlated with subsequent success in solving the problems. The explanations of good students tended to expand or refine the conditions of an action and to relate the consequences of one action to another. Many of their explanations were guided by an attempt to explicate the principles embodied in the text and to coordinate the principles with their associated procedures. Poor students spent less time studying the worked out examples and generated many fewer self-explanations.

During the transfer session, the poor students devoted considerably more time to rereading the worked out examples in search of a solution procedure. Good students were far more selective in focusing on the specific aspects of the worked out examples that yielded information about the solutions. The authors concluded that good self-explanations help

understanding and problem solving because they lead to the creation of inference rules that are instantiations of principles and definitions introduced in the text (Chi et al., 1989). These inference rules are useful because they better articulate the specific conditions or situations in which an action is to be taken and are consequently more operational during problem-solving than the principles themselves.

Transfer and Learning

Learning and instruction have become active areas of research in cognitive science in recent years (Glaser & Bassok, 1989). The study of transfer has become one of the focal points of learning research. Transfer can be defined as learning of conceptual knowledge and or procedures that can be applied in contexts that are novel to the learner. This would preclude rote learning of procedures or the memorization of passages. Transfer has been the source of investigations in diverse disciplines of science education, experimental psychology and artificial intelligence, covering topics such as analogical transfer (Gick & Holyoak, 1980), the acquisition of cognitive skills (Anderson, 1982; Anzai & Yokohama, 1984), concept induction and categorization (Medin & Ross, 1989; Michalski, 1989), and in scientific reasoning (Clement, 1988).

A common theme emerging from research in these areas is the inherent difficulty that students and individuals have in transferring knowledge across contexts and the failure of instruction to promote robust transferable knowledge (Wittrock, 1985). Salomon and Perkins (1989) have developed a framework for characterizing the mechanisms of different kinds of transfer and the conditions necessary to induce successful transfer. The central issue in transfer is to explain how previously learned elements

(procedures, principles, categories of description) can be evoked and successfully applied in different situations.

Salomon and Perkins (1989) distinguish two primary ways in which transfer occurs. *Low road transfer* depends on extensive and varied practice of cognitive and sensory-motor-skills and occurs by the automatic triggering of well-learned behaviour in new contexts. *High road transfer* occurs by intentional "mindful abstraction" from one context to another. Low road transfer is a function of practicing a skill in varied contexts. These skills encompass a wide range of abilities from performing arithmetic procedures to driving a car. The skill or knowledge element that is successfully transferred is elicited automatically by features or the demands of a situation. For example, an object in the middle of a road seen by the driver of a car at the last moment, elicits a sudden swerve response. The action is automatic and immediate. Automaticity tends to inhibit analytic reflection and efficient low road transfer may in fact impede high road transfer (Salomon & Perkins, 1989).

The distinction between low and high road transfer is closely related to the difference between automatic and controlled processing (Shiffrin & Schneider, 1977). Automatic processes are completed without any conscious control by the subject. Low road transfer occurs via an automatic learning process. Controlled processing requires the subjects conscious attention. High road transfer necessitates a kind of controlled processing.

In this paper, we are principally interested in high road transfer. The defining feature of high road transfer is *mindful abstraction* (Salomon & Perkins, 1989). An abstraction is a representation that is more general and less detailed than another representation. The process of abstraction involves the extraction from or the identification of some generic or basic

category, quality, or pattern of elements (Salomon & Perkins, 1989). Abstraction is not unique to high road transfer. What is unique is that it is a mindful process, meaning it is deliberate, metacognitively guided and effortful.

High road transfer can be distinguished in terms of the relative time point at which the transfer is to be achieved (Salomon & Perkins, 1989). Forward-reaching transfer occurs when one abstracts basic elements in anticipation for later application. Backward-reaching transfer is required when one faces a new situation and deliberately searches for relevant knowledge already acquired.

The distinctions raised by Salomon and Perkins may be used to account for failures in transfer and to prescribe instructional strategies to fulfill particular kinds of learning goals. These authors emphasize that different kinds of transfer have specific prerequisite conditions for learning to occur. However, it is very likely that the demarcation between the high and low road transfer is not as sharp as Salomon and Perkins suggest. There is considerable psychological evidence that abstraction and generalization cannot be viewed as a strictly autonomous and intentional process that strips away surface details and uncovers only the principled relations (Medin & Ross, 1989). Studies suggest that concrete details characteristic of prototypical examples play an important part role in learning and problem-solving.

Analogical transfer has been a very productive area of research in experimental psychology and in science education research. Analogies can be conceived as nonliteral comparisons between superficially dissimilar knowledge domains (Zook, 1991). According to Zook, there are two primary ways in which analogical comparisons may affect learning. Learners may

generate analogies for themselves as they attempt to understand a new situation by relating it to a well-understood familiar situation—a kind of backward reaching transfer. The second way is via formal instruction. Teachers frequently present analogies to explicate novel concepts and/or principles.

There have been numerous empirical studies in experimental psychology that have investigated the determinants of analogical transfer. The primary focus of earlier work was on simple experimental tasks that, for example, presented words or sentences that required judgments of the kind A is to B as C is to D, where D is the term to be identified or chosen from several possibilities (see Sternberg, 1977). These tasks were also commonly used in intelligence testing (Sternberg, 1982).

Holyoak and colleagues were among the first investigators to study analogy in the context of more complex problem-solving situations. Gick and Holyoak (1980) conducted a series of studies using Duncker's (1945) radiation problem. The problem presents a dilemma of a patient with a tumor in his stomach. Radiation of sufficient intensity can be used to destroy the tumor. However, at this intensity the rays will destroy too much healthy tissue. The goal is to find a way to destroy the tumor without destroying too much healthy tissue. The solution is to deliver the radiation from multiple sources.

A typical experiment presented an analogous story such as a military problem (and sometimes its solution) prior to presenting the radiation problem. The primary result is that a relatively low percentage (30%) of subjects spontaneously produced the convergence solution. In contrast, when subjects were provided with an explicit hint that the stories were related, 75% of the subjects produced the correct solution. The authors concluded

that the difficulties in transfer are attributable to failures to spontaneously recognize the potential usefulness of an analog.

The problem of analogy is essentially one of abstracting relational features from the surface features of a source analog and mapping the relations to a seemingly disparate target analog (Gentner, 1983). Gick and Holyoak (1983) explored the possibility that the induction of a problem schema from concrete analogs would facilitate analogical transfer. They attempted several manipulations to induce an abstract schema such as having subjects summarize the problem, and augmenting the story with a principle about the solution statement. The only manipulation that succeeded in substantially facilitating transfer was the presentation of two story analogs.

Holyoak and Koh (1987) attempted to further elucidate factors that determine the success of analogical transfer. They suggested that both surface and structural features common to both the source and target analogs exert considerable influence on transfer. They proposed that retrieval of analogies is based on a summation of activation of multiple shared features. The experiments manipulated the surface and structural similarity. The results indicated that robust spontaneous analogical transfer occurred (even after several days delay) under the conditions in which there was both a surface and structural similarity between source and analogs. However, the structural features exert a more prominent role at the mapping stage, once the relevance of an analog has been pointed out.

Novick (1988a) extended the work of Holyoak and Koh (1987) by comparing analogical transfer along a dimension of expertise.[3] It is well

[3]The use of expert-novice designation is somewhat unusual in this study. Expertise is defined according to subjects' score on the math section of the scholastic aptitude test. Perhaps math ability would be a more appropriate descriptor.

established that experts and novices form qualitatively different problem representations. For example, experts tend to extract structural features of a problem, while novices are more bound to the surface features of a problem. It was therefore likely that surface and structural features could exert differential effects, on subjects who varied in their domain expertise, in analogical transfer tasks. Novick conducted three studies, using mathematical word problems. The results indicated that when two problems share structural features but not surface features, spontaneous positive transfer was more evident in subjects with greater expertise. If the source and target problems shared only surface features, then a negative spontaneous transfer was induced. That is to say, subjects were more inclined to use an incorrect but similar solution procedure than subjects who were not exposed to a potential analog. The negative transfer effect was significantly more pronounced in novice subjects.

Transfer of knowledge is a fundamental goal of education, yet the experimental findings concerning the lack of transfer would seem to undermine that goal (Bassok & Holyoak, 1989). Particular domains like mathematics and logic are taught with the objective of teaching structural relations, and domain content is introduced to exemplify those relations and to demonstrate their conditions of applicability (Bassok, 1989). Bassok and Holyoak (1989) investigated the interdomain transfer of procedures between algebra word problems and physics problems. In particular, the study examined whether subjects would exhibit a transfer of knowledge between a set of arithmetic-progressions problems in algebra and a set of constant-acceleration problems in physics. These two domains are structurally isomorphic. Subjects learned to solve problems in one of the two domains and then were tested on the other domain. The goal was to determine if they

were able to apply the solution method learned in one domain to the other domain.

The results indicated an asymmetrical transfer. Algebra-trained subjects immediately retrieved the relevant equation and applied the solution method without any indication that they were solving novel problems. Physics-trained subjects almost never exhibited any spontaneous transfer to the algebra problems. The authors concluded that students with moderate levels of knowledge of the typical conditions of applicability for mathematical procedures are able to effectively screen out content-specific details of algebra word problems. In contrast, students who are trained to solve physics problems, learn the content specific applicability conditions which precludes transfer to structurally similar domains. The learned physics procedures are embedded in content.

In a follow-up study, Bassok (1991) attempted to further explicate the negative and positive factors that influence transfer in content-rich quantitative domains. She hypothesized that if lack of transfer was merely a function of the content embeddedness of a procedure, then a hint about its relevance should induce subjects to recognize the applicability of the learned procedure. The first experiment was similar to the previously described one, except that banking and finance were used as one of the structurally isomorphic domains to algebra, replacing physics. The surprising finding was that substantial transfer was observed from the banking problems to the algebra problems despite the considerable degree of content embedding.

The second experiment assessed the degree to which specific surface features related to the quantity type of the variable would effect transfer (Bassok, 1991). In particular, *extensive quantities* involve only one entity (e.g., number of potatoes) and *intensive quantities* involve two elements (e.g.,

miles per hour). Physics problems were used again as the other analog domain. The results indicated that there was substantial transfer between problems of matching quantity types from physics to algebra despite the content embedding. There was virtually no transfer observed when the quantity types did not match, replicating the earlier experiments of Bassok and Holyoak (1989). Bassok concluded that the effect of embedding content was limited to the surface masking of structural features due to the addition of distinctive features. Students may be particularly sensitive to semantic features related to the interpretation of a problem's variables. These features that are known to affect the classification of problems may affect the access of appropriate analogs as well as their use in transfer situations. These findings also suggest that the experts in Novick's study (1988a) may have exhibited less negative transfer than the novices because they were less bound by the specific semantic constituents of the problems.

The previously discussed studies of analogy explicitly looked at experimental conditions which attempted to induce analogical transfer by providing subjects with a potential source analog. Several studies have focused on the role of spontaneously generated analogies during the course of problem-solving. Gentner and Gentner (1983) looked at analogical models used by subjects to understand simple electrical circuits. They found that they could identify two analogical models used distinctively by different subjects: the *flowing-fluid model* (water-flowing through a pipe) and the *moving-crowd model* (crowds moving through a corridor). The interesting finding is that the pattern of inferences in the electrical circuit problems could be predicted by the model adopted. In addition, the selection of a particular analog greatly influenced the types of problems subjects could solve.

The central role of analogy in scientific reasoning and in particular scientific discovery, has been well established (Langley, Simon, Bradshaw, & Zytkow, 1987). Clement (1988) evaluated the methods of spontaneously generating analogies by experienced problem solvers from technical domains (e.g., computer scientist) in solving a 'coiled-spring' problem. Clement documented three primary methods of analogy generation. The first method is *generation from a formal principle,* which involves recognizing a situation in which a principle or equation may apply, and retrieving an analogous example of that principle. *Generation via a transformation* is the second observed method. This occurs when a subject creates an analogous situation B by modifying the original situation A and thereby changing one or more features that were invariant features of the original problem. The third method is *analogy by association,* which involves the retrieval from memory of an analogous situation. This latter method is the method most commonly studied in the literature. However, Clement found that the most common method used by subjects was analogy via a transformation.

This result may suggest that a lack of analogical transfer between disparate domains may not only be due to failures in access and mapping. Transfer failures may reflect an inability on the part of the subjects to transform a situation into a form in which the correspondences become transparent. This may explain the lack of transfer in the Bassok and Holyoak (1989) studies.

There have been numerous theoretical models of analogy from diverse disciplines, such as AI (Carbonnel, 1983), linguistics and philosophy (Lakoff, 1987), and psychology (Holyoak & Thagard, 1988). Gentner's (1983) structure-mapping theory is one of the most influential. I will focus on this model, not only for its use in accounting for analogical transfer, but because

of its methodological implications for knowledge representation. Salomon and Perkins (1989) characterized the kinds of transfer that occur and the cognitive mechanisms involved. Gentner is primarily interested in addressing the issue of what elements are mapped during the course of analogical reasoning. Analogy is viewed as a mapping of knowledge from one domain into another domain in which a system of relations that holds among the objects in the base domain also hold among the objects in the target domain (Gentner, 1989). Objects are placed in correspondence by virtue of their role in a common relational structure. In this view analogy can be distinguished from other kinds of comparisons, like surface similarity mappings or mere appearance mappings.

The theory posits a set of rules based on syntactic properties of the knowledge representation independent of the specific content of the domain. The system distinguishes between objects, object-attributes and relations between objects (Gentner, 1983). Knowledge is represented as propositional nodes and predicates. Attributes are predicates that take one argument and relations take two or more arguments. For example, ROUND (ball) is an attribute, while COLLIDE (cue, ball) is a relation. Gentner (1989) also distinguishes between first-order predicates and higher-order predicates. If COLLIDE (cue, ball) and INSIDE (ball, pocket) are first order relations, then CAUSE [COLLIDE (cue, ball), INSIDE (ball, pocket)] is a second-order predicate. Typically higher-order relations involve CAUSE and IMPLIES. The order of an item is an indication of the depth of the structure below it. The representations are intended to reflect the way people interpret a situation rather than what is logically possible. The mapping process is governed by the *principle of systematicity*, which states that individuals are more likely to map connected systems of relations guided by higher-order

relations (Gentner, 1989). The mapping process involves establishing object correspondences, and preserving relations according to the systematicity principle. Finally, object attributes are deleted.

Gentner's structure-mapping theory can be used to explain a wide range of empirical results in analogy research from expert-novice differences to developmental stages to the learning of complex physical models (Forbus & Gentner, 1986). However, the theory has been criticized by Holyoak (1985) for failing to consider the goal-driven aspect of analogy in problem-solving situations. He contends that different goals can lead to different mappings and systematicity is determined by those elements which are pragmatically relevant for goal attainment. In addition, syntactic mappings cannot explain the changes in state that are needed to account for analogy in a problem-solving context.

The debate over the two theories can, in part be, explained by their emphasis on differing aspects of analogy. The structure mapping process accounts for the process of mapping once representations of the source and target analogs have already been constructed. Gentner recognizes that goals play a central role in the construction of the representations and in evaluating the analogy once it has been developed (Gentner, 1989). Holyoak's model builds the analogy mechanism around plans and goals that are generated in the course of problem solving. It is interesting to note that because of the high correlation between goal-related features and higher-order relations, the two approaches will often make the same predictions for a given set of stimuli (Novick, 1988b). While the two competing theories offer radically different computational models of analogy, there is very likely a common underlying element. This may suggest that goals in analogical

problem-solving situations frequently gravitate towards the mapping of particular kinds of syntactic structures.

Conceptions and Misconceptions

Empirical studies of many different domains in science indicate that students begin their study of science with strongly held misconceptions of phenomena (Eylon & Linn, 1988). These misconceptions are grounded in experience and are extremely resistant to change, even after instruction (Driver, 1989). Misconception research has very likely become the most active area of research within the science education community. The construct of misconceptions has appeared in the literature under various other terminological guises such as alternative frameworks, preconceptions, naive theories, and informal knowledge (Confrey, 1991). Pfundt and Duit have compiled a bibliography of over 1500 citations (cited in Confrey, 1991) in science education alone, encompassing more than 600 distinct studies. In recent years, there have been a number of surveys of the literature published. Eylon and Linn (1988) refer to sixteen science topic areas, covering twenty misconception types, and with subject populations ranging from very young children (age 6 to 8) to adults.

Many studies of misconceptions tend to share a common experimental approach. Students are asked to solve sets of problems exemplifying particular principles, such as Newton's Second Law, and are then interviewed about their scientific beliefs. The same concept is represented in different problems of varied complexity. As the problems increase in complexity, some students substitute a misconception for an accurate representation. The patterns of consistency in the student's representations provide converging evidence about the nature of the misconception and the origins of the

conceptual difficulties (Eylon & Linn, 1988). The origins may include experiential knowledge acquired from observations of phenomena or from prior learning in formal settings. In this section, I will first present some of the empirical research examining conceptions and misconceptions and then address the theoretical issues and implications.

Some of the most compelling research into students' misconceptions has been done by McCloskey and colleagues (e.g., McCloskey, Caramazza, & Green, 1980) in the domain of mechanics. In these experiments, subjects were presented with a series of problems that require them to predict the trajectory of objects in motion. In one study, students were presented with a series of diagrams of curvilinear cylindrical tubes (McCloskey, et al., 1980). Subjects were asked to predict the trajectory of a metal ball when it is shot out of one end of the tube. Many of the subjects, including those who had received one or more years of physics instruction at the university level, erroneously predicted that the ball will maintain a curved path even when there are no external forces acting upon it. These subjects reasoned that an object moving through a curved tube acquires a force or momentum that cause it to continue in curvilinear motion for some time after it emerges from the tube (McCloskey, et al., 1980).

According to Newton's first law, in the absence of a net applied force, an object in motion will travel in a straight line (McCloskey, 1983). Students, who apparently had a good understanding of the laws of mechanics, expressed beliefs that an object set in motion acquires a force or *impetus* that gradually dissipates due to external forces. This misconception has been documented by McCloskey (1983) in a variety of problem situations, such as objects dropped out of planes, balls rolling off cliffs, and a ball released from a swinging pendulum.

Clement (1983) has also studied students conceptions of the forces exerted on objects in motion. For example, in one problem, a coin is tossed up in the air and is caught. The subject is asked to describe the forces operating on the coin in its upward and downward motions. A typical student response is that the force from one's hand is propelling the coin upward and a gravitational pull is the force directed downward. The correct response is that once the coin leaves your hand, the gravitational pull is the only force acting upon the coin (excluding air resistance). Many of the students expressed beliefs that "motion implies force". These invented forces are especially common in the face of an opposing force (Clement, 1983). When the opposing force supersedes the invented force the object ceases to move or reverses the direction of motion. Clement also found that after an introductory course in mechanics, students made fewer errors, but still exhibited the same misconceptions.

Many studies have documented the fact that students' misconceptions are remarkably uniform before, during, and after instruction (Eylon & Linn, 1988). Students can still achieve highly satisfactory course grades, despite holding onto very fundamental misconceptions. However, their conceptual understanding is usually bounded by prototypical examples found in textbooks and quite often, in examinations (Perkins & Simmons, 1988). Goldberg and McDermott (1987) examined students understanding of subject matter pertaining to geometrical optics. Half of the subjects had just completed an introduction to optics in a physics course. The study investigated whether students can apply the concepts learned in the class to the real world phenomena of projected images from lenses and mirrors.

The results indicated that students were able to demonstrate competency in generating algebraic equations and symbolic representations

of the laboratory situation. However, when students were asked to predict and explain alterations in the experimental situation, such as removing a lens, all the students experienced difficulty. The students who had recently completed the optics course faired somewhat better than the naive students but still exhibited substantial difficulty in applying the principles to the task situations. Furthermore, students demonstrated very basic misunderstandings about the functions of lenses and mirrors in forming an image. Also, light-rays were referred to as if they were physical entities rather than a geometrical representation that is useful for describing how light behaves under certain circumstances (Goldberg & McDermott, 1987). This study demonstrates that many students who demonstrate competency in typical academic situations are often unable to apply concepts, principles, and procedures to "real-life situations".

Misconceptions are common in adult populations, as well as in student populations. Kempton (1986) studied homeowners' understanding of the mechanisms of home heat control. Based upon interview data and observations of thermostat-setting behaviour, he was able to characterize two types of "naive theories" that they held and that guided them to adjust their thermostats accordingly. The first theory is referred to as a *valve theory*. This theory maintains that the thermostat controls the rate of heat flow, much like the gas pedal of an automobile controls the flow of gas and determines the speed of the car. According to the feedback theory, the thermostat turns the furnace on and off depending on room temperature. The setting controlled by a movable dial determines the on-off temperature. The feedback theory is a simplified account of the correct theory of home heating.

Both theories have direct behavioural implications. Kempton (1986) estimates that between 25% to 50% of Americans believe some form of the valve theory. The valve theory erroneously predicts that if you raise the temperature of your thermostat to an even higher degree than desired, the home will warm up at a faster rate than if you just set it at the preferred temperature. The behavioural implication is that one should continually adjust the temperature dial to achieve a certain level of home heating comfort. It also correctly predicts (although for wrong reasons) that when it is colder outside, you must turn up the heat to a higher temperature. The valve theory is highly functional in normal daily use, but is potentially costly in terms of heating costs and in the wasteful use of energy resources (Kempton, 1986).

Theoretical Issues in Science Concept Understanding

Conceptual Change, Radical Restructuring and Scientific Revolutions.

The fact that students exhibit significant misconceptions and that these misconceptions are resistant to change even after instruction is indisputable. However, there is considerable controversy over the origins of misconceptions. This controversy has important ramifications for research and instruction.

Several of the investigators of misconceptions review the history of the particular scientific field and draw analogies between antiquated theories of science and students' *naive theories* (Driver, 1989). This position is closely associated with the views of McCloskey (1983). He argues that students hold theories of motion that very closely resemble the medieval pre-Galilean impetus theory. The main thrust of the impetus theory is that an object set in motion acquires an impetus needed to maintain that motion (McCloskey, et

al., 1980). The impetus gradually dissipates, causing the object to decelerate and eventually come to a stop. This characterization is completely consistent with observations from everyday experience and very much at odds with Newtonian mechanics. These naive theories are believed to be quite consistent across individuals. The theories do not reflect narrow beliefs but consist of a highly interconnected coherent conceptual systems that have behavioural implications across a wide range of situations (Hills, 1989).

The implication is that students possess intuitive scientific theories that are robust, remarkably well-articulated and consistent with evolved historical schemes. There have been a wide range of such characterizations. For example, Wiser and Carey (1983) compared students beliefs about heat and temperature to theoretical notions that preceded the ideas of Black (an influential scientist who contributed to our understanding of thermal physics) in the eighteenth century and the caloric theory of heat. Brumby (1984) claimed that medical students have pre-Darwinian conceptions of natural selection that resemble the ideas of Lamarck.

Carey (1986) has claimed that learning science and supplanting naive theories with more current scientific theories is analogous to undergoing conceptual change of the same magnitude as a scientific revolution or a paradigm shift in the Kuhnian sense (Kuhn, 1970). The notion of conceptual change has been taken very seriously in the science education community. It has resulted in some investigators calling for a radical reform of science curricula and replacing current methods of instruction with an instructional program based on a conceptual change epistemology (Strike & Posner, 1985). The underlying assumption is that students naive theories and intuitions should be taken very seriously (Driver, 1989).

To better understand the nature of the controversy over conceptual change it is useful to consider a framework introduced by Rumelhart and Norman (1981). They characterize learning in terms of a schema-based representational system and in terms of three qualitatively different modes. *Accretion* is the encoding of new information in terms of existing schemata. New information is interpreted in terms of pre-existing schemata. *Tuning* is the modification and refinement of a schema as a function of the application of the schema. Tuning allows for a schema to develop so that it becomes particularly well suited to situations in which it is applied. *Restructuring* is the process whereby new schemata and concepts are created.

Carey (1985) has further refined the notion of restructuring. *Weak restructuring* involves a reorganization of knowledge structures in which new relations among concepts are represented and new schemata come into being that allow for the solutions of new problems and more efficient and superior solutions to the old ones. The conceptual entities, such as *force*, preserve their essential meaning in weak restructuring. Weak restructuring is characteristic of the expert-novice differences described by Chi et al. (1981).

The second sense of restructuring, radical restructuring, involves a fundamental change in the meaning of the individual core concepts of successive systems. Radical restructuring entails change in ontological commitments, differentiations, and the emergence of completely new theories. In keeping with a Kuhnian history of science perspective (Kuhn, 1970), Carey (1985) has likened weak restructuring to "theory change" during periods of "normal science" and radical restructuring to a "paradigm shift" or "scientific revolution". Paradigm shifts emerge out of an effort to resolve fundamental anomalies and necessitate the development of a completely new theory in which the conceptual entities are either replaced or take on

completely new meanings. For example terms such as *force, velocity, time* and *mass* have fundamentally different meanings in Aristotelian and Newtonian mechanics (Carey, 1986). Carey believes that acquiring new scientific conceptions frequently necessitates radical restructuring.

Carey's views are shared by many in the science education research community. As mentioned previously, this has led some researchers to suggest a radical reform of science instruction built around a *conceptual change epistemology* (Strike & Posner, 1985). According to Posner and colleagues (Posner et al., 1982; Strike & Posner, 1985) the (ideal) learning conditions for conceptual change are very much analogous to those which produce paradigm shifts in scientific communities. These conditions include: a dissatisfaction with their existing conceptions provoked by anomalies in which current conceptions are seen as incommensurate with evidence; new conceptions must be minimally understood and appear plausible; and the new concepts should suggest productive research activities. These authors argue that these conditions should form the basis of curricular objectives and inform teaching strategies. This view has become the focal point of new instructional methods which emphasize *confronting* students with their anomalous beliefs, and challenging students epistemological commitments (e.g., views about the nature of theories and evidence) towards the goal of replacing misconceptions with scientifically valid conceptions.

The conceptual change view of science learning has become widely accepted. Smith, diSessa, & Roschelle (in press) challenge the core assumptions of the conceptual change framework and present a compelling critique of misconceptions research. They do not deny the existence of misconceptions and give credit to this research endeavor for having rejected the *tabula rasa* view of learner's cognitive state prior to organized

instruction. They argue that conceptual change perspective is at variance with constructivism. The theory of misconceptions has emphasized the flawed character of student knowledge, while constructivism focuses on the "recrafting" of existing knowledge. This leads us to the *learner's paradox*: if students' knowledge is flawed in fundamental ways, then how is it possible for existing cognitive structures to be transformed into substantially more complex forms (Smith et al., in press). From a constructivist perspective, the only way effective cognitive structures can be learned is if they exist in some potential or emergent form in the first place.

Similarly, replacement is not seen as a viable learning process (nor a meaningful metaphor) and depends on a very simple model of cognitive structure of subject-matter knowledge (Smith et al., in press). Misconceptions are treated as if they are unitary, independent and separable cognitive elements, rather than part of a broader network of conceptual knowledge. The authors propose that an adequate theory of learning will require an understanding of how knowledge participates in a complex system. The theory will have to account for how the cognitive system evolves in content, and how more effective means are developed for recognizing applicability conditions of pieces of knowledge in the transition processes from the initial state to more advanced states. Misconceptions result from the extension of prior knowledge that is productive and functional in one or many contexts. Extending, refining, and integrating new knowledge are more appropriate goals for instruction than are replacement.

Smith and colleagues are also critical of expertise research for drawing simple dichotomies, such as abstract-concrete, surface-principled and formal-informal, that characterize the knowledge-based differences between novice and expert. The authors argue that there is substantial continuity between

expert and novice in the sense that they are both capable of reasoning abstractly and concretely in everyday or scientific reasoning. In addition, like novices, experts rely on simplified problem representations and intuitive knowledge. However, the expert's representation can be fortified by principled understanding that could be engaged when justification and elaboration are necessary. Elements of initial prior knowledge fulfill essential roles in the development and are continuously reused and refined.

diSessa (1983; in press) offers a strikingly original alternative to the traditional view of conceptual change. The work is also steeped in the Piagetian tradition. In fact, he states that the ultimate goal of the research endeavor is to develop a "computationally explicit genetic epistemology" (diSessa, in press). His view is that scientific understanding involves a major structural change toward systematicity, rather than a shift in content. He challenges the view that intuitive theories are well developed and exceedingly robust systems. He proposes that these theories are a fragmented, loosely connected, collection of ideas, having none of the commitment or systematicity attributable to theories. Knowledge is believed to be "distributed in pieces" in both initial and advanced states of understanding (diSessa, 1988). The development of expertise is not a function of a shift from intuitive everyday concepts but from the beginner's flat and fragmentary knowledge to the experts' systematic multi-layered knowledge structures.

diSessa (1983) suggests that many of these fragments, which he refers to as *phenomenological primitives* or "p-prims", can be understood as minimal abstractions from common experience. For example, the "spring scale" p-prim correspond to the belief that "squishy things" (like a coiled spring) compress an amount proportional to the force exerted on them. They are primitive in the sense that they are self evident and generally need no

explanation. They represent intermediate elements between sensory elements and consciously accessible schemata. P-prims reflect an intuitive sense of mechanism and causality. They constitute a rich vocabulary through which people remember and interpret their experience. diSessa (in press) has documented and classified a wide range of p-prims.

diSessa (1988) argues that beginner physics students may possess quite a few p-prims, but the core of ideas do not exhibit any theoretical coherence beyond a very limited context. Through instruction and formal learning, p-prims get tuned to newer contexts, refined, and reprioritized as the knowledge system is reorganized. They become supplanted in many contexts by more complex explicit knowledge structures which include physical laws. P-prims continue to exert substantial influence even in the reasoning of experts.

Locating the Sources of Misunderstanding

The debate over the nature of misconceptions has not sufficiently focused on elucidating the sources that gives rise to conceptual difficulty. Perkins and Simmons (1988) examine the reasons and sources of misconception from a more pragmatic perspective. They attempt to identify general factors across domains of science and mathematics that present particular difficulties for the student. They identify several levels of knowledge, which they call frames, which are indicative of the fact that each of these is a system of schemata internally coherent and partially independent from each other.

The *content frame* contains facts, definitions, and algorithms that are most central to the particular subject matter domain. The content frame can be faulty in a number of ways. For example, an individual's knowledge

structures may contain undifferentiated or malprioritized concepts. Newly acquired knowledge is likely to be inert, particularly if the knowledge was obtained in a didactic fashion, from a lecture for example. Misunderstandings inevitably involve the content frame, but appear to be exacerbated by weaknesses in other areas (Perkins & Simmons, 1988).

The *problem-solving frame* includes knowledge related to progress monitoring strategies and domain-specific strategies. Novice problem-solving frames often consist of counter productive strategies such as *trial* and *error*, or *perseveration* and *quitting*. Students frequently engage in what Perkins and Simmons (1988) refer to as *equation cranking*. This pertains to a process whereby students blindly plug in familiar equations because the situations look somewhat familiar and the variables seem to fit. This method invariably fails when the situation departs slightly from expectation.

The *epistemic frame* focuses on general norms having to do with the grounding of concepts and constraints in a domain. For example, in domains of science, one ought to have a theory that is consistent with the evidence. Science demands extraordinary high standards of coherence that go far beyond the scope of everyday experience (Perkins & Simmons, 1988). Problems may be experienced, for example, when students' intuitions are given priority over internal coherence. Students may approach a problem or data with a tendency to confirm their preconceptions, without properly weighing the evidence or considering alternatives.

Conceptual Competence

Perkins and Simmons (1988) provide us with some guidance in delineating sources of misunderstanding. The next progression would be a framework that would be suggestive of ways in which we can characterize

domain competence. An approach is needed to direct the investigations of conceptual understanding and provide a formal means for characterizing student performance and domain competence. There is a need to describe the kinds of knowledge an individual brings to a domain and how that knowledge undergoes change as a function of formal instruction and evaluate the extent to which learning meets with expectations.

Greeno and colleagues (Greeno, Riley, & Heller, 1984) have developed a framework for characterizing the conceptual competence required for performing cognitive tasks. The framework was developed in detail for the domain of children's counting, but possesses a certain generality. *Performance* hypotheses identify propositions about cognitive processes and structures that are used in performing tasks (Greeno, et al., 1984). Hypotheses about *competence* are concerned with general concepts and principles that are used in constructing or acquiring procedures for use in a conceptual domain. Generativity and robustness are the cornerstones of competence. Generativity is indicated by a flexible ability to generate procedures for achieving a goal in a variety of task settings. Robustness is demonstrated by the ability to adapt a procedure to accommodate unusual task constraints.

In a given domain, principles are associated with a range of situations and diverse performance procedures, which all share a set of properties that are required by the principles. Principles provide a basis for defining knowledge in a domain. Principles embody constraints on the kinds of inputs that can be processed as data and can selectively direct attention to those aspects of the problem that need attention (Gelman & Greeno, 1989). Gelman and Greeno suggest that to infer that someone possesses an understanding of some of the principles that govern a domain of knowledge,

one needs to demonstrate that the individual can exhibit correct performance in circumstances that would preclude the possibility of rote-learning. The principles may be implicit, so it is possible that the person may not be able to articulate these principles.

Competence consists of three interrelated components (Greeno, et al., 1984). *Conceptual competence* is the implicit understanding of general principles of the domain. *Procedural competence* refers to the understanding of general principles of action and planning. *Utilizational competence* is the understanding of relations between features of a task setting and the requirements of performance. Utilizational competence provides the interpretive knowledge to assess a situation and indicates the relationship between the actions of a procedure and the domain principles. This characterization permits us to assess errors, and more generally deficiencies in understanding corresponding to particular components of competence. Instructional methods can then direct their attention towards specific components of competency.

Mental Models

Mental model is a theoretical construct that has been used to describe diverse kinds of knowledge and cognitive processes from psychomotor performance on manual control tasks (Rouse & Morris, 1986) to general models of reasoning and inference (e.g., Johnson-Laird, 1983). The construct is particularly well suited for describing how individuals form internal models of systems from interacting with physical systems (Norman, 1983). These are sometimes referred to as causal mental models (Brewer, 1987). An individual's mental models provide predictive and explanatory capabilities of the function of a physical system.

The ontology of a domain corresponds to the conceptual entities or cognitive objects and relations that can be expressed. Mental models can be used to characterize the representations of objects, the topological connections to other objects, and operations over such representations or the "running" of a mental model (Williams, Hollan & Stevens, 1983). The running of a model corresponds to a process of mental simulation that can generate possible future states of a system from observed or hypothetical states and associated parameter values. An investigator can characterize subjects' models and elucidate aspects of subjects' representations that are flawed in terms of structure of a system or in terms of the inferences used to evaluate the systemic processes.

Forbus and Gentner (1986) developed a framework for characterizing learning physical systems as a progression of mental models. Initial models are experiential and perceptually based. These have very little predictive power, except in the most prototypical instances. Subsequent models include increasingly more elaborate and coherent causal theories that support a wider range of inferences and explanations. In expert models, individuals are able to translate quantitative models into qualitative ones (and vice versa). These subjects can make very precise predictions and recognize powerful generalizations of domain principles more easily. Forbus and Gentner also describe a representational and computational framework based on Gentner's structure-mapping theory (1983) and Forbus's qualitative process theory (1985). These are of methodological significance to the study presented in the paper and will be discussed in subsequent chapters.

White and Frederiksen (1987) have also developed a framework for characterizing how individuals learn how physical systems work. The framework is embodied in an intelligent learning environment that can teach

students how to reason about electronic circuits. The central idea is that students need alternative mental models that represent the system from different but coordinated perspectives, such as at the macroscopic and microscopic level. A source of difficulty in learning results from the fact that individuals spontaneously impose intuitions about causality, mechanism and purpose. These intuitions clash with the kind of formal quantitative *constraint-based* models that are common in physics textbooks (White & Frederiksen, 1987).

The instructional framework emphasizes the acquisition of *progressions of qualitative mental models* (White & Frederiksen, 1988). Each mental model should support increasingly evolved causal explanations of system behaviour, each of which is adequate for solving some subset of problems within the domain. The models vary in their order of complexity. *Zero-order models* can be used to reason about binary states such as, the presence or absence of resistance, voltage or current; *first-order models* support reasoning about changes in state and can answer questions such as "Is the light getting brighter"; *second-order models* can be used to reason about the rate at which a variable is changing. The progression of models reflect a series of instructional goals for the student necessary to master the model that is driving the simulation environment.

Summary

This chapter covers a wide range of empirical and theoretical issues. Can there possibly be any underlying theme that connects the topics that comprise this chapter? I would suggest that the central issue is conceptual understanding and the transfer of scientific knowledge. Related to this issue are the particular kinds of difficulty that result in a lack of transfer and the ensuing consequences. The minimum prerequisites of a learning theory include: a characterization of models of competence; an explanation of the how and why student performance diverges from the standard set by the model; and a set of transition mechanisms that can account for transformations in knowledge structures and problem-solving skills that support superior standards of performance.

It is generally agreed that an expert's problem-solving ability is a result of years of domain related experience, in which he or she builds up a rich, highly interconnected network of information units, commonly referred to as schemata. This network serves as an index to rapidly guide experts to relevant parts of their knowledge store (Larkin et al., 1980). The declarative knowledge contained in the schema generates potential problem configurations and specifies the conditions of applicability, which are then tested against the information in the problem (Chi et al., 1982). The procedural knowledge generates potential solution strategies. Expert's are more capable of recognizing the conditions for applicability of the appropriate procedures. This can account for the increase in speed, efficiency and the effective use of strategies that are hallmarks of expert performance.

Expert-novice comparisons provide us with some indices for characterizing competent performance, which in turn can give us some ideas for differentiating superior student performance from inferior student

performance. Students lack the rich interconnected knowledge structures that experts possess. The development of knowledge structures is one of increasing differentiation built around domain principles. The increasing ability to recognize the applicability conditions of these principles emerge a lot sooner in the better students (Thibodeau, et al., 1989; Chi, et al. 1989). Superior students seem to acquire categories at an appropriate level of abstraction that provides a more principled basis for further acquiring and refining knowledge.

The crucial issue in transfer is to explain how previously learned elements can be evoked and successfully applied in different situations. The kinds of transfer described by Salomon and Perkins (1989) may be used to fine-tune instructional strategies to fulfill particular kinds of learning goals and provide the conditions suitable for transfer to occur.

Analogical transfer has been widely investigated in experimental psychology. These studies have documented the difficulties students have in relating knowledge from a source or familiar domain to a targeted unfamiliar domain. Difficulties in analogical transfer are a result of several factors including: the failure to recognize or *access* potentially useful analogs (Gick & Holyoak, 1980; 1983); and an excessive reliance on the surface features of a problem for retrieving and in particular, *mapping* features from a familiar problem to a novel situation (Holyoak & Koh, 1987; Novick, 1988a). The transfers of concepts, procedures, or principles are affected by the degree to which they are embedded in a given problem domain (Bassok & Holyoak, 1990). The effect of the "embedding" is to obscure the structural features because the surface features may become highly salient (Bassok, 1991).

Several studies have examined the role of spontaneously generated analogies during the course of problem-solving. The results suggest that one

can predict the pattern of inference and the probability of success given the kinds of analogies generated by subjects (Gentner & Gentner, 1983). Clement (1988) found that experienced problem-solvers tend to generate most analogies via a process that transforms the problem into a situation in which the relational mappings to a more familiar domain become transparent. The transformational process of analogy generation has been largely overlooked in the literature, which emphasizes analogy by association.

Misconceptions can be characterized as both failures of transfer and impediments to future transfer. The studies discussed in this chapter suggest that misconceptions: are robust, reappearing in different situations embodying the same principles (McCloskey, 1983); can be resistant to formal instruction (Clement, 1983); become evident when students attempt to apply knowledge, acquired in restricted formal learning contexts (e.g., from textbooks and didactic lectures), to real-life situations (Goldberg & McDermott, 1987); and can have clear behavioural implications in reasoning in everyday situations (Kempton, 1986).

The nature and origin of misconceptions have been the subject of considerable discussion. Several authors have drawn interesting parallels between students' naive conceptions and antiquated scientific theories (McCloskey, 1983). This provides us with some insight into the kinds of conceptual change or knowledge restructuring that needs to occur in learning science. However, the framework is insufficient to elucidate significant mechanisms of learning or to prescribe new instructional methods.

Smith and colleagues (in press) criticize the core assumptions of misconceptions and conceptual change viewpoint. In particular, they claim that the theories are at variance with tenets of constructivism. They raise the issue of the *learner's paradox*, which begs the question, if students' prior

knowledge is essentially flawed, then how is it possible for students to succeed in transforming their knowledge structures into more productive forms. Constructivism emphasizes the ways in which refined and elaborate knowledge emerges from simpler and incomplete forms.

Perkins and Simmons (1988) characterize potential sources of misunderstanding. Two of the sources discussed are: the content frame, which refers to concepts, definitions, and learned procedures that comprise a particular subject matter domain; and the *problem-solving frame* that includes knowledge related to metacognitive and domain-specific strategies. These two topics have been investigated extensively. The third frame, the *epistemic frame*, focuses on general norms and standards having to do with the grounding of concepts and constraints in a domain and the practice of scientific methods. The issue of adherence to standards of science has not received much attention from researchers.

Accounting for patterns of misunderstanding suggests the possibility of a model of competence. Greeno and colleagues (Gelman & Greeno, 1989) discuss a framework for characterizing conceptual competence. Competence is related to the general concepts and principles that are used in constructing or acquiring procedures for use in a conceptual domain. Principles are associated with a range of situations and diverse performance procedures, which share common properties. Competence for a given domain is a function of: generativity, which refers to a flexible ability to generate procedures for accomplishing goals in diverse task settings; and robustness, which is demonstrated by the ability to accommodate novel and seemingly anomalous task constraints.

To describe how individuals come to understand the working of physical systems requires an account of their intuitive sense of mechanism

and causality. diSessa (in press) has developed a framework for describing how people's sense of physical causality is framed by minimal abstractions derived from experience. P-prims constitute a rich vocabulary through which people remember and interpret their experience. Similarly, mental models (causal mental models) provide a framework for characterizing how people develop models of physical systems through formal and informal interaction. A characterization of an individuals' mental model can account for their predictions and explanations. The constituents of a students' mental model include a representation of the structure, function, and behaviour of a system (Williams, Hollan & Stevens, 1983). These constituents can be systematically analyzed to identify flaws or misconceptions. It is possible to characterize the acquisition of expertise through a series of qualitatively distinct process models (Forbus & Gentner, 1986) and prescribe methods for developing progressions of qualitative models of increasing robustness and generativity (White & Frederiksen, 1986).

This chapter provides a sketch of the kinds of research and theories that have evolved to characterize conceptual understanding of science concepts. The diversity of approaches highlights the complexity and multi-dimensional nature of the problem. This chapter primarily focused on conceptual understanding in physical domains. In the next chapter, I address issues of conceptual understanding in the biological and biomedical sciences.

CHAPTER FOUR

UNDERSTANDING BIOLOGICAL AND BIOMEDICAL CONCEPTS

The biological and biomedical sciences have undergone a dramatic transformation over the last twenty-five years (Tosteson, 1990). The study of biological cognition[4] has not received very much attention in science education or cognitive science research, despite the fact that it is the most widely taught of all the sciences from elementary school through college (Stewart, 1991). However, there is some indication this research activity has increased in recent years. Although the studies are far fewer in number than for the physical sciences the same kinds of issues, such as expertise, problem representation, analogical transfer, and misconceptions have been addressed in relation to the biological sciences. Like the topic of mechanics in the physical science concept research, genetics has been the most widely studied of the biomedical or biological domains. This chapter discusses empirical and theoretical issues related to the study of understanding biological and biomedical concepts and draws comparisons with the physical sciences.

Some Epistemological Issues

Biology is the science of living organisms (Johnson, 1983). According to the Committee on Models for Biomedical Research (CMBR) (1985), biomedical science encompasses a vast array of research activities that have as their ultimate objective improved understanding of the human organism in health and disease. The distinction between studies in biological and biomedical research principally reflects differences between communities of

[4]Biological cognition is used here to refer to the process of thinking about biology rather than the biological basis of cognition.

research rather than conceptual or methodological demarcations. Any basic biological research can be potentially applicable in biomedical contexts (CMBR, 1985).

There are many substantive differences between the biological sciences and the physical sciences. Physical science knowledge is embodied in laws and principles, which are typically conceived as universal statements relating classes of empirical findings and processes (Hull, 1974). The theoretical structure of classical physics consists of a set of postulates such as Newton's Law or Maxwell's Law (CMBR, 1985).

Schaffner (1980) argues that most theories in the biomedical sciences are not now, and cannot be, universal theories. Possible exceptions cited are the genetic code, the theory of protein synthesis and, to a certain degree, evolutionary theory. He contends that most biomedical theories can be best characterized as "middle-range" theories. This kind of theory falls between biochemistry at one extreme and evolutionary theory at the other extreme on the continuum from molecules to populations. As discussed in chapter 2, biomedical theories are characterized as *interlevel*, because of the way in which theories become elaborated by development in the downward direction and because of the strong interconnections between separate biomedical disciplines. For example, genetics is a vital part of immunology, and neurology draws upon biochemistry and cell biology (Schaffner, 1980).

Models in both the physical sciences and the biomedical sciences are analogs, in the sense that the models possess the same or similar structures or functions as the system under investigation. Biomedical research has an additional analytical tool at its disposal—homology, which is correspondence in structure and function derived from a common evolutionary origin (Committee on Models for Biomedical Research, 1985). There are many

shared genetic sequences and common functions between organisms. Models by homology are of heuristic value in the search for good analogs, which are chosen on the basis of whether they are good models by analogy for the phenomenon or structure being studied. Different organisms, sometimes genetically distant ones, provide appropriate models for studying different processes and functions. For example, the spontaneously diabetic Wistar BB rat is an excellent model in the study of juvenile diabetes (CMBR, 1985). This serves to illustrate a point that despite lacking universal postulates, biology possesses a number of generalizations whose validity rests on evolutionary relationships.

Domains can be characterized according to their degree of well-structuredness (Simon, 1973). The physical and mathematical sciences are very well structured in the sense that there are definite goal states and the problem space is reasonably well constrained. The biological sciences are somewhat less structured in the sense that they are predominantly nonmathematical and because they lack a system of axiomatized knowledge (Ploger, 1988).

The structuredness issue is a concern for education in the biological sciences because students cannot be taught precise algorithms and the standards of coherence are not as evident as in the physical sciences. For example, in mathematical problem solving a student can apply specific axioms and subsequently evaluate the results. In the biomedical sciences, the standards are less formal and the effectiveness of the solution strategy is less immediately apparent. In general, problem solving in the biomedical sciences may not afford the same opportunities for the epistemic challenges that are necessary to induce conceptual change.

Research in Biological Concept Understanding

The Development of Biological Knowledge

The nature of biological knowledge has been an active area of research in cognitive development. A vexing problem in developmental psychology is that experience is inadequate to justify or account for the kinds of inductions that children routinely and universally make (Gelman, 1990). A commonly expressed view, reiterated by Gelman (1990, p. 5) is that "the mind brings domain-specific organizing principles to bear on the assimilation or structuring of facts and concepts, that the learners can narrow the range of possible interpretations of the environment because they have implicit assumptions that guide their search for relevant data."

Carey's (1985) research addresses this question in the context of the development of biological knowledge. Although, an extended discussion of her work is beyond the scope of this chapter, certain issues are of particular relevance. Specifically, the research has implications for characterizing the understanding of biological and biomedical knowledge in students at advanced levels of schooling. She argues that children have a few theory-like cognitive structures in which their notions of causality are embedded and which can be used to organize experience. Cognitive development consists, in part, of the emergence of new theories out of older ones with an accompanying restructuring of "ontologically important concepts".

Carey traced the development of children's understanding of basic biological concepts, such as a *living thing* and *animal,* through early childhood (ages 4-10). She presented a series of clever experiments in which children are requested to make judgments. For example, in one study children were asked to determine whether a set of biological properties such as *eats, sleeps, and thinks,* could be attributed to people, unknown animals

(e.g., aardvarks), plants, and inanimate objects. The goal was to determine whether children represent a concept (in the above example-the concept of animal) with the same extension as do adults.

The results of these studies demonstrate that the inductive inferences of young children differ substantially from those of older children and adults. For example, young children decide what things in the world have certain properties, such as breathing or thinking, by comparing them to people, and determining whether people have these properties. Ten year-old children and adults tend to rely on category membership and knowledge of biological function. In addition, the results suggest that young children conceptualize processes such as *death*, *growth*, and *reproduction* in terms of behaviour of the whole person rather than in terms of the function of internal body parts.

Carey (1985) claims that young children possess a "naive-psychology" theory of biology. The theory structure appears to be embedded in social and psychological contexts and explanations are provided in terms of motivation, intention and social conventions. For example, when asked why people eat, 4 year-old children answered, "Because they are Hungry" or "Because it is Dinner Time". What emerges in older children is an "intuitive biology" based on an implicit understanding of biological principles. She concludes that the transition process is very likely one of radical conceptual restructuring that involves a fundamental change in ontological commitments and core concepts.

Biological Problem-Solving and Expertise

There have been comparatively few studies of expertise in the biological sciences. Smith and Good (1984) studied the performance of students and instructors at three levels of expertise in the domain of genetics.

As is characteristic of expert performance in other domains, the study found that expert subjects use a forward-reasoning strategy in solving problems, while novices tended to use a backward reasoning or means-ends analysis.

Smith and Good (1984) also documented a range of behaviours that tended to result in either success or failure. For example, experts and to a lesser extent successful novices, used a broader range of domain-specific heuristics for generating solutions as well as for validating solutions. In addition, students who had just completed a course in genetics demonstrated that they could retrieve such knowledge but the subjects were typically ineffectual in applying this knowledge to solve genetics problems.

There are two ways in which biomedical domains differ from the physical sciences, which have clear implications for problem-solving behaviour. The first has to do with the levels of knowledge evident in biomedical domains, and the necessity of traversing levels to solve problems (Schaffner, 1980). The second has to do with the issue of abnormal function, which is of primary importance in the biomedical sciences (Ploger, 1988).

Ploger (1988) also characterized two effective strategies for solving metabolism problems in biochemistry: the *normal function strategy,* which involves reasoning about normal function before making reference to abnormal function; and the *known pathology strategy*, which involves first introducing a known pathology and then determining whether it is relevant to the problem. The results of the study indicated that experts were more likely to use a variation of the normal function strategy in their problem-solving and in their explanations of problems. Novices attempted to focus on abnormal function and tried to characterize local reaction mechanisms. Experts were also able to categorize a problem at a more general level, as an instance of a particular principle.

There has been a tendency to characterize expertise and expert knowledge as a homogeneous entity. In many domains, medicine in particular (Patel, Groen, & Arocha, 1990), there are many kinds of expertise. Smith (1990) studied the difference between students and experts in solving and classifying genetics problems. There were two groups of genetic experts; university teachers and researchers formed one group, and genetic counselors constituted the other group. Both expert groups were able to solve the problems with a considerable degree of accuracy, exhibiting a performance clearly superior to the novice students.

In this study (Smith, 1990) the novice subjects grouped the problems according to the surface structure elements, which is consistent with previously described studies. However, the two groups of experts differed considerably on their classificatory schemes. The faculty experts classified the problems according to the underlying domain principles. The genetic counselors classified the problems according to the problem knowns and unknowns. Academics and counselors or practitioners are required to perform different functions in their professional work. A faculty member's task is to teach students to understand principles and engage in research that furthers the scientific communities' understanding. The genetic counselor's task is to collect and analyze information and advise his or her clients as to the best possible solutions. This result may be explained by the fact that the experts have actively structured their knowledge to address the different tasks their work may entail (Smith, 1990).

Most studies of analogical transfer that have demonstrated an inability of subjects to spontaneously generate analogies have focused on problems that demand little prior knowledge. When subjects are trained in a knowledge-rich domain, the tendency towards the use of analogies is significantly greater (e.g., Bassok & Holyoak, 1989).

Dunbar and Schunn (1990) looked at the effects of analogical transfer in two domains of molecular biology in a computer-based simulated scientific discovery task. The source domain included a series of problems related to virus reproduction. The target domain was a genetics problem which involved finding the mechanism for how genes are controlled by other genes. The mechanism was one of *negative regulation*, in which a secreted substance inhibits the effect of another substance. There were three 'source' conditions: in one of the virus conditions, subjects received a problem in which the mechanism was one of negative regulation; in a second condition, the mechanism was positive regulation and in a third condition, subject received no training in the source domain.

The results indicated a substantial facilitation effect for the group in the negative virus condition. Eighty percent of the subjects who solved the negative virus problem also solved the genetics problem. Only 35% of the subjects in the other two groups were able to solve the problem. A striking finding was that subjects' verbal protocols and subsequent explanations suggest that there was no explicit or conscious analogical transfer. Subjects were not cognizant of the fact that the two problems were structurally isomorphic. The authors suggest that the virus problem may have primed the concept of negative regulation without an explicit awareness. They further suggest that by learning through experimentation, subjects may have

acquired an abstract concept without the contextual or surface information. The implicit transfer of principled knowledge has rarely been demonstrated in scientific concept learning research.

Conceptions and Misconceptions

Biological functions can be thought about in terms of their mechanism of action or in teleological terms (Richardson, 1990). While it is advantageous for students to have a principled mechanistic understanding of scientific concepts, teleological or goal-oriented explanations are often presented in textbooks and in lectures to orient students to the functions of a particular bodily mechanism.

Richardson (1990) studied the degree to which physiology students preferred mechanistic or teleological explanations via their responses on a multiple choice questionnaire. Students would be presented with questions such as:

> During physical activity, oxygen enters muscle tissue from the blood because:
>
> A) oxygen content inside muscle tissue decreases as oxygen is used.
> B) muscles require oxygen to produce energy.

In this example, choice A is the mechanistic explanation and choice B is the teleological explanation. He found that students who were taking elementary physiology courses and students who were enrolled in advanced physiology course greatly favoured teleological responses over mechanistic ones.

A teleological bias may impede students from acquiring mechanistic accounts of physiology. This kind of bias is analogous to the psychological and social explanation of bodily function provided by young children in Carey's (1985) research. Richardson also demonstrated that students

explanations could be modified by a discussion of the differences between the two kinds of explanation, while young children undergo a radical conceptual restructuring of knowledge structures. Nevertheless, the study may suggest that teleological explanations and simplifications may have a considerable seductive appeal to students learning biological science.

Misconceptions appear to be equally evident in the biological sciences as they are in the physical sciences. Fisher (1985) documented a common and pervasive misconception in biology students. Students express a mistaken belief that amino acids are produced by genetic translation (protein synthesis) despite demonstrating a knowledge of considerable factual information related to the translation and protein synthesis. Fisher was able to characterize multiple contributing factors for this misconception. These factors involve confusion over the fact that amino acids have a dual role as an *activational factor* in protein synthesis and *as a product* of protein synthesis. An additional source of difficulty is that students lack knowledge about how amino acids are synthesized in cellular reactions. Fisher suggests that cellular biology is a difficult subject to learn or teach because it involves complex systems with components that are highly interrelated. Typically textbooks and lectures compartmentalize and fragment knowledge by presenting topics separately and minimizing the complex inter-relationships.

In the science of biology, the theory of evolution provides a unifying framework to account for a diverse body of seemingly disparate empirical findings (CMBR, 1990). Bishop and Anderson (1990) found that many students exhibited misconceptions concerning the mechanism of evolution. Biologists recognize that two distinct processes influence traits exhibited by population over time (Bishop & Anderson, 1990). They also recognize that traits originate due to random changes in genetic material through mutation

or sexual recombination and through the process of natural selection in which certain traits survive or disappear due to environmental factors. Students tended to conflate this distinction and suggested that species change gradually, during their lifetimes (rather than over generations), to accommodate environmental demands. Student explanations for evolution tended to be teleological in nature. For example, they explain evolution in terms of *needs* — organisms develop new traits because they need them to survive. Brumby (1984) documented similar misconceptions, reflecting a *Lamarkian* perspective (pre-Darwinian), in first year medical students' understanding of concepts of natural selection and genetic change. These misconceptions were also not altered by an introductory course in genetics.

Arnaudin and Mintzes (1985) studied students' conceptions of the human circulatory system. The subjects included students from elementary school to college level biology students. The study found that students, at all levels, held erroneous beliefs about the structure of the heart, the function of the blood, and the circulatory/respiratory relationship. Many of the students, particularly those in earlier grades, explained the function of the blood with *vitalistic* responses, such as "it keeps you alive". Several students believed that the circulatory system acted as a singular flow system, rather than as a double pump mechanical system (pulmonary/systemic). This study clearly demonstrates that, like the physical sciences, certain misconceptions develop in the early years of school and can remain stable into university levels of education. However, one's interaction with the biological world is less transparent in that internal biological structures and processes are covert and relatively inaccessible to inspection. The authors also found that certain misconceptions or preconceptions are readily removed during the course of

formal learning. This raises the question of why specific misconceptions are highly resistant to change, while others are changeable.

Chi, Chiu, and de Leeuw (1990) examined students mental models of the circulatory system. They were particularly interested in characterizing the ways in which conceptions and misconceptions differ in the biological sciences and physical sciences. The authors hypothesized that an understanding of the circulatory system requires an integrated model of the functional, structural, and behavioural interrelations between the heart, the blood, and the vessels. This necessitates an understanding of system components and the organized interaction between these components. Chi and colleagues presented students at two levels of ability, as determined by their CAT score (an aptitude test), with a pretest interview to evaluate their understanding of the circulatory system. They were then presented with a descriptive text describing aspects of the circulatory system about which they were asked to explain while talking out-loud. Subsequently they were asked to respond to different kinds of questions, which included questions explicitly taken from the text, more complex inferential questions, and questions intended to assess whether students held misconceptions similar to theories of scientists in the era that predated Harvey, the 17th century physician who provided the first contemporary scientific model of the circulatory system.

The results suggested that students improved significantly from the pre-test to the post-test. High ability students showed a greater gain in the correct responses on implicit questions and were able to better elaborate on system function than were low ability students. Misconceptions were evident in subjects' responses but were not consistent with previous scientific theories, as is the case in the physical sciences. Most misconceptions were removed after subjects read the text suggesting that these were typically less

resistant to change as compared to physics concepts. When information in the text was perceived as inconsistent with their prior knowledge, subjects were able to revise and update their mental models. The results were interpreted as being consistent with a theory of conceptual change (Chi, 1992). The substance and implications of this theory are discussed later in the chapter.

Cardiopulmonary physiology embodies many concepts, from molecular to the organ systems level, that have referents at multiple levels of representation. To understand a concept requires that a student coordinate multiple structure, function and behaviour at multiple levels of representation. Patel, Kaufman, and Magder (1991) evaluated the ability of first year medical students to use the concept of ventilation/perfusion matching in the lungs to explain a problem of a patient with an embolus obstructing blood flow. The results revealed systematic misconceptions by students in developing a *pathophysiological model* of the problem. The subjects demonstrated an inability to coordinate events in the right and left lungs and in the dysfunctional and functional regions of lung tissue. Students frequently were not able to conceptualize the cardio-pulmonary system as a closed system, with an event in one region propagating effects throughout the other regions of the lung. They also exhibited difficulty in relating explanations at different levels of abstraction: cellular to organ to patient levels.

Complex Biomedical Concepts

Feltovich and colleagues have developed a framework for investigating medical students understanding of complex concepts (Feltovich, Spiro, & Coulson, in press). Their approach is discussed in some detail because it has

a significant bearing on our research. The framework is based on a method involving a highly concentrated analysis of networks of concepts. The method includes a scheme for analyzing conceptual structure, which can be used to identify areas of potential cognitive difficulty. Concepts are decomposed into their most basic elements. For each concept studied a probe set of questions is developed. The first questions tend to be open-ended and span the entire scope of the concept. The questions then address the basic elements of the concept. Subsequent questions require more complex kinds of integration and synthesis. The final items of a probe set include selected application questions intended to reveal classes of misconceptions. Analysis is directed at various kinds of commonalties in responses, to identify the kinds of conceptual models exhibited by student and their limitations.

The framework has been used to study large-scale misconceptions in medicine, especially in the domain of cardiology (Feltovich, Spiro, & Coulson, 1989). These misconceptions can be decomposed into component misconceptions. The components are interdependent and can be represented as "reciprocating networks of faulty ideas that mutually bolster each other". Each component misconception has multiple sources that converge and reinforce the erroneous knowledge. These sources include one or more psychological reductive biases that favour the development of simplified conceptual models. The authors also suggest that specific instructional practices and materials can contribute to reductive biases.

A clear example of their work is reflected in a misconception related to congestive heart failure (Feltovich, et al., 1989). This is a syndrome in which the heart's effectiveness as a pump can diminish greatly and as a result the rate of blood flow slows dramatically. The misconception that was expressed by over 60% of first and second year medical students in a study, and by some

medical practitioners, suggest that heart failure is caused by the heart getting too big which in turn stretches the cardiac muscle fibers. The force of contraction is determined by *mechanical/anatomical* factors and activational factors (energetic). The primary cause of congestive heart failure is activational, while the misconception emphasizes the mechanical overstretching as the cause for heart failure.

Several component misconceptions were identified. These misconceptions interact and support each other yielding a robust conceptual structure. The components include an inappropriate analogy that an individual cardiac muscle fiber is like an individual skeletal fiber. The two kinds of fibers differ on the dimensions of importance (length-tension). However, students have a better acquaintance with skeletal muscle fiber and instructors use the analogy to introduce the cardiac dynamics.

Another component misconception is the belief that the behaviour of an individual isolated cardiac muscle fiber is an accurate reflection of the behaviour of an intact ventricle (chamber of the heart). Textbooks frequently present a graphic depiction of single cardiac muscle fiber to illustrate the length-tension relationship.[5] As a consequence, students reason that the length-tension relationship is isomorphic to the volume-pressure relationship in a contracting ventricle. Students assume that the parts of the system add up to account for the function of an intact system, failing to consider the emergent properties of a higher level of aggregation.

The discussion of this misconception amplifies several important themes. Misconceptions emanate from multiple converging sources of students knowledge. The sources or pieces of knowledge by themselves can

[5]The length-tension relationship explains the tension developed in the heart as determined by the degree of stretch of cardiac fibers. This is specified by the Frank-Starling Law of the Heart.

be partially correct or fully correct, but may be inappropriately inserted as a causal mechanism. The second important theme is that instructional practices and resources (textbooks) can be a primary contributor to a component misconception. This could be as a result of an educator's own misconception or more frequently is a result of simplifying a complex concept so that students may grasp it more easily.

Conceptual understanding is limited by psychologically reductive biases. Biases involve systematic ways of reducing complexity to make concepts more amenable to understanding. Feltovich and colleagues have developed a taxonomy of reductive biases (Feltovich, et al., 1989, p. 127-128). These include the following:

> **Static Bias**—The representation of a dynamic, continuous, changing process in static terms.
>
> **Step-wise Bias**—Continuous processes are broken down into discrete steps, with a loss of properties at the holistic level.
>
> **Prior Analogy Bias**—New concepts are interpreted through already held simpler models, often imported from extra-instructional experience.
>
> **Common Connotation Bias**—Technical terms are interpreted according to their everyday, common language meaning.
>
> **Restrictional Scoping**—General principles are thought to apply only in specific instances.

These reductive biases can in part be traced to *acquisition biases*, which are modes of approaching complex ideas from either the learners' or the instructors perspective (Feltovich, et al., 1989, p. 127-128). They include: *underdimensioning*--a representational approach of teaching or learning

multidimensional phenomena, one dimension at a time, with a goal of putting all the pieces together; *atomization and extirpation*--a bias that extracts and isolates components from a multi-component system with the assumption that their behaviour in isolation will accurately reflect their behaviour in context; and *sanitizing*—focusing on the clearest, least complicated example of a concept with the rationale being that these can serve as a bridge to introduce more complicated instances.

The research of Feltovich and colleagues suggest the need for a comprehensive framework for characterizing conceptual understanding in a biomedical domain. This framework incorporates an investigative method and method of analysis for identifying sources of conceptual difficulties. These sources have multiple origins, including the students knowledge-base and a tendency to reduce complexity by the student which is reinforced by methods of formal instruction.

Conceptual Understanding in the Biological and Physical Sciences

There are many commonalties as well as many differences between learning in the physical sciences and the biological sciences. Chi (1992) has recently proposed a theory of conceptual change that focuses on the different kinds of change evidenced in the physical and biological sciences. This framework serves as a starting point for discussion and summary of issues related to conceptual understanding in the biological sciences. At the heart of this distinction is the nature of ontological categories. Ontological categories are fundamental classes of objects and events that partition our knowledge of the world (Keil, 1989). Psychological processes (e.g., abstraction) cannot transform a concept in one ontological category to a concept in another

ontological category (Chi, 1992). For example, an inanimate object cannot become a living thing.

The primary assertion in Chi's theory (1992) is that conceptual change within ontological categories require a different set of processes than conceptual change across ontological categories. The kinds of learning processes for achieving different kinds of conceptual change may be fundamentally different. Radical conceptual change requires that a concept be reassigned from one ontological category to another. Chi argues that the scientific meaning of physical science concepts belongs to a different ontological category than naive intuitive meanings. For example, scientists view the concept of force as belonging to the category of "constraint-based events". A constraint-based event is an event that exists under the constraints of other entities. To illustrate this point, a force does not exist, unless an object is moved into a force field (Chi, 1992).

Students perceive physical concepts, such as force, as belonging to the category of material substances. This description is consistent with McCloskey's (1983) and others' research that shows that students believe force to be a property of a body in motion.

Chi (1992) argues that there are three kinds of empirical evidence that support the argument that learning physical science requires radical conceptual change. These include: 1) the fact that misconceptions in the physical sciences are robust coherent conceptual structures, resistant to change over long periods of time, 2) these naive beliefs resemble the beliefs of medieval scientists; and 3) the process of discovery that moved scientists to newer theories required radical conceptual shifts.

Chi claims that the biological sciences involve a kind of learning or conceptual change that would not necessitate a shift in ontological categories

and therefore would not involve a radical conceptual change (Chi et al., in 1990). She identifies four dimensions in which biological science domains differ from physical science domains. The first dimension has to do with the nature of explanations. In the physical sciences, explanations are expressed in terms of deductions from principles, which are regularities that are expressed in mathematical equations. In the biological sciences, explanations involve an explication of structural, functional and behavioural inter-relations.

A second dimension of difference is reflected in the nature of misconceptions. As previously discussed, misconceptions in physics are due to people's intuitions about physical events that belong to a fundamentally different ontological category (substance-based objects) from accepted scientific theory. Misconceptions in biology are more likely to be due to a lack of knowledge, which is more readily correctable. They may also occur because biological processes are covert—the entities undergoing some change is not readily observable, as are physical processes.

A third source in which these two branches of science differ is in the pattern of misconceptions. Chi and colleagues (Chi, et al., in 1990) argue that misconceptions in biology are less consistent across studies, populations, ages, and across historical periods. She suggests that their appears to be less consistency across students, and there does not appear to be any systematic underlying cognitive structure. The fourth dimension of difference is that learning in the biological sciences should be more readily attainable than in the physical sciences.

The dimensions of difference as proposed by Chi and colleagues provide an interesting starting point for characterizing conceptual understanding in the biological sciences. Although there is much in the framework to

recommend, I wish to express some points of divergence. The first point has to do with the nature of explanations. It appears that explanations in most biological domains are not expressed in terms of universal laws. However, genetics is a notable exception in which principled explanations are similar to those expressed in the physical sciences. Secondly, some domains of biology may be better thought of as constraint-based rather than in terms of the inter-relations between structure, function, and behaviour. Hemodynamics is a good example of a constraint-based domain. In this domain, the structure-function relations are not as explicit and physical principles, such as those that govern pressure-volume relations, play a significant role.

Finally, contrary to Chi's characterization, certain misconceptions seem to be quite robust, consistent across individuals and ages and are extremely resistant to change (e.g., Fisher, 1985). Although, students' "Lamarkian-like" beliefs about evolution are a notable exception (Bishop & Anderson, 1990; Brumby, 1984), there seems to be support for the idea that biological misconceptions do not resemble antiquated scientific theories. In addition, Feltovich and colleagues (1989) clearly demonstrate that there can be a very systematic underlying conceptual structure supporting misconceptions in a biological domain.

Chi raises a very significant issue related to the fact that biological processes are covert and students are less likely to have the same kinds of intuitions that they develop from observing physical processes from everyday life. Individuals experience with biological events are very different. Children's earliest experiences have to do with fulfilling their own biological needs such as hunger. As Carey (1985) has pointed out, young children develop psychological theories of biological processes. Biological systems are viewed as *psychological agents*, deliberately acting to fulfill some goal and

scientific theories of biology emphasize mechanistic accounts of biological phenomena.

In between the scientific and animistic accounts provided by young children are teleological descriptions provided by students in different disciplines. Teleological descriptions focus on the *purpose* of a system. Biological systems are viewed as devoted to achieving a set of goals. The students do not quite anthropomorphize the systems, as do young children. Rather they tend to obscure function with purpose (Richardson, 1990).

The Lamarkian view of evolution adopted by students is also consistent with a teleological perspective (Brumby, 1984). Teleological descriptions are frequently provided by instructors and textbooks to explicate the function of a complex system. However, they may lead to simplified mental models that could impede students from learning mechanistic accounts. It is very likely that individuals evolve a set of phenomenological primitives for biological concepts as is the case in physics (diSessa, in press). However, they make take a very different form. These p-prims and our conceptual understanding may correspond to teleological intuitions in which systems are viewed as fulfilling some purpose. It should be noted that this discussion is highly speculative at this point.

The biological sciences have strong interlevel connections that bridge different disciplines such as cellular biology and biochemistry (Schaffner, 1980). The disciplines are also typically lacking in universal postulates. Experts have developed strategies for traversing between levels to solve complex problems (Ploger, 1988). The development of this skill entails the acquisition and coordination of multiple models of systems at different levels of abstraction. The cognitive difficulties experienced by students may be traced to one or more of the source models or to an inability to coordinate

these models (Patel, Kaufman, & Magder, 1991). Misconceptions are more likely to evolve from multiple converging sources, particularly in the biomedical sciences (Feltovich, et al., 1989). Furthermore, the etiology of these misconceptions is more commonly a function of formal learning rather than everyday experience (Feltovich, et al., in press).

This chapter characterizes the progress towards a model of conceptual understanding in the biological sciences. At this time, the model is still rather sketchy and there is a need for considerable research. We are at the stage where we can frame the questions more cogently, which can provide us with some of the answers.

CHAPTER FIVE

CARDIOVASCULAR AND CIRCULATORY PHYSIOLOGY

The subject matter of this research is the mechanics of cardiovascular and circulatory physiology. There are two superordinate concepts, which provide the focal point of the study: cardiac output and venous return. *Cardiac output* is defined as the total amount of blood pumped from the heart per unit time. *Venous return* is the amount of blood returning to the heart per unit time. This chapter presents a survey of some of the pertinent concepts. The primary sources of information for this discussion are to be found in two physiology textbooks: Circulatory Physiology by Smith and Kampine (1990); and Fundamental Cardiovascular and Pulmonary Physiology by Green (1987).

Structure and Function of the Systemic Circulation

The circulatory system, as illustrated in Figure 1, is the transport system for the delivery of oxygen and the removal of carbon dioxide. There are two main components of the circulatory system: the smaller pulmonary division consisting of the pulmonary arteries, capillaries, and pulmonary veins; and the larger systemic division consisting of the aorta, arterial branches, capillaries, veins, and the vena cavae. The systemic vessels supply and drain all the organs and tissues of the body.

The heart propels the blood through both divisions. It has four chambers, a right and left atrium and a right and a left ventricle. The ventricles are the primary pump elements. The right ventricle propels blood through the pulmonary artery to the lungs (pulmonary circulation) and the left ventricle through the aorta and systemic arteries to the remainder of the

body (Smith & Kampine, 1990). This comprises the systemic circulation. The atria are smaller pumps that assist the flow of blood into the ventricles.

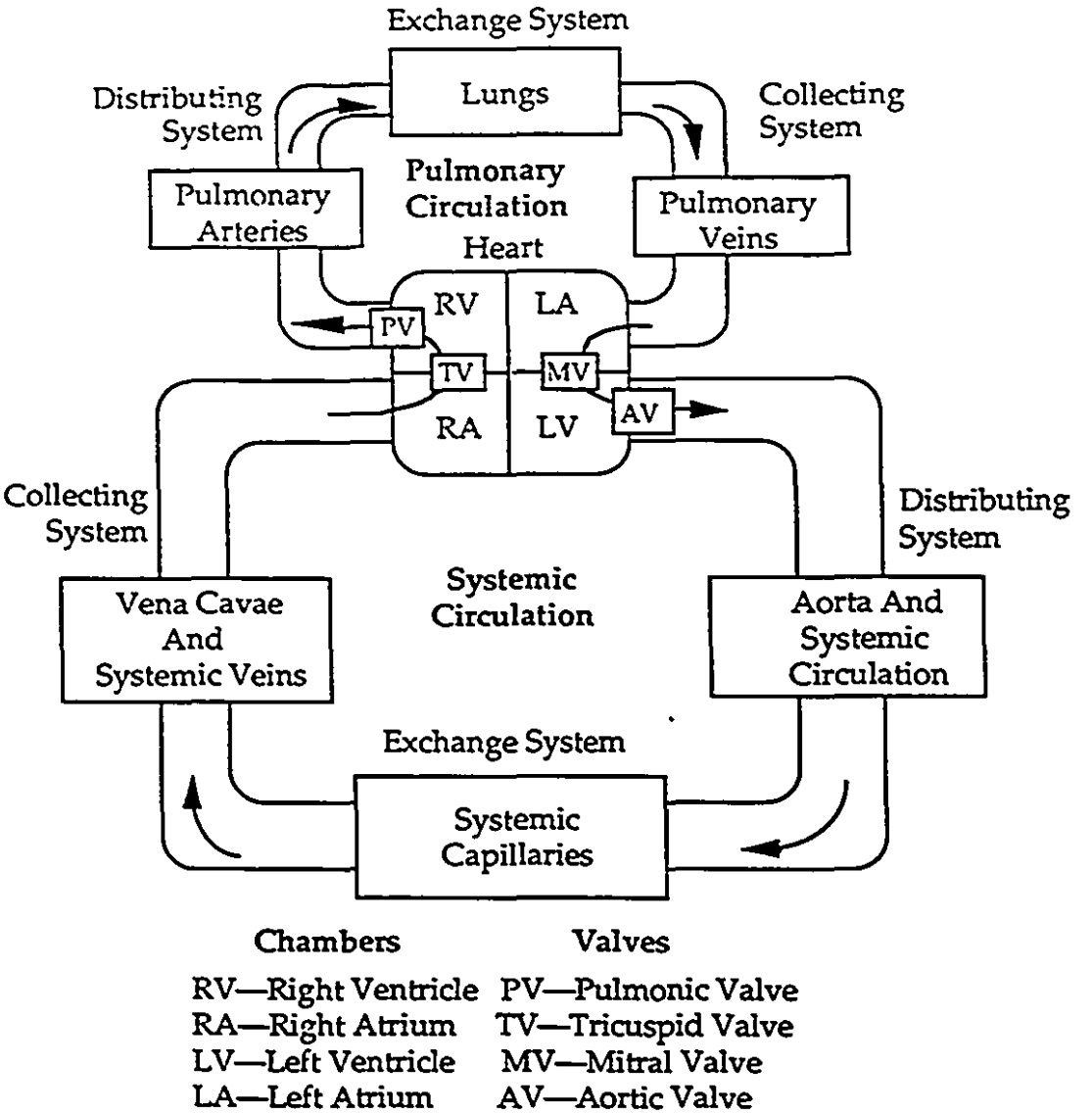


Figure 1: **Functional Division of the Circulatory System. Adapted from Smith and Kampine (1990, p. 3).**

Blood flows from the atria to the ventricles and then to the large arteries at a flow rate that is determined by pressure differences between chambers. We can characterize flow through the system in terms of pressure

gradients, which include an upstream or driving pressure, and an downstream or opposing pressure.

Each chamber of the heart has an inlet and outlet valve, which open and close in sequence according to the phases of the cardiac cycle. These valves function to prevent the back flow of blood into a chamber. The aortic and pulmonic valves are located at the exits of the right and left ventricles, respectively. The atrioventricular valves, the tricuspid on the right side and the mitral on the left side, permit blood to flow from the atria to the ventricles.

The Cardiac Cycle

The cardiac cycle represents a combination of mechanical, electrical, and valvular events whose interrelationship is complex (Smith & Kampine, 1990). At rest, the normal adult heart beats at a rate of about 70 beats per minute. The cardiac cycle is divided into a contraction phase, *systole*, and a relaxation phase, *diastole*. During systole the internal ventricular pressures rise rapidly to a peak; the ventricles then relax, and the internal pressures fall quickly to near zero followed immediately by the ventricular filling phase (diastole).

The most important physical characteristics of the circulation are *volume*, *pressure*, and *time*. Flow is defined as unit volume per unit time in a vessel. Pressure-volume relations define the basic qualities that distinguish important functional properties of arteries and veins. Pressure and flow refer to the moving stream of blood and in this way characterize the dynamics of circulation. The main changes in cardiac output are determined by the metabolic requirements of the body. The heart from a flow standpoint, plays a predominantly permissive role in regulating its output. The output of the

heart therefore, represents a balance between the venous return, which reflects the demand, and the ability of the heart to meet the demand.

Cardiac Output

Cardiac output is a product of two factors, *heart rate* and *stroke volume*. Heart rate is the number of contractions or heart beats per minute. Stroke volume is the amount of blood ejected by the ventricle during contraction. Stroke volume is determined by three factors: 1) *preload,* which refers to the initial stretch of the cardiac muscle before contraction; 2) *afterload,* which is the tension in the cardiac fibers after they begin to shorten; and 3) *contractility,* the functional state of the heart muscle that is defined by the rate and extent of shortening for a given afterload and preload.

One of the fundamental principles of cardiovascular physiology is the Frank-Starling[6] law of the heart. This states that the force or tension generated by the contracting muscle is dependent, within physiological limits, on the degree of stretch of the muscle prior to contraction (Smith & Kampine, 1990). This initial stretch is the preload stimulus. This would suggest that, during diastole, a greater inflow of blood will cause the ensuing contractions to be more forceful and greater shortening. However, the rate of shortening or the time needed to achieve peak tension is independent of the preload.

Afterload refers to the load the muscle must lift, after it begins to shorten. It is closely associated with aortic pressure. An increase in afterload, all other things being equal, will lower the speed of shortening, lessen the extent of muscle shortening and thus reduce the stroke volume. A

[6]The Frank-Starling law of the heart is most appropriately explained at the subcellular level, in terms of the constituents of cardiac muscle fiber. However, such a discussion is beyond the scope of the work discussed here.

healthy intact heart will react over time by increasing the preload to overcome a larger afterload.

Contractility is a function of extrinsic determinants, such as neurohormonal factors, chemical and pharmacological effects, and pathological effects (e.g., toxic substances). An increase in heart rate will also tend to increase contractility. An increase in contractility causes the muscle to contract faster and to achieve a higher peak tension.

Heart rate is primarily effected by changes in the autonomic nervous system. Heart rate effects cardiac output in two ways, (a) a mechanical or indirect effect output by virtue of the influence of rate on the length of diastole, and therefore on end-diastolic volume (volume remaining in a ventricle at the end of diastole) and stroke volume, and (b) the direct effect on contractility (Smith & Kampine, 1990). An increase in heart rate will produce concomitant increases in stroke volume, to a very high heart rate, where diastolic filling time is compromised.

Textbooks frequently use graphic illustrations to express functional relationships of cardiovascular physiology. It is convenient at this point to introduce a diagram referred to as a ventricular pressure-volume curve or loop. This diagram, as presented in Figure 2, illustrates the pressure-volume relationships at different times during the cardiac cycle.

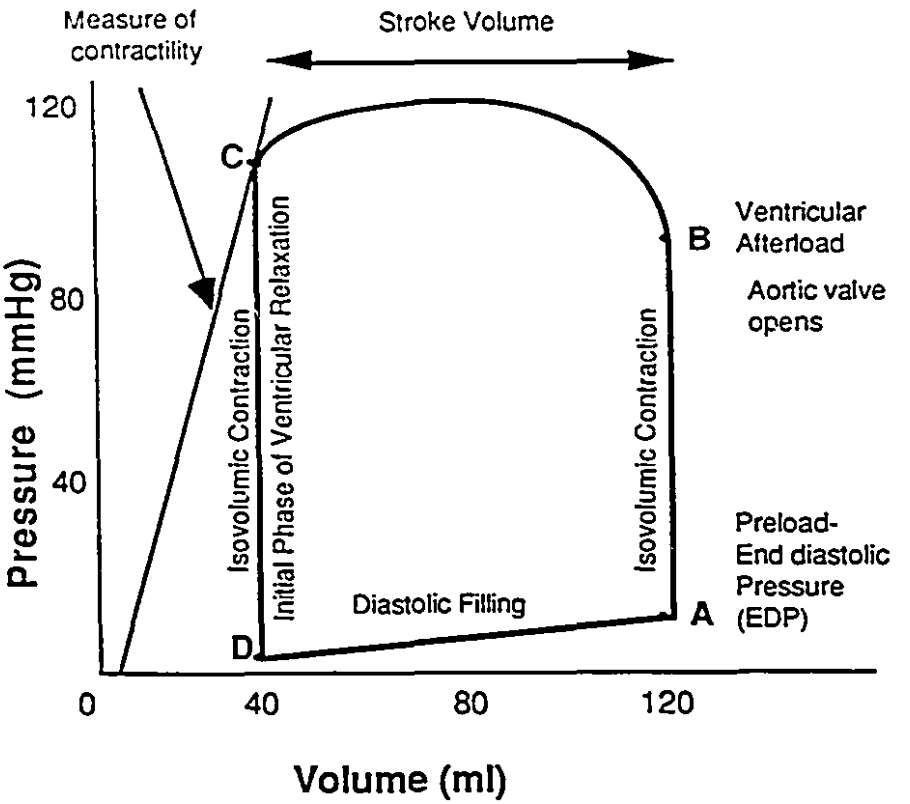


Figure 2. Ventricular pressure-volume diagram during a single cardiac cycle. Adapted from Green (1987, p. 46).

The area of the curve, the product of ventricular pressure and ventricular volume reflects the *work of the heart* (Green, 1987). Line segment D-A represents diastolic filling of the left ventricle. The ventricle begins to contract (point A) at an *end-diastolic pressure* that represents the preload of the ventricle. As the ventricular myocardium develops tension, ventricular pressure rises, resulting in the closing of the mitral valve (point A). Until the aortic valve opens (point B), the contraction is isovolumic—no volume leaves or gets in. Ejection begins at point B, and the ventricular pressure at this point (equal to the aortic pressure) represents ventricular afterload. Ejection continues until point C, where the ventricular pressure falls below aortic pressure. The volume ejected during the period between points B and C is

the stroke volume. *End-systolic volume* is the volume remaining once this ejection period has finished. The *ejection fraction* is the ratio of stroke volume to end-diastolic volume. As pressure in the left ventricle falls below that in the left atrium (point D), the period of diastolic filling begins and blood flows into the ventricle until point A. This inflow of blood stretches the ventricular wall and produces the preload for the following contraction and a new cardiac cycle.

An increase in preload, as reflected in greater pressure at point A, results in a larger stroke volume. An increase in afterload, as indicated by an increased aortic pressure, causes a diminished stroke volume and a larger end-systolic volume. The isovolumic contraction line, intersecting point C, reflects the maximum pressure one can get for any given preload. The slope of this line, represents the contractility of the ventricle. An increase in contractility changes the slope of the line decreasing the end-systolic volume and therefore, increasing stroke volume. An increase in heart rate, by definition, results in more rapid successions of cardiac cycles.

The term cardiac function is commonly used to describe the aspects of cardiac output under control of the heart pumps. The four previously described factors are referred to as the determinants of cardiac function. This distinction is useful to discriminate the contribution of the heart independent of venous return.

Venous Return

Venous return is determined primarily by *vascular compliance* and by *venous resistance.* Vascular compliance describes the properties of a vessel to distend to accommodate more blood volume per unit pressure. Vascular resistance is the forces opposing blood flow determined by the frictional loss

of energy due to geometric and viscous factors. That is, resistance is primarily determined by the radius of the vessel and the viscosity of the blood.[7]

Typically, the degree of compliance is measured by volume changes resulting from a distending pressure. The veins typically hold about 3 to 4 times more blood volume than the arteries (Smith & Kampine, 1990). However, the arterial compliance is approximately 1/30 of the venous compliance (Guyton, Jones, & Coleman, 1973). Therefore, the venous system has a storage capacity of up to 30 times more volume than the arterial system. The small veins and venules are much more compliant than the rest of the venous circuit.

An increase in resistance to flow is calculated as the ratio of driving pressure to flow. The primary determinant of resistance is the radius of the vessel. Since the vessels of the circulatory system are distensible, resistance can change at different time points. The pressure in the venous system is only about 1/10 as much as that of the arterial system. The pressure drop across the veins is relatively small compared to the arteries. However, venous resistance is a crucial determinant of venous return.

Mean systemic pressure is a pressure that reflects the forces that propel blood towards the right atrium. It is the driving pressure for venous return. Mean systemic pressure is the average of the filling pressures in all segments of the systemic circulation, when each of these pressures is weighted in proportion of the compliance of its respective segment. The mean systemic pressure can be experimentally measured when blood flow is

[7]Resistance applies mainly to steady flow systems. Flow in many of the vessels can be characterized as pulsatile. Impedance is the term used to refer to pulsatile resistance. However, since the phasic flow is not an issue in this study, the term resistance is used.

stopped. *Stressed volume* is the volume that stretches the elastic walls of vessels and thus produces pressure in the vasculature. It represents about 30% of the volume in the vasculature.[8] *Unstressed volume* is the volume contained within the compliant structure when the pressure within the compliant structure is zero. The mean systemic pressure is determined by the blood volume and the compliance of the peripheral circuit. Mean systemic pressure is essentially independent of cardiac function (Magder, 1992).

The downstream pressure for venous return is the pressure at the outflow to the venous system, which is the right atrial pressure. This is true under circumstances when the right atrial pressure exceeds the atmospheric pressure (Green, 1987). The atmospheric pressure is considered to be zero. When right atrial pressure falls to a subatmospheric value, the great veins collapse at their point of entry. This does not stop flow but limits further changes in flow, since the veins fluctuate between an open and closed position under these circumstances. Maximum venous return is achieved when the right atrial pressure is at zero mm Hg.

Integration of Venous Return and Cardiac Output

The circulatory system is a closed system and therefore the blood pumped out by the heart must inevitably return to the heart. Over time, cardiac output has to equal venous return. The right atrial pressure is not only the back pressure to the systemic circulation, but is also the simultaneous inflow pressure for the heart. The right atrial pressure is a function of the amount of blood returned to the heart and the pumping ability of the heart. The right atrial pressure couples cardiac function to the systemic circulation by directly affecting the pressure gradient for venous

[8]This percentage is an estimate based on animal models.

return (Green, 1987). An increase in cardiac function produces a decrease in right atrial pressure, which allows for a greater venous return.

Summary

This chapter presents an overview of concepts related to the mechanics of cardiovascular and circulatory physiology. The specific focus is on the determinants of cardiac output, the blood ejected by the heart per unit time, and venous return, the blood returning to the heart per unit time. In review, cardiac output is the product of heart rate and stroke volume. Stroke volume is determined by three factors, preload, afterload, and contractility. Venous return is governed primarily by vascular compliance, venous resistance, stressed volume, and right atrial pressure. Mean systemic pressure is a driving pressure for venous return. It is determined by vascular compliance and stressed volume. Right atrial pressure changes to accommodate increases in venous return and cardiac output. This mechanical coupling between heart and circuit is essential if cardiac output is to remain equal to venous return.

CHAPTER SIX

THEORETICAL AND METHODOLOGICAL RATIONALE

The purpose of this chapter is to provide a theoretical and methodological rationale for the study. The theoretical considerations draw liberally on ideas and findings discussed in chapters two, three, and four, to advance a framework for investigating conceptual understanding in a biomedical domain. The second part of the chapter attempts to situate the research within a particular investigative approach, and also focuses on methodological concerns raised by this study.

Theoretical Issues

The purpose of this research is to investigate conceptual understanding of complex biomedical concepts, specifically cardiac output and its regulation. We begin with the premise that biomedical knowledge provides a foundation for clinical medicine. The empirical evidence suggests that the role of basic science knowledge is complex and multi-dimensional. Its use is dependent on the specific medical domain, the difficulty of the problem (biomedical knowledge is not used to solve routine problems), and the subject's level of expertise. Furthermore, basic science knowledge can frustrate as well as facilitate medical problem solving.

It is important to recognize that biomedical knowledge is qualitatively different from clinical knowledge, embodying causal systems at several levels of abstraction (Schaffner, 1986; Blois, 1990). Each new level has different conceptual entities and a unique language of description. If we assume that biomedical knowledge is multi-leveled, then it is likely that different levels of knowledge have different kinds of correspondences with the clinical world.

For example, biochemistry and systems level circulatory physiology intersect with the domain of cardiology at different points and vary in their relevance to specific classes of cardiovascular disorders.

Research in medical AI suggests two general models concerning the structure and uses of biomedical knowledge (Chandrasakeran, et al., 1989). One is reflected in the retrieval of stored causal knowledge, which can be accessed and used to resolve ambiguities in the presentation of a clinical problem. The second model suggests an explicit representation of structure, function and behaviour. A physician could use this knowledge to account for a patient's condition by running the model and envisioning the consequences in terms of different behavioural outcomes.

The primary focus of this study is not on the use of biomedical knowledge in clinical contexts, rather it is on the understanding of a particular class of interrelated concepts. At present, there is no general theory of conceptual understanding of sufficient scope and precision to characterize a complex domain such as cardiovascular physiology. A cognitive theory would provide a basis for testing predictions concerning structures and processes that adheres to accepted cannons of scientific explanation (Patel & Groen, 1992). Since none is available, it is necessary to develop a framework to make the understanding of complex biomedical concepts a tractable research problem. Following Anderson (1983), a framework is a general pool of constructs for understanding a domain, but it is not tightly enough organized to constitute a predictive theory. Fortunately, research into cognitive science and science education provides a rich sample of constructs to develop a framework for investigating this rather complex problem.

The study of expertise seeks to understand and account for what distinguishes outstanding individuals in a domain from less outstanding individuals (Ericsson & Smith, 1991). The designation of expert can be a function of achieving a certain level of performance as exemplified by a certain ratings level in chess, or by virtue of being certified by a sanctioned licensing body, as is characteristic of medicine. In either case, the achievement of expertise requires about 10 years of full-time performance (Ericsson & Smith, 1991).

Contrasting experts with novices provides us with the opportunity to explore the aspects of performance that develop and result in enhanced problem-solving and reasoning abilities (Lesgold, 1984). In a given complex domain, expertise is not a monolithic entity, there is considerable variation. Expert knowledge is related to its functional utility. This explains why performance differences are observed between genetic counselors and academic geneticists (Smith, 1990), and between cardiology researchers and practitioners (Patel & Groen, 1991). In many disciplines, individuals can be expected to perform at an expert level, only within a very narrow context.

Empirical research into the nature of expertise has provided some dimensions for distinguishing students at different levels of ability (e.g., Thibodeau, et al., 1989). A consistent finding is that principles play a fundamental role in the organization of conceptual knowledge and procedural knowledge and distinguish the performance of both experts and superior students from novices or average students in different science domains. However, it is evident that the scope of application of basic science principles is not as evident in the practice of medicine, as in the applied physical domains.

Analogy is a construct that can provide us with some insight into the nature of conceptual understanding. Analogies are comparisons between relational structures from a familiar base domain into a typically less familiar target domain (Zook, 1991). It has been well-established that students experience formidable difficulties in accessing and mapping appropriate analogies. Nevertheless, spontaneous analogies play a central role in facilitating the structuring of new knowledge and the access of prior knowledge, particularly in domains of science. A characterization of spontaneous analogies can furnish us with considerable insight into the kinds of prior knowledge that a subject brings to understanding and explaining concepts.

The notion of mental model is well suited for describing how individuals form internal models of systems from interacting with causal systems (Norman, 1983). A subject's mental model provides predictive and explanatory capabilities of the function of à physical system. One can characterize subjects' models and elucidate aspects of subjects' representations that support different kinds of inference patterns, as well as, characterize flaws in terms of structure of a system or in terms of the inferences used to evaluate systemic processes.

Mental models are imperfect, imprecise, and result in predictable patterns of misunderstanding, which are commonly referred to as misconceptions. Misconceptions reflect strongly held beliefs or firmly entrenched conceptual knowledge and are pervasive in domains of physical and biological science. Misconceptions are rooted in both experience and formal learning, and in knowledge that is productive and functional in diverse contexts (Smith, diSessa, & Roschelle, 1992). These patterns of misunderstanding are not the result a single piece of wrong knowledge.

Rather they reflect reciprocating networks of knowledge elements, which in themselves can be correct, partially correct, or flawed (Feltovich, Spiro & Coulson, 1989). Therefore, to understand misconceptions, it is necessary to uncover the multiple contributing sources of knowledge that comprise them.

Many misconceptions are at least partially rooted in phenomenological experience. P-prims reflect an intuitive sense of mechanism and causality (diSessa, in press). They constitute a rich vocabulary through which people remember and interpret their experience. In the physical world, they are a reflection of the everyday experiences of observing the interaction of physical objects. In the biological sense, it is possible that an intuitive sense of causality is rooted in one's internal sense for fulfilling biological needs. This can manifest itself in terms of teleological reasoning, whereby biological systems are viewed, first and foremost, in terms of achieving a purpose, rather than as a mechanistic causal system.

In summary, the theory of expertise, the central role of analogy in cognition, the construct of causal models, and the issue of misconceptions constitute the elements of a theoretical framework for characterizing conceptual understanding. This framework needs to be instantiated within the context of epistemological assumptions and psychological evidence concerning the role of basic science knowledge in medicine and the essential character of biological knowledge.

Methodological Considerations

The goal of scientific research is to develop theories that enable the explanation, prediction, and control of events within a particular domain of inquiry. Experimental research in psychology and education is skewed towards the use of empirical methods for confirming or disconfirming

hypotheses that are assumed to be well formulated rather than toward building an adequate basis for theory development (diSessa, 1991). The empirical research presented here is primarily concerned with providing a foundation for theory development. That is not to say that the work is necessary exploratory. This research builds upon a rich database of previous empirical research, theories and well-established methods that have been widely used in domains of cognitive science and science education research. At present, there exists no single pre-packaged approach or technique for testing conceptual understanding of biomedical concepts. Therefore, the purpose of the research design is to assemble existing methods to investigate a rather difficult and complex research problem. The study of complex content domains, such as medicine, can be made tractable through integrated and multidisciplinary approaches (Kaufman & Patel, 1991; Patel, Evans, & Kaufman, 1989).

The methods incorporate elements of a verbal protocol problem-solving study and those employed in the clinical interview approach, common to many science learning studies. The use of verbal protocols, at this point in time, has been scientifically validated as a reliable and effective method for investigating cognitive issues (Ericsson & Simon, 1984). Similarly, the clinical interview has become a widely used approach in diverse areas of science and mathematics education research (Ginsburg, Kossan, Schwartz, & Swanson, 1983).

Although these approaches are well established, the content-based coding schemes and various other methods of analysis tend to be idiosyncratically tailored to accommodate the specifics of the problem at hand. In this respect, this study is no exception. This is especially true since the guidelines for problem-solving analyses are more clearly delineated than

they are for conceptual understanding. Therefore, there are specific issues of reliability and validity to be dealt with.

A recent issue of the Journal of the Learning Sciences (1992) addressed the issue of research methods for studying learning and provides some guidelines. In particular, Schoenfeld (1992) presents a discussion of *standards for novel methods*. These include the following (p. 181):

1. Establish the context, describing the issues to be addressed.
2. Describe the rationale for the method.
3. Describe the method in sufficient detail that readers who wish can apply the method.
4. Provide a body of data that is large enough to allow readers to (a) analyze it in their own terms, (b) employ the author's method to see if it produces the author's analyses.
5. Offer a methodological discussion that specifies the scope and limitations of the method, as well as the circumstances in which it can be profitably be used, and that treats the issues of reliability and validity.

The first standard, establishing the context, has been dealt with in some length in the first part of this chapter and in prior chapters. The third standard is discussed in some detail in the method section. Standards 4 and 5 are primarily dealt with in the results and discussion, and the conclusions chapters, respectively. Some of the pertinent methodological considerations related to points 4 and 5 are dealt with further on in this chapter. I wish to raise some issues pertaining to the rationale for the methods chosen.

The issue of conceptual understanding suggests that the experimental material be sufficiently rich and complex so a subject can demonstrate a

certain mastery of knowledge in circumstances that would preclude the possibility of rote-learning. Following Gelman and Greeno (1989), we believe that generativity and robustness are the cornerstones of understanding. Generativity is indicated by a flexible ability to use knowledge to achieve goals in a variety of task settings. Robustness is demonstrated by the ability to adapt knowledge to accommodate unusual and novel problems. The questions and problems used as stimulus materials in this study were developed with these guidelines in mind. In the study, concepts were presented in situations of varying complexity and converged on similar domain-specific themes.

The material for the study was developed following an epistemological analysis of the of the subject matter domain, in consultation with an expert cardiologist/physiologist and using various texts. As presented in the previous chapter, the mechanics of cardiovascular physiology is a content domain with a relatively well developed hierarchical schematic framework for partitioning knowledge. This makes it a viable subject matter for studying conceptual understanding while attaining a satisfactory level of content validity.

In the study, there is a greater emphasis on explanation than on problem-solving. The explanation paradigm is one that has received increasing attention in science learning (Chi et al., 1989) and in medicine (Feltovich & Barrows, 1984; Groen & Patel, 1991). While characterizations of problem-solving provide a most suitable paradigm for investigations of the development of procedural knowledge and strategies, explanation is more appropriate for investigating and drawing inferences about conceptual structure and understanding. Explanation has the added advantage of being most naturally expressed in a verbal manner. Thinking-aloud while

engaging in problem-solving can be an arduous task for some individuals. Protocols obtained in this manner can sometimes be discontinuous and fragmented.[9] Verbal explanation tends to be more fluid and coherent.

The granularity issue (grain-size) reflects a set of fundamental decisions concerning the design of the experiment (Brown, 1992). This issue can be decomposed into three interrelated components: the scope of the study sample; the depth and breadth of the subject matter under investigation; and the focus of the methods of analyses and the presentation of data.

To address the issue of changes in conceptual understanding along a dimension of expertise, it is important to include a continuum of subjects from novices through various stages of intermediate training to experts. Yet if the goal is to intensively study subjects understanding, and this necessitates the inclusion of multiple variables, then the sample size has to be reasonably small to make the study tractable. The study sample size falls along the continuum, with a microgenetic analysis of the performance a single subject along one end of the continuum (e.g., Schoenfeld, 1992) and a large scale statistical study at the other end of the continuum.

To investigate understanding in a complex domain, one must define the parameters of content inclusion. This choice is a particularly difficult one to make in a biomedical domain, since concepts tend to be interrelated across vertical dimensions (for example, between cellular and systems level physiology) and horizontal dimensions (for example, cardiovascular and pulmonary physiology have many points of overlap). On the other hand, certain concepts and principles (e.g., the Frank-Starling Law of the Heart) can be rather intricate and are by themselves worthy of an intensive focus.

[9]This can be an asset for a characterization of problem-solving, since problem-solving studies often wish to capture false starts, missteps, and sudden insight into a problem.

Again, this study, attempts to strike a balance between the issues of breadth and depth in the range of subject matter included.

The third component to the grain-size problem has to do with the methods of analysis. Verbal protocols are rich data-sources and can be analyzed in any number of ways. One approach is to attempt a relatively comprehensive and microscopic analysis of every single utterance or maneuver. At the other end of the spectrum, one can develop a highly specific coding scheme for looking at particular kinds of inference and classes of explanation.

The approach taken in this study tends to veer somewhat towards the latter macroscopic analysis. The questions and problems presented to subjects were developed for very specific purposes related to characterizing understanding in the domain under consideration. For every question and problem presented to subjects, there is a *reference model response* that represents the correct answer and the relevant particulars (e.g., causal mechanisms) related to the explanation. We are interested in the ways in which subjects converge and diverge from the reference model for a single question and across questions that focus on a particular theme. On the other hand, to meaningfully characterize understanding, it is important to consider the beliefs, ideas, and spontaneous analogies that subjects produce even if they appear tangential. Therefore, these factors are given consideration in the analysis of data.

The presentation of data is closely associated with the methods of analysis issue. Protocol analysis studies produce large bodies of data and large volumes of analyses. It is possible to present only a small subset of this analysis. For example, a content analysis of a set of subjects' responses may include a wide range of statements, while the tabular presentation of this

information needs to be much more focused and succinct. For every response and question, an effort is made to present an overview of how each subject responded along the continuum of expertise, as well as, a more in-depth analysis of individual responses.

When complex methods of protocol analysis are used, the nature of the actual responses are sometimes obscured. As suggested by Schoenfeld (standard 4), it is important to provide a body of data that is large enough to allow readers to inspect it on their own terms. This is achieved by the use of extensive excerpts from the subjects' protocols. Raw, unanalyzed excerpts can sometimes be used to amplify a point to a better effect than any complex analysis, particularly if the question or problem is tightly focused. Excerpts also provide an ideal complement in the sense that it can make the analysis more transparent.

CHAPTER SEVEN

METHOD

The design of this study was motivated by investigations of conceptual understanding in complex science domains. The questions and problems[10] were designed to fulfill some of the following requirements. These include: the use of open-ended questions to provide a glimpse of subjects' mental models; to cover the major concepts that arise in the analysis of the domain related to cardiac output, venous return, pressure-volume, and pressure-flow relationships; to require subjects to make predictions and furnish explanations; to provide problems that are likely to test the boundary conditions for the applicability of principles; and to include problems that the subjects are not likely to have encountered before to test generative processes and robustness of knowledge structures.

The experiment presents detailed analyses of subjects' conceptual understanding of cardiac output and the mechanics of circulatory physiology. The questions were designed and selected to strike a balance between breadth and depth. For example, the subcellular structures of myocardial muscle and biochemical processes involving electrophysiological events are certainly related to cardiac output. However, these topics were excluded because they would have extended the breadth of the study beyond a manageable limit. Similarly, particular concepts, such as the Frank-Starling Law of the heart could have been explored in much greater depth. Again, this would have necessitated extending the length of the study beyond

[10]Questions are direct and typically require shorter responses and less reasoning. Problems tend to be more complex, requiring more extended chains of reasoning or problem-solving and synthesis of knowledge. The distinction reflects a continuum, rather than sharp boundaries of demarcation.

reasonable confines. An additional consideration was that protocol analysis and interview type studies are very time-consuming and demanding. In this case, it was important to have sufficient number of subjects to sample a range of individual differences within and between different levels of expertise.

Subjects

The subjects consisted of 15 volunteers at several levels of training. The first level included one student who had completed a degree in biology and had applied to medical school. He is referred to in the study as the premedical student. Medical students (9 in total) were selected from of each of the four years of medical school at McGill University. These subjects include 3 first year students, and 2 in each of years 2, 3, and 4 of medical school. In addition, two residents, a physiologist, and two cardiologists were also included as subjects in the study. Each medical student had a bachelor's degree in a science discipline other than physiology.[11]

McGill University medical school has a conventional curriculum. For the first one and a half years, students take basic science courses (e.g., physiology, anatomy). This is followed by a "Link Period" or junior clerkship, in which they are exposed to courses in clinical sciences and commence their practical clinical training in hospitals. The Link Period lasts for 34 weeks. The final phase of medical school, lasting for 58 weeks, is known as the Clerkship. The clinical clerk is a regular member of a clinical teaching unit and assumes considerable responsibility for patient care.

[11]Medical students with degrees in physiology were not included in the study because they would have had considerably more exposure to the subject matter. In a given year, physiology students constitute among 20-25% of the students entering medical school at McGill University. Students without a Bachelor of Science degree were also excluded because they were not likely to have had sufficient familiarity with the content domain.

The three first year students were tested before beginning the cardiovascular section of a physiology course. One of these subjects had a Bachelor's degree in general science, a second student had a degree in physics and biomechanical engineering, and a third subject had a degree in physics and chemistry.

Second year students had completed most of their basic science courses. These students had been exposed to clinical science teaching, but had not begun clinical training. Both second year students had completed degrees in biochemistry.

Third year students had started in the junior clerkship program, which represents the beginning of applied clinical training. Neither student had as yet begun the core general medicine clerkship rotation. This rotation is typically considered to be the most important since it exposes students to a wide range of clinical problems and provides ample opportunity to acquire various investigative skills. One of the third year students had a degree in anatomy and the second one had a degree in electrical engineering.

Fourth year students had completed most of their clerkship training, including their rotations in general medicine. These students would have considerably more clinical experience than the third year students. At the time of the study, they were about 6-8 months from finishing medical school. One of these students had a bachelor's degree in microbiology and the second student had completed a masters in biochemistry.

The two senior residents were in their fourth year of residency and were in their first year of the Cardiology specialization program at McGill affiliated teaching hospitals. The experts consisted of one physiologist, who was an instructor at the medical school, a cardiologist in private practice, and an academic cardiologist, who divides his time between research and hospital

practice. All the students from second year on and one of the residents had, taken the same cardiovascular physiology course that was taught by the same instructor at McGill University.

Material

The materials for the study consisted of 49 stimulus questions and problems presented on cue cards. The experiment was divided into 4 sections. Each section presents an increment in inferential complexity and in the kinds of knowledge required to accurately respond to the questions. The **first section** included 2 open-ended questions, which were designed to elicit subjects' beliefs about the factors that affect cardiac output and venous return. Subjects were free to respond to these two questions in any way that they saw fit.

The **second section** consisted of 25 basic physiology questions that asked about specific factors influencing cardiac output, venous return, pressure-volume, and pressure-flow relationships. These questions required subjects to make predictions, provide explanations, and to respond to general knowledge queries. In this section, the questions demanded a response either in the form of the retrieval of a piece of knowledge from memory or a short chain of reasoning (1-3 inferences).

Seventeen of the 25 questions required predictions. Since several of the questions required multiple predictions, there was a total of 27 predictions to be made. These questions required that the subjects predict the effects of changes (increase or decrease) in particular factors (e.g., an increase in preload) on certain measures (e.g., end-diastolic volume). The possible responses are the dependent variable *increases*, *decreases*, *doesn't change*. A fourth possibility is that there is a temporal component involved,

in which there is no initial change and then there is a subsequent increase or decrease. Each of the prediction questions also asked for explanations. The eight general knowledge questions asked about facts or were intended to elicit subjects' definitions of concepts and principles.

The **third section** included 10 questions that relate cardiac factors to venous return and venous return factors to cardiac output. For example, one question asked "How does a marked increase in arterial resistance affect venous return and cardiac output?" These questions were typically more complex than the questions in the first section, and required longer chains of inferences and integration of different concepts. They were intended to assess the degree to which subjects have coherent models of the circulatory system. Nine out of 10 questions required predictions, for a total of 12 predictions.

The **fourth** and final section included 12 problems which present situations in which these concepts are to be applied. Two of the problems required subjects to reason in applied physiology contexts (e.g., exercise). Seven problems presented pathophysiological descriptions. These problems described the cardiovascular pathophysiological manifestations of medical disorders, such as, hyperthyroidism or other perturbations in structure, (e.g., a hemorrhage). Subjects were informed of the concomitant effects on cardiac output and venous return and were asked to provide a causal explanation accounting for the increase or decrease in blood flow.[12] Three of these problems presented brief clinical situations and were designed to test subjects' abilities to apply these concepts to diagnostic and therapeutic

[12]Subjects were informed of these effects because it was expected that many of them (particularly medical students) would not be sufficiently familiar with these conditions. Moreover, the intent of these problems was to investigate subjects' abilities to apply specific concepts rather than to test their knowledge of particular medical conditions.

situations. These questions were designed to assess whether the subjects could recognize the conditions of applicability and use these concepts in context. In this section, 3 questions asked for a total of 4 predictions. Across all sections of the study, there were 29 questions, requiring a total of 43 predictions.

To respond to problems in section 4, there are a set of abstract procedures to follow.[13] These are as follows: 1) Determine if the problem is predominantly affecting cardiac factors, venous return factors or both; 2) Identify the particular factor(s) (e.g., preload) and their direction of change (increase, decrease); 3) If necessary, construct a mechanistic account (e.g., changes in pressure-volume relationships in the right heart); 4) If necessary, propagate the state changes through the relevant parts of the circulatory system; and 5) Generate an explanation that accounts for the end-state (e.g., increased cardiac output) described in the problem statement.

Procedure

The experiment combined the methods of a problem-solving approach with a focused clinical interview approach common in science education. Subjects were presented with a series of questions and problems on cue cards, one at a time. They were asked to read the question out loud, and "talk-aloud", and to answer the questions and problems as completely as possible. They were probed for further information when their responses did not fully answer the question or when their responses did not address the particular issue. Probes were also used to further explore subjects' conceptions. When subjects could not remember the meaning of a particular term, they were

[13]Subjects were not provided with any such guidelines. These procedures reflect the demand characteristics of the problems.

provided with a hint. For example, if a subject could not recall the Frank-Starling Law of the heart, they were told to "think in terms of length-tension and its relationship to pressure-volume".

The questions were presented in two different orders of randomization; and were randomized within each of the last three sections. The order of presentation for the sections was kept constant. The subjects were provided with a pen and a pad of paper and were free to draw diagrams, make notes or work through problems, if they so desired. There was no time limit imposed on subjects' responses. However, when subjects perseverated on a problem without any success, it was suggested to them that they should move on to the next one. The subjects were tested individually and each session was audio taped.

Pilot testing revealed that subjects experienced difficulty calibrating their judgments. In particular, subjects could not easily generate predictions in the second part of the study (basic physiology section), without an external reference point. The subjects were provided with a diagram, illustrated in Figure 3, depicting a ventricular pressure-volume curve. This diagram is nearly identical to the one presented in Figure 2. However, much of the annotation was removed so that subjects would not be provided with additional clues. Subjects were informed that this was to serve as a reference or a memory aid and that they were free to use the diagram in way they wished.

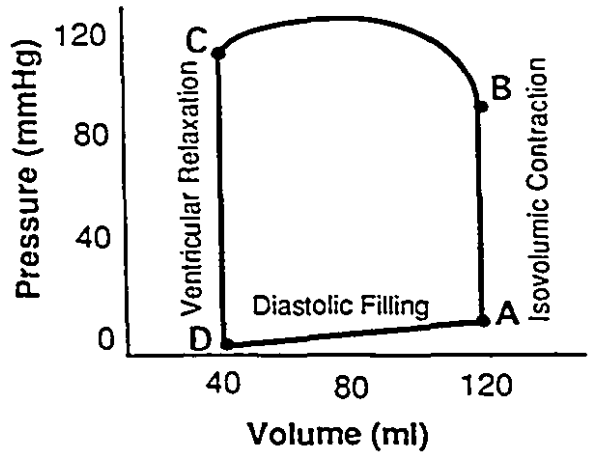


Figure 3: Ventricular pressure-volume diagram presented to subjects.

Methods of Analysis

The focus of the research is both on individual subjects and on groups of subjects. Therefore, the methods of analysis incorporate both qualitative and quantitative measures. The analyses include various methods used in verbal protocol analysis in cognitive science (Ericsson & Simon, 1984) and methods of analysis common to science education (Chaiklin, 1985).

The subjects' protocols were transcribed literally. Nonverbal vocalizations (e.g., ah or uhm) and pauses exceeding a few seconds were noted. The transcripts were divided by question and each answer was segmented. The segments consisted of manageable units, such as phrases, short sentences or ideas (Ericsson & Simon, 1984). The segmentation process was performed to facilitate further analysis, rather than as a formal method of analysis in which inferences about performance are to be derived.

For each problem, a *reference* response, describing the correct answer, was prepared with the assistance of a consulting expert cardiologist and physiologist. This reference response was used to assess the answers of each subject. Predictions can be characterized as correct or incorrect and

percentage of correct responses could be tabulated by category for each subject. Explanations provided more insight into subjects' conceptual understanding.

A content analysis was performed for the answer to each question. There was a different criteria for coding each question. The responses were coded: for specific mention of concepts, (for example if the subject referred to venous resistance as a causal agent); whether the components of a correct response was produced, as determined by the reference response; and original content produced by the subjects were used to form additional coding categories. In others, the responses were coded according to specific predetermined criteria and unanticipated content produced by subjects. Patterns of subjects responding across questions provided us with information concerning gaps in knowledge, misconceptions, and biases.

Selected responses were coded independently by two individuals. Much of the coding of responses involve recognizing literal or paraphrased statements in a protocol. In general, we were able to achieve considerable inter-rater reliability.

Two formal methods of analysis were used to analyze subjects' explanations. These are *semantic networks* and *functional dependency diagrams*. Semantic network, were used to represent subjects' responses to individual questions. Reference responses were also coded as semantic networks. This provides a more precise means for examining and comparing chains of inferences generated by subjects' with those of the reference response. *Functional dependency* diagrams are a method used to characterize subjects' knowledge of causal relations between concepts across questions. Similarly, a reference model functional dependency diagram can be used as a

benchmark for evaluating subjects' responses. Both methods are described in detail below.

Semantic Networks

A semantic network is a type of data structure for representing knowledge and graphically expressing natural language concepts (Sowa, 1984). Semantic networks are directed, labeled graph structure in which the basic elements are nodes representing concepts and arcs (links) representing relations (Rumelhart & Norman, 1985). The network is defined by a concatenation of concept-relation-concept triples (Frederiksen, 1975). Specific types of semantic networks place restrictions on the type of concepts and types of relations that are permissible.

The semantic networks adapted for medicine by Patel & Groen (1986) and described in detail in Groen and Patel (1988), are based on Frederiksen's propositional grammar (1975). There is a need to make a distinction between levels of representation. The propositional level of representation is an intermediate level of representation, above the syntactic or lexical level, that expresses fine-grained semantic structures encoded into texts. The conceptual level is a higher level of representation that corresponds to an individual's declarative knowledge structures and need not correspond to the literal propositional content of a subject's protocol. Semantic networks represent a conceptual-level formalism, designed to capture a subject's cognitive models of phenomena in a particular knowledge domain.[14] They also have the advantage of being similar in kind to the methods of

[14]In theory, semantic and causal networks can express exactly the same information as a propositional analysis (Rumelhart & Norman, 1985). However, in the analysis of causal explanations, it is convenient to omit predicate descriptors, such as, *agent* and *recipient*, and *attributes* that are not deemed as relevant.

representation used by Gentner (1983) and the method of concept maps, commonly used in science education (White, 1985).

Causal explanations in biomedicine are typically process descriptions of a set of discrete events ordered in time and space. The primary relations of interest in these networks are binary dependency relations, specifically *causal*, *conditional*, and *logical* relations—and, *alternating or*, and *exclusive or* relations. It is important to note that these relations reflect attributions made by subjects. For example a causal relation merely reflects attributed causality and does not reflect causality in the logical or classical sense of the term.

Semantic networks are applicable to any domain of knowledge. The claim is that this formalism has sufficient generality to represent any phenomena within the domain of medicine. Medicine or cardiovascular physiology, like any other domain of knowledge, has a specific typology of objects and relations. In addition, there are specific lexical phrases common to the domain of medicine, that refer to specific types of relations (e.g., *secondary to* implies *causation*).

Algebraic relations, identifying relations and categorical relations are common to semantic networks. Uncertainty in relations, can be represented by modal qualifiers, and truth values can be indicated when they deviate from the default truth value—*positive*. Relations may be further described within the context of a temporal order system by specifying tense and aspect operators (e.g., whether an action is continuing, has stopped or is repeating itself). Concept nodes can be characterized according to specific object types. For example, objects may represent *structures*, *functions* or *events* undergoing a process of change.

The typology of relations used in semantic networks included in this paper are: **ACT**–engages in an action or process, as in an agent, such as the heart initiating an action or physical process like ejecting blood. **AND:** –the logical connective indicating conjunction, **CAT:** –*A* is a member or an example of category *B*. For example, preload is a member of the category determinants of cardiac function. **CAU**–causality; **COND**–directional conditionality; ***DIR***–direction, as in the direction of flow of blood from the aorta. **DEG:** —a qualification in terms of a degree or numerically specified change, as in a *dramatic* increase in venous resistance or in the heart rate will increase *threefold*; ***DUR*** —A specified interval of elapsed time (e.g., 30 seconds); **EQUIV**–equivalent in some property, For example, when cardiac output and venous return are in equilibrium, the equivalent amount of blood ejected from the heart, returns to the heart. **GOAL:** –The goal or purpose of an agent or action, as in the purpose of cardiac output is to supply the tissues of the body with oxygen. **IDENT**–identity relation, For example venous return is (identical to) the blood flow returning to the heart. **IF**–enabling precondition, This is used when there are sets of preconditions to be satisfied before a process is possible. **LOC**–location, **OBJ:** –the object of an action. **OR-ALT:**— Alternating disjunction, as in A and/or B. **OR-EXCL:** —Exclusive disjunction— either A or B. ***POSS*** —possess, as in the veins have valves. **RSLT**–result of an action; **TEM: ORD:** –Order in time, as in event A precedes event B. Arrows between nodes indicate directionality. Horizontal arrows attached to nodes indicate an increase or a decrease of the variable represented in the node. Sometimes a long chain of inferences is used as a premise for a particular argument or a set of arguments. When this circumstance arises a box is placed around the entire chain to denote that is grouped as an antecedent or consequent.

Functional Dependency Diagrams

A *functional dependency diagram* was generated to represent the set of entities and relations involved in the problem set (Figure 4). This method allows us to characterize aspects of their mental models' of the mechanics of the cardiovascular system and diagnose conceptual errors. This representation is similar to ones used in qualitative simulation of physical systems (e.g., Kuipers & Kassirer, 1984). This method of representation is most conceptually similar to what Forbus and Gentner (1986) refer to as qualitative proportionalities[15]. Qualitative proportionalities express functional dependencies between quantities. They express partial information, since the exact nature of the function relating parameters is not known.

The directional functional dependencies between the primary variables included in the study is illustrated in a reference functional dependency diagram in Figure 4. The variables represent quantities, that when changed, can initiate a process that will effect other variables in predictable ways. A variable can exert a positive, negative or neutral influence on another variable. If an independent variable increases and exerts a positive influence, then the dependent measure will be more likely to increase. Conversely, if the relationship is negative, the dependent variable will be more likely to decrease. The diagram contains 20 variables and 30 functional relationships.

[15]Forbus and Gentner represent qualitative proportionalities in a schema-type formalism.

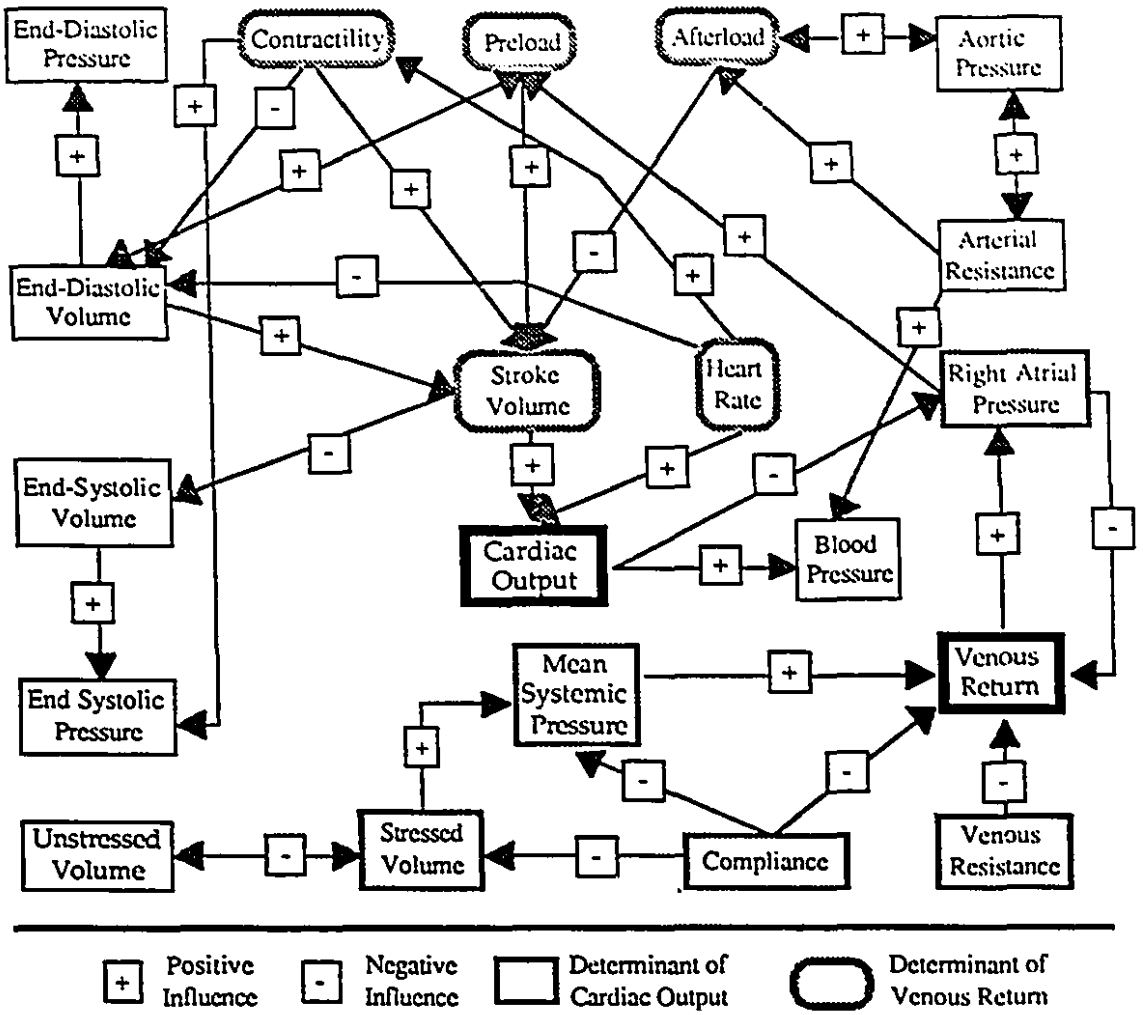


Figure 4. Reference functional dependency diagram.

There are other relationships that can be deduced from the network. However, temporal relations or enabling conditions that can delimit the circumstances when an influence can be exerted are not explicitly represented. For example, if cardiac output is at its maximum, it will not be influenced by factors that would tend to otherwise increase it. The network also does not explicate how to resolve ambiguities from conflicting influences.

Functional dependency diagrams were generated for each subject. This analysis was used represent their beliefs concerning relationships

between variables, and was then contrasted with the reference functional dependency diagram. The networks reflect subject's explanations and predictions across all questions and problems. Although, the responses to the applied problems were not very amenable to this type of analysis and were excluded. When a functional relationship is coded, this reflects a stable pattern of responding on the part of the subject. In other words, the subject repeatedly judged a variable to exert a particular influence over another variable in a consistent manner. The diagram can also be used to denote that a subject did not understand a specific concept or was uncertain about a functional relationship between two variables.

Hypotheses

On the basis of previous research, the nature of the content domain, and the structure of this study, It is possible to formulate the following hypotheses:

1. Subjects with greater degrees of expertise, and students more advanced in their training should exhibit greater degrees of generativity and robustness in applying domain-specific concepts and provide superior explanations and more accurate predictions.

2. The differences between the most novice subjects, including the premedical student and the first year medical students, and the other subjects should be most prominent, because basic science training is completed by the end of second year and these novice subjects have yet to take the cardiovascular physiology course.

3. Most students should respond better to the basic physiology questions than to the more applied problems. Physicians would be more likely to respond better to the applied clinical problems.

4. Students entering medical school would be expected to have preconceptions or naive theories about the structure and function of the heart, and pressure-volume and pressure-flow relationships.

5. Certain misconceptions are likely to be rooted in experience. In the case of biomedical concepts, it is possible that this will manifest itself in terms of teleological reasoning.

6. Misconceptions are more likely to arise in problems of greater complexity where standard solution strategies are not likely to be as effective.

7. Certain misconceptions are likely to carry over into the later years of medical school and perhaps even into clinical practice.

8. Determinants of venous return are not likely to be as well understood as determinants of cardiac function.[16]

9. Biases will be prominent in the thinking of many of the subjects, including both students and physicians. In particular, we propose that venous return factors will receive less consideration than cardiac function concepts in response to problems.

10. Spontaneous analogies will be used to provide explanations for various questions and problems.

11. Students are likely to use analogies most effectively, when there exists a surface and relational similarity between the source and target domains. The more advanced subjects may be able to better use more abstract analogies.

12. Expert cardiologists and the expert physiologist may possess fundamentally different models of conceptual understanding.

This study addresses these hypotheses, as well as others, that emerge in the analysis and characterization of conceptual understanding of cardiac output and its regulation. It is this characterization that reflects the studies original contribution to knowledge.

[16]Hypothesis 8, and the aspect of hypotheses 9 related to venous return were suggested by the instructor who taught the cardiovascular physiology course.

CHAPTER EIGHT

RESULTS AND DISCUSSION

This chapter is organized first, by the four sections of the study, and then grouped, by concept (e.g., preload) and by question. The first section presents the results of subjects' responses to the two open-ended questions. This is followed by the presentation of an analysis of the predictive accuracy of subjects across question categories. The subsequent section presents the results of analyses pertaining to basic concepts related to pressure-volume and pressure-flow relationships. The two following divisions include analyses pertaining to subjects' understandings of the core concepts related to cardiac output and venous return. The following section presents the results of the third part of the study; an analysis of subjects' responses to the integration questions. The subjects' understanding of cardiac output and its regulation, as reflected in their responses to the questions up to this point, are evaluated in the context of a characterization of their mental models. The final part of the chapter presents analyses of subjects' responses to the applied problems. Because of the length and complexity of the study, a discussion of the results is included in each section.

Each section is organized according to groupings of specific questions and problems. For example, questions that pertain to afterload are presented together. An explanation and reference response is presented for each question. The results are first presented in a tabular form, which reflects the coding of subjects' response for the correct answer, as well as, for original content. Following an evaluation of the overall pattern of responses, there is a discussion of the effects of expertise on conceptual understanding. This is followed by an evaluation of selected individual's responses to a single

question or to sets of questions. The focus in this analysis is on both particular strengths and weaknesses exhibited by subjects. Specifically, we are interested in characterizing, misconceptions, errors in problem representation or analysis, the spontaneous use of analogies, and strategies for synthesizing information.

The exposition of these results can take the form of the presentation of excerpts from subjects' protocols and/or the use of semantic networks. The choice between these two forms of presentation is made on the basis of clarity of expression, that is, which format is best suited for conveying a particular point. Excerpts are a more effective means for expressing linear sequences of thought, including the development and justification of an argument. Semantic networks are most effective in conveying subjects' explanation of a particular aspect of a system (e.g., diastolic-filling of the left ventricle) undergoing a process of change. This format provides us with an opportunity for characterizing a subject's causal model of the system.

The sections of considerable length are followed by a summary section. A summary of the results from the entire study is presented at the beginning of the conclusion and implications chapter.

The average total time taken for each subject to complete the experiment was 79.8 minutes (sd=26.3). It is convenient to group the subjects into three groups, the premedical student, the medical students, and the advanced group. The premedical student needed about 150 minutes to complete the study. The medical students took, on average, considerably more time, 87.6 minutes (sd=28.8) than the advanced subjects (the two residents, the physiologists, and the two cardiologists) who needed a mean time of 64.2 minutes (sd=9.9). The range of times taken to complete the experiment for students was between the 61 minutes needed by a fourth year

student (4.1) and the 124 minutes required by a first year student (1.3). The range for the advanced group was between the 57 minutes required by a resident (R1) and the cardiologist practitioner to the 79 minutes taken by the other resident (R2). These measures of time are not considered in any analysis of the study, rather they provide a general sense of the length of the experiment.

Open-Ended Questions

This section discusses the results of subjects' responses to the first two questions of the study. These questions were phrased in a manner that allowed subjects' considerable freedom to discuss any aspect of the subject matter. There is not necessarily a single correct answer to these questions. The questions are as follows:

G1. Discuss the cardiovascular factors that regulate cardiac output.

G2. Discuss the factors that affect blood returning to the heart.

Factors that Regulate Cardiac Output

The first question asked about the cardiovascular factors that regulate cardiac output. Cardiac output is the volume of blood ejected from the heart per unit time. The only presupposition is that there are particular mechanistic factors that determine cardiac output. This question provides some insight into the organization of subjects' knowledge and their theories of cardiovascular and circulatory physiology. It also permits a characterization of certain biases and conceptual errors.

It was anticipated that many subjects would focus on the determinants of cardiac function, such as, *heart rate* and *stroke volume*. Subjects could also

expand on the determinants of stroke volume (e.g., preload) and heart rate (e.g., sympathetic stimulation). Table 2 presents a list of these factors coded against subjects' responses. The table also lists the other commonly cited factors.

There is considerable variation in subjects' responses. Subjects most frequently referred to the primary determinants of cardiac function as factors that regulate cardiac output. In particular, 12 out of 15 subjects mentioned heart rate. Only two of the first year students and the academic cardiologist did not mention heart rate as a causal factor. Eight subjects discussed the determinants of heart rate (sympathetic, parasympathetic, or autonomic).

Table 2

Subjects' Responses to Factors that Regulate Cardiac Output (G1).

Factors	P	1.1	1.2	1.3	2.1	2.2	3.1	3.2	4.1	4.2	R1	R2	Ph	CP	AC	Totals
Stroke volume					1	1	1	1		1	1	1	1			8
Heart rate	1	1			1	1	1	1	1	1	1	1	1	1		12
Preload					1	1	1	1		1		1	1	1		8
Afterload		1			1	1		1				1	1	1	1	8
Contractility				1	1	1	1			1		1	1	1		8
Neurohumoral Factors				1		1	1	1		1	1	1	1			8
Venous return								1			1		1		1	4
Resistance/ impedance	1	1	1					1				1		1	1	7
Blood pressure	1	1							1					1		4
Totals	3	4	1	2	5	6	5	7	2	5	4	7	7	6	3	67

P = Premedical Student,
Medical students are referred to by year, and then by subject.
For example, 2.1 = Second year medical student, subject 1
R = Resident, Ph = Physiologist, CP = Cardiologist Practitioner,
AC = Academic Cardiologist.
This legend is applicable to all subsequent Tables.

Stroke volume, the volume of blood ejected from the heart on a single beat, was identified as a regulating factor by 8 subjects. Most subjects also

alluded to the determinants of stroke volume and four subjects, who did not mention stroke volume, discussed one or more of its determinants. Four subjects identified venous return as a regulating factor, reflecting a belief that circuit factors play a significant role in changes in cardiac output. Six subjects suggested that impedance or resistance is a regulating factor. Aortic and arterial impedance or resistance is closely associated with afterload. Four subjects erroneously identified blood pressure as a regulating factor. This conceptual error is discussed further on.

The premedical student and the first year medical students could not identify many of the relevant factors. They also lacked a process vocabulary for expressing their thoughts. For example, heart rate was referred to as "the amount of times it would beat" by a first year student (1.1) and contractility was alluded to as the "electrical stimulation of the heart muscle" by another first year student (1.3). The premedical student and first year students (in particular subject 1.1) tended to use teleological descriptions rather than mechanistic ones. This kind of description was particularly evident in both of the first two questions.

Seven subjects including the two second year and the two third year students, a fourth year student (4.2), a resident, and the physiologist, focused on the primary determinants of cardiac function. The other resident did not elaborate on the determinants of stroke volume, and a cardiologist did not explicitly mention stroke volume but described its determinant. A fourth year student discussed resistance, heart rate, and blood pressure as causal factors. The academic cardiologists articulated a belief that is indicated in an excerpt from his response:

Excerpt from the academic cardiologist

8. Peripheral factors tend to regulate cardiac output
9. more than central factors,
10. except that the heart may fail under certain circumstances
11. and not be able to increase cardiac output
12. beyond a certain amount.

This suggests that the circuit factors (factors like resistance and compliance, that affect the arterial and venous vessels) predominate over cardiac function mechanism except under conditions in which the heart is unable to respond to blood flow. This an increasingly accepted view in the discipline of cardiology.

The most complete and coherent responses were offered by a resident (R2) and the physiologist. A semantic network representation of the resident's response is presented in Figure 5.

The resident stated that cardiac output is a product of heart rate and stroke volume. He proceeded to break down the factors into constituent components. For example, stroke volume is affected by changes in the loading conditions and changes in the contractile properties of the vessels. Preload and afterload are categories of loading conditions. Preload is determined by filling pressures and afterload is associated with the opposition or impedance to the process in which the ventricle ejects blood. The subject's representation is suggestive of a very coherent and well-organized knowledge structure.

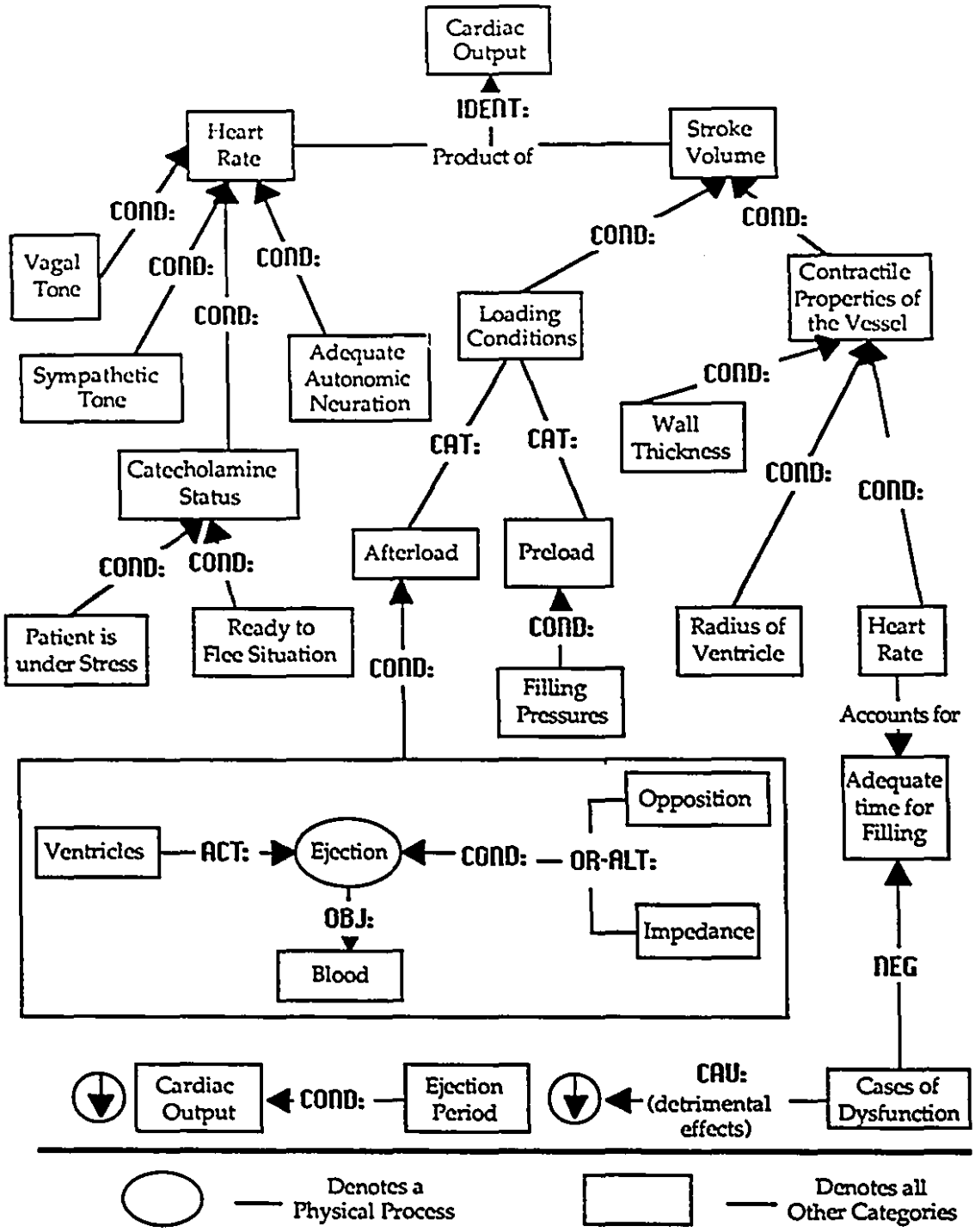


Figure 5. Semantic Network Representation of Response by a Resident (R2) to Question G1.

Conceptual errors or misconceptions were expressed by some subjects in response to the first question. Several errors are evident in the response of the premedical student.

Excerpt from the premedical student

1. Cardiovascular factors,
2. I believe one of them will be blood pressure.
3. I'm just trying to remember what they are.
4. One of them will be blood pressure,
5. which is dependent upon how just how narrow the arteries are.
6. If the arteries are very much clogged up,
7. then I think the heart would have to beat that much faster,
8. that much harder.
9. Therefore, cardiac output would be greater.
10. I believe also, the amount of oxygen that the body requires at that particular time.
11. So the tissues need more oxygen,
12. I would imagine that the cardiac output would have to be greater.

It is evident in his use of terminology that he favours a teleological mode of explanation. The heart is viewed as a device that responds to meet a need. Two examples are, the heart "would have to beat that much faster" and the "tissues need more oxygen". There is nothing inherently wrong with these statements or more generally, with teleological explanations if they do not obscure an understanding of the mechanisms.

This subject expressed two clear conceptual errors. The first has to do with the fact that *blood pressure* is a determinant of cardiac output. In fact, blood pressure is a product of cardiac output and arterial resistance. The blood pressure is a component of the afterload to left ventricular ejection. The third error is reflected in the belief that cardiac output would increase when "the arteries are very much clogged up". The assumption is that the heart would perceive a need and respond both in a massive way and with a

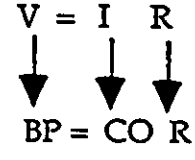
clear goal in mind. This counter-intuitive notion accentuates the potential pitfalls of teleological reasoning.

Three other subjects also identified blood pressure as a causal factor in regulating cardiac output, including a first year (1.1) and a fourth year student (4.1), and the cardiologist practitioner. The cardiologists' response, was the most interesting. It is presented below.

Excerpt from cardiologist (CP)

1. I always go back to Ohm's Law,
2. V = IR,
3. just because it's one equation to remember,
4. and then I substitute in the appropriate cardiovascular things
5. and uh, so voltage would be a pressure drop,
6. resistance is still resistance,
7. and times your cardiac output, uh
8. so then you could see that cardiac output
9. is a function of blood pressure
10. and resistance,
11. peripheral resistance.
12. You can also look at cardiac output from a Starling point of view,
13. where it depends on the preload,
14. the afterload,
15. which again is a function of your blood pressure
16. and also the wall stress,
17. as well as the contractility of your ventricle.

The physician used two sources of knowledge to retrieve the factors that regulate cardiac output: Ohms' Law and Starling's Law. He draws an analogy between a well-known physical principle Ohm's Law (V = IR) and the regulation of cardiac output. He then proceeded to map the equations, substituting for each of the variables in Ohm's Law. The mapping is essentially correct and he is left with the equation:



Changes in blood pressure (BP) are mapped onto changes in voltage (V), cardiac output (CO) is mapped onto current (I), and resistance (R) remains the same. Therefore, blood pressure is equal to cardiac output times resistance. This is the correct equation for blood pressure. However, the focal issue is what regulates cardiac output. He reverses this equation, and the dependent and independent variables, to suggest that cardiac output is dependent on blood pressure. Blood pressure is not a regulating factor, rather it is a product of cardiac output. The same conceptual error is reiterated in the context of Starling's Law. One can use the equation and perform an algebraic substitution to solve for cardiac output, if one already has a measure of blood pressure and resistance. However, this expression cannot account for the direction of the causal relationship.

Factors that Affect Blood Returning to the Heart

The second question asked about the factors affecting blood returning to the heart. The primary focus of the question is on factors that affect venous return. Although subjects could have interpreted it to mean factors that concern the entire peripheral circuit, including the arterial branch, as well as, the venous branch.

The subjects' protocols were coded for content specific responses related to venous return and other response types. As in the previous question, it was expected that subjects would focus on certain factors. Specifically, we anticipated that subjects would identify determinants of venous return such as, venous resistance, compliance, mean systemic pressure, right atrial pressure, and stressed volume. Subjects' coded responses are presented in Table 3.

Table 3

Subjects' Responses to Factors that Affect Blood Returning to the Heart (G2).

Factors	P	1.1	1.2	1.3	2.1	2.2	3.1	3.2	4.1	4.2	R1	R2	Ph	CP	AC	Totals
Venous resistance									1			1	1		1	4
Compliance/ capacitance							1	1	1	1			1		1	6
Mean systemic pressure											1		1		1	3
Right atrial pressure					1						1	1	1			4
Stressed volume													1			1
Contraction of musculature						1			1			1				3
Contraction of vessels					1											1
Pressure gradients		1	1	1			1				1	1	1		1	8
Cardiac output	1	1	1				1			1				1		6
Totals	1	2	2	1	2	1	3	1	3	2	3	4	6	1	4	36

The most common response was that blood returning to the heart is a function of pressure differences. Relatively few subjects identified the primary determinants of venous return. Six subjects mentioned compliance as a significant factor and only 4 subjects discussed venous resistance. The specific pressure gradients, mean systemic pressure was referred to by 3 subjects, and 4 subjects mentioned right atrial pressure as issues for discussion. Only the physiologist identified stressed volume as an important factor.

Except for a fourth year student (4.1), none of the students identified more than one of the determinants of venous return. The premedical student and the first year medical students did not identify any of these relevant factors. The physiologist provided the most complete response. He discussed each of the significant factors related to venous return. He responded in a

similar manner to the first question, identifying the primary determinants of cardiac output.

Six subjects stated that cardiac output was the most important determinant of blood returning to the heart. This response pattern is consistent with a *cardiocentric bias* in which the heart is the device that regulates the blood flow through the circulatory system. This bias is illustrated in a few excerpts from subjects' responses to this question.

Excerpt from premedical student

1. It would be the same factors that are affecting
   the output,
2. because the greater the output,
3. the greater the incoming blood has to be also,
4. to keep up with demand.

The quote from the premedical student is indicative of this biased mode of thinking. The bias is based on an essential fact of circulatory physiology. That is, since the circulatory system is a closed system, whatever volume the heart pumps out, has to flow back to the heart. However, the heart is not the only agent that causes blood to flow. This bias is also reflected in a common truism expressed rather eloquently by a fourth year medical student (4.2).

Excerpt from fourth year student (4.2)

1. The old adage in cardiovascular physiology
2. is that whatever the heart pumps out,
3. it has to get back,

The excerpt suggests that this is common wisdom in cardiovascular physiology. In fact, similar quotes appear in textbooks and very likely, in class lectures. This tends to obscure one's understanding of the role of the

venous system and leads to misconceptions about the functional properties of the veins. Such a misconception is evident in the response of a third year student (3.1).

Excerpt from third year student (3.1)

5. There is nothing really pumping blood back.
6. Your veins are sort of a passive reservoir
7. and if they stretch, well the pressure in them will be higher.
8. Cause your central venous pressure is roughly zero
9. and your capillary pressure, I believe is not very high.
10. So it's sort of because blood comes in through the arteries into the capillaries.

The notion is most succinctly expressed in the phrase "Your veins are sort of a passive reservoir". This expresses the idea that the venous system plays no active role in the distribution and flow of blood. It is the heart and the other vessels that perform all the work. The student developed this argument based on the correct knowledge that the pressures are very low on the venous side.

This bias is a function of three components, which are all based on correct pieces of knowledge. The first one is that in a closed loop, everything that flows out in one direction must come back. The second source of confusion is based on the fact that pressures on the venous side are so low. Therefore, how can the venous system exert any effect on the flow of blood? The third component that contributes to this bias is that the venous system holds over 70% of all the blood in the circulatory system. It is commonly noted that the primary function of the veins is as "storers of blood". This leads to the misconception that the veins are passive reservoirs.

Subjects did not generally produce responses as complete and as coherent as the responses for the first question about the factors that regulate cardiac output. The less advanced students were particularly unsuccessful in developing adequate accounts of the factors that affect blood returning to the heart.

The response of a first year student exemplifies the difficulty students had in answering the question. This response was far superior to the responses of the other first year students, in that she demonstrated a partial understanding of the mechanisms influencing the return of blood to the heart. Her response is presented in a semantic network in Figure 6.

In constructing an answer, she makes use of the knowledge she has of particular anatomical structures, the function of the heart under different conditions, and behaviour of the circulatory system. She used a mix of teleological and mechanistic reasoning. The response presented four different but related arguments. She began by stating that since there are no muscles around the veins (this is contradicted in the second argument), the heart pushes the blood along to the arteries. The arteries develop pressure and push the blood back to the heart. The subject then provided an example. She suggested that if you are using your arm muscles, this is going to push down on the veins and since the veins have valves, the blood is going to flow in one direction and this increases blood flow. Exercise is cited as an example in which there is an increase in blood returning to the heart. Blood clots are provided as an example of something that would decrease venous return.

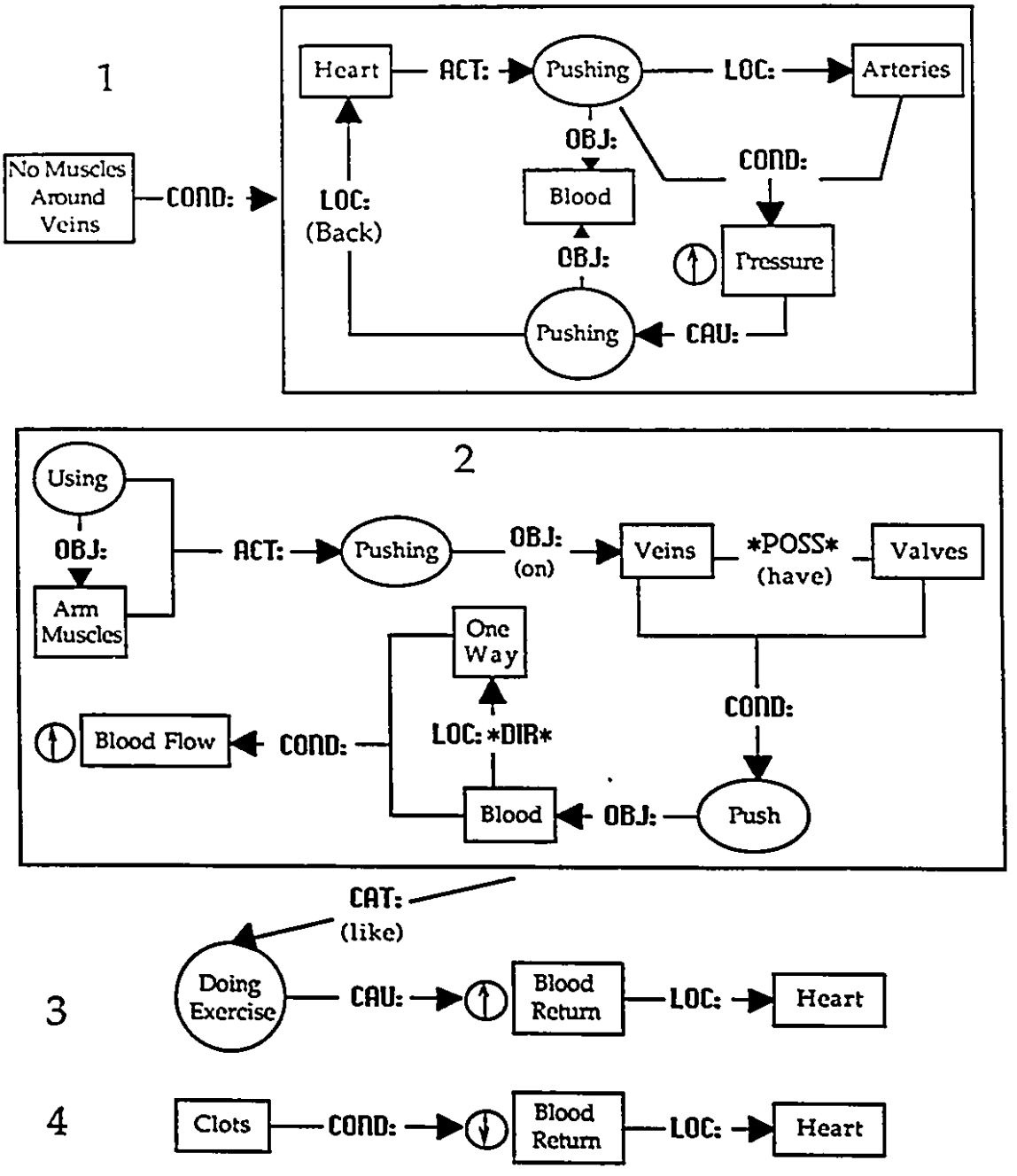


Figure 6. **Semantic Network Representation of Response by a First Year Medical Student (1.1) to Question G2.**

This response provides some insight into the subjects' mental model, at an early stage of development. The student was able to exploit a basic knowledge of anatomical structures and cardiovascular function to explain

how blood returns to the heart. She was also aware of particular instances where the rate of blood flow is affected. The subject demonstrated a lack of a process vocabulary and substitutes terms such as *pushing* rather than *contracting* when describing the actions of the heart. There are also internal contradictions in the model. Overall, this response indicates that there is an emergent mental model of the cardiovascular system that can be used towards developing a more complete and coherent understanding.

Summary

This section presented results related to subjects' responses to the two open-ended questions. The questions asked about factors that regulate cardiac output and venous return. Subjects' responses provided some insight into the organization of subjects' knowledge pertaining to these two superordinate concepts.

The more expert subjects tended to provide the better responses, listing more factors and providing superior explanations. The expert physiologist provided the most complete response to both questions.

The majority of subjects responded to the first question by discussing the primary determinants of cardiac output such as stroke volume and heart rate. Specific misconceptions were evident in subjects' responses. In particular, blood pressure was cited as a regulating factor for cardiac output.

In responding to the second question, subjects generally did not identify the primary determinants of venous return. The responses to this question tended to be less complete and less coherent. In addition, certain subjects expressed a cardiocentric bias, indicating that the heart controls the flow of blood and the venous system is a passive reservoir for storing blood.

It would appear that most subjects have a more coherent and better differentiated model of cardiac output than of venous return.

Predictive Accuracy

This section discusses the accuracy of subjects' predictions in responding to different question types. The predictions were categorized in several ways. There were 29 questions requiring a total of 43 predictions. These can be divided into predictions relating to cardiac output factors (28 predictions), venous return factors (14 predictions), and one prediction pertaining to pressure-flow relationships. The predictions were divided into the sections of the study: basic questions (27), integrated questions (12), and applied questions (4). They were further partitioned into section by type (e.g., basic-cardiac output).

It is convenient to group the subjects into two groups: the medical students (9 subjects); and the advanced group (5 subjects), which included the two residents, the physiologist, and the two cardiologists. The group data is presented first, followed by individual subjects' percentage of correct predictions. The mean percentages of correct predictions by group are presented in Table 4.

The mean percentage of correct prediction across all questions and subjects was 72.4% (SD = 13.59). Subjects, on average, correctly predicted 73.1% (SD = 13.42) of cardiac output outcomes, and 69.5% (SD = 16.74) of venous return outcomes. The average percentage of correct predictions for most categories of questions was about 70%. Subjects predicted an average of 80% of the applied questions, which only contained 4 predictions.

Table 4

Percentage of Accurate Predictions by Group and Category.

		All Subjects * (N=15)		Medical Students (N=9)		Advanced Subjects (N=5)	
Type	Predictions	$\bar{X}$	SD	$\bar{X}$	SD	$\bar{X}$	SD
TOTALS	43	72.40	13.59	71.32	10.2	80.47	11.0
CO	28	73.10	13.42	72.22	10.63	80.00	13.03
VR	14	69.52	16.74	67.46	14.34	80.00	11.74
Basic	27	72.59	12.97	72.02	10.32	79.26	10.99
Integ.	12	69.44	22.20	69.44	15.02	80.00	20.92
Applied	4	80.00	19.36	72.22	19.54	90.00	13.69
B.CO	20	73.33	13.72	73.33	11.73	78.00	14.83
B.VR	6	66.67	21.82	62.96	21.70	80.00	13.94
INT.CO	7	69.52	22.18	66.67	15.97	82.86	23.47
INT.VR	5	69.33	27.12	73.33	20.00	76.00	21.91

CO—cardiac output, VR—venous return, Basic—basic physiology section, INTEG—integrated questions, Applied—applied questions, B.CO—basic cardiac output, B.VR—basic venous return, INT.CO—Integrated cardiac output, INT.VR—integrated venous return.
*The premedical student is included only in this category.

The advanced group predicted an average of 80.5% (SD = 11) of the correct responses, including 80.0% (SD = 13) of the cardiac output questions and 80% (SD = 11.74) of the venous return questions. The medical students correctly predicted 71.32% (SD = 10.2) of the total responses, 73.1% (SD = 10.63) of the cardiac output predictions and 67.5% (SD = 14.3) of the venous return predictions. There was a consistent difference between the two groups

across categories, with the advanced group demonstrating superior performance. There was substantial individual variation within both groups.

The predictions by category are presented for each subject in Table 5. The premedical student generated the lowest percentage of correct predictions (41.85%) and the academic cardiologist (90.7%) and the physiologist (88.37%) correctly predicted the highest percentage of responses. The premedical student showed significant gaps in knowledge and made many erroneous predictions in all phases of the experiment. He also exhibited a fundamental misconception about pressure-volume relationships, that will be discussed in detail further on. A resident (R2) and a fourth year medical student also scored very highly, correctly predicting 81.4% and 86.05% of the correct responses, respectively.

The other resident (R1) and the other fourth year student performed well below expectations predicting only 62.79% and 53.49% of the correct responses. This resident responded better to venous return questions (71.4%) than to cardiac output questions (57.14%). The fourth year student scored lower than any of the subjects, except for the premedical student, even though his explanations demonstrated that he understood the concepts and could apply them in context. This subject exhibited a very particular error pattern that could account for several of his erroneous predictions.

Medical students in the first three years, responded with greater consistency to the cardiac output predictions than the venous return predictions. A fourth year student correctly predicted all of the venous return outcomes. One of the first year subjects (1.1) performed at a higher level than would have been expected, given that she had not taken the cardiovascular physiology course at this point. The second and third year students were the groups who had most recently completed the

cardiovascular physiology course and, in general, were able to recall specific causal relationships. These subjects accurately predicted a relatively high percentage of the outcomes to cardiac output questions and fewer correct venous return predictions.

Table 5

Percentage of Accurate Predictions by Subject and Category.

Subject	Totals	CO	VR	Basics	Integ.	Applied	B.CO	B.VR	INT.CO	INT.VR
P	41.86	46.43	35.71	44.44	16.67	100.00	50.00	33.33	28.57	0.00
1.1	74.42	78.57	64.29	70.37	83.33	75.00	75.00	50.00	85.71	80.00
1.2	62.79	67.86	50.00	62.96	66.67	50.00	70.00	33.33	57.14	80.00
1.3	62.79	64.29	57.14	59.26	66.67	75.00	60.00	50.00	71.43	60.00
2.1	69.77	71.43	64.29	81.48	50.00	50.00	85.00	66.67	42.86	60.00
2.2	76.74	78.57	71.43	74.07	83.33	75.00	80.00	50.00	71.43	100.00
3.1	81.40	85.71	71.43	85.19	75.00	75.00	85.00	83.33	85.71	60.00
3.2	74.42	75.00	71.43	70.37	75.00	100.00	75.00	50.00	71.43	80.00
4.1	53.49	50.00	57.14	59.26	41.67	50.00	50.00	83.33	42.86	40.00
4.2	86.05	78.57	100.00	85.19	83.33	100.00	80.00	100.00	71.43	100.00
R1	62.79	57.14	71.43	62.96	50.00	100.00	55.00	83.33	57.14	40.00
R2	81.40	85.71	71.43	88.89	66.67	75.00	95.00	66.67	57.14	80.00
Ph	88.37	85.71	92.86	81.48	100.00	100.00	80.00	83.33	100.00	100.00
CP	79.07	82.14	71.43	74.07	91.67	75.00	75.00	66.67	100.00	80.00
AC	90.70	89.29	92.86	88.89	91.67	100.00	85.00	100.00	100.00	80.00

Among the experts, the physiologist and the academic cardiologist consistently predicted a higher percentage of correct answers across all

categories than did the cardiologist practitioner. The difference was most evident in the questions pertaining to venous return. It was evident that the practitioner found the basic physiology questions to be quite laborious, and though he was able to correctly predict most of the correct outcomes, he had difficulty deducing particular relationships.

There was no consistent pattern of differences between responses to the basic questions and the integrated questions. The students up to the fourth year tended to experience greater difficulty with the basic venous return questions than with the cardiac output questions. Most subjects correctly predicted at least 3 out of the 4 outcomes for the applied questions. Across categories, there is a tendency towards better performance with higher levels of expertise.

Explanation and Understanding of Basic Concepts

The open-ended questions provided some insight into subjects' mental models of cardiac output and its regulation. The percentage of accurate predictions generated by subjects can be used as a general index to determine the extent to which subjects' understand the relationships between particular variables. This section presents an analysis of subjects' predictions and explanations pertaining to the basic concepts related to cardiac output and venous return.

Pressure-Flow Relationships

To understand cardiac output and circulatory physiology, it is essential to have a basic understanding of the physical principles that govern the flow of fluids. However, the fundamental principles of fluid dynamics, such as

Poisseuille's Law.[18] apply to rigid tubes which are of a constant radius. The vessels of the circulatory system are distensible and collapsible to varying degrees, and students need to understand the properties of different vessels that affect pressure-flow relationships.

In the study, there were four questions that dealt specifically with pressure-flow relationships and several others in which flow through the system was a related issue. There were two questions that addressed the issue of pressure-gradients in the circulatory system. They are as follows:

PF1 What pressure gradients would you use to determine the pressure drop across the entire systemic circulation?

PF2 What pressure gradients would you use to determine the pressure drop across the venous system?

The questions attempt to assess how subjects' partition the systemic circulation and the venous circulation in terms of pressure gradients. The correct answer to the first question is the pressure drop is from the *aortic pressure* to the *right atrial pressure.*[19] Except for a first year student (1.1) every subject was able to respond accurately to the question. The first year student correctly identified the forward pressure as the aorta, but suggested that the capillary beds were the back pressure.

The driving pressure for the venous system is mean systemic pressure which is about equal to the pressure in the venules. The back pressure is the

[18]Poisseuille's Law states that the volume of fluid flowing past a point in the tube, per unit time, is proportional to the difference in pressure between the inflow and outflow end of the tube and the fourth power of the radius of the tube, and is inversely proportional to the length of the tube and the viscosity of the fluid.

[19]This is a simplification. In reality, there are multiple critical pressures throughout the circulatory system that determine flow. This was alluded to by the academic cardiologist.

right atrial pressure. Every subject correctly identified right atrial pressure as the opposing pressure. There was a wide range of responses for the upstream pressure. Only the physiologist and the academic cardiologist mentioned mean systemic pressure. Nine other subjects identified either the venules or the capillaries, which are a reasonably close approximation. The premedical student suggested the pulmonary artery, which would be the driving pressure for pulmonary circulation. A first year student (1.1) suggested the aorta as a source of driving pressure. A fourth year student (4.2) and a resident (R1) suggested the mean arterial pressure as driving pressure. This is a somewhat surprising mistake since there is a substantial dissipation of pressure across the arterioles (smallest arteries) and the capillaries. Venous pressure is largely unaffected by changes in mean arterial pressure. This conceptual error is repeated by the fourth year student in subsequent questions.

The pressure-flow relationship of a vessel is determined by the *inflow* and *outflow pressure*, assuming the outflow pressure has a *positive transmural* pressure. If the outflow pressure falls below surrounding pressure (approximately zero mm Hg), then the vessel collapses at the outflow end. The pressure-flow relationship is then determined by the difference between the inflow pressure and the surrounding pressure. Subjects were presented with the two following scenarios:

PF3 What happens to flow through a vessel when the surrounding pressure exceeds the inflow pressure in a collapsible tube?

PF4 What pressure gradients would you use to determine the pressure-flow relationship, when inflow pressure exceeds the surrounding pressure in a vessel, but the surrounding pressure exceeds the outflow pressure?

The correct response to the scenario in question PF3 is quite straightforward. If the surrounding pressure exceeds the inflow pressure, then the vessel would collapse and there is *no flow*. Every subject, except for the premedical student provided the correct response. He suggested that flow would decrease.

The second situation (PF4) proved to be somewhat more difficult.[20] In this case the pressure-flow relationship is determined by the difference between the inflow and the surrounding pressure. This situation is sometimes referred to as a 'Starling Resistor' or a 'vascular waterfall'. Most subjects perseverated for quite a bit of time in answering the question. Many chose to draw diagrams and considered multiple hypotheses. Here are a few excerpts.

Excerpt from a first year student (1.1)

9. Okay,
10. if you got a collapsible vessel
11. and you start off the veins is flat
12. and not even filled out.
13. If your inflow pressure is greater that your *Psurrounding*,
14. then of course it's going to open up the valve,
15. uh the vessel, I should say.
16. And so at this point,
17. um you will have blood moving in
18. and extending the walls of the vessel.
19. So it goes in and that's fine.
20. Then we go to another point
21. where you're starting to measure the pressure outflow.
22. If this pressure of the outflow here
23. is less than pressure surrounding
24. that's going to want to collapse the vein.
25. So what's going to happen is that
26. it will push the blood forward,

[20]This can be partly attributed to the confusing wording related to the term 'pressure-gradients'. This necessitated considerable explaining on the part of the experimenter.

In this excerpt, a first year student is able to draw a series of inferences that accurately characterizes the situation. However, ultimately she is unable to provide an answer to the question. Another first year student (1.2) attempted several strategies, including generating numbers for each parameter. He finally attempted to draw on his knowledge of physical principles.

Excerpt from a first year student 1.2

34. Okay,
35. well we know that the total.
36. I'm not sure if this would hold,
37. but the total mass of material moving through.
38. Okay, if it wasn't a collapsible tube,
39. it would have to be held constant in some way.
40. If not compensated by increased flow.
41. Okay, for example if you put a small diameter in,
42. it would have to flow faster
43. to make the same amount of things go through.
44. So it would be slowest,
45. it should be slowest in this wide region.
46. So you might want to measure here and here.

Unfortunately, this strategy also turned out to be less than completely successful. It is readily apparent that the subject could not correctly represent the situation and could not address the question. A third year student attempted to construct the situation by drawing a diagram. Here is an excerpt from her response.

Excerpt from a third year student 3.1

1. Okay I'm drawing a blood vessel here,
2. with one high pressure end and one low pressure end.
3. I'm drawing an arrow squishing it down
4. reflecting the surrounding pressure
5. and you're asking me
6. when inflow pressure exceeds the surrounding pressure in a vessel,
7. but the surrounding pressure exceeds the outflow pressure.
8. I presume though

9. what's going to happen is
10. your vessel is going to collapse to look like this.
11. Sort of like a wind sock that closed
12. and there is going to be no flow
13. and the pressure at the closed end
14. that's been collapsed by the surrounding pressure,
15. is going to become equal to the surrounding pressure.
16. Which is eventually going to become equal to the inflow pressure
17. and so the flow is going to be zero.

She accurately represents the situation up to segment 9, but fails to consider that the inflow pressure will keep the vessel open and allow blood to continue to flow. Even the physiologist expressed exasperation at this question. Here is an excerpt from his response.

Excerpt from the physiologist

12. It's this vascular water fall business
13. and I'm trying to think about uh,
14. I haven't dealt with this problem in a long time,
15. can you give me a hint or something?

The excerpts illustrate the kinds of difficulty subjects experienced in responding to this question. Most subjects were not able to correctly represent the problem, even with the use of diagrams and attempted to draw on different kinds of knowledge. Several of the subjects who represented the situation correctly, still had difficulty in reaching the correct conclusions. Seven subjects were able to respond correctly to the question including a second year student (2.1), who recognized that this was a Starling Resistor, a third year student (3.2), a fourth year student (4.2), both residents and both cardiologists.

Pressure-Gradient Misconception

A basic understanding of pressure-gradients is essential to develop a coherent model of cardiac output and circulatory physiology. The nature of pressure-gradients and its relationship to the flow of blood in the circulatory system is a recurrent theme in many of the questions in the study.

The premedical student exhibited a partial understanding of pressure-volume and pressure-flow relationships. He understood that, all other things being equal, *an increase in volume* results in *an increase in pressure*. However, he reversed and extended the relationships to suggest that any increase in pressure, upstream or opposing, leads to an increase in volume and an increase *in flow*. This manifests itself in terms of a fundamental misconception about the nature of pressure-gradients.

In a question about the effects of heart rate on the pressure-volume loop, this subject expressed his belief about the nature of pressure-volume relationships, that an increase in pressure leads to an increase in volume, as illustrated below. In this context, the inference was essentially correct. When the pressure is a forward flow pressure, flow really does increase.

Excerpt from premedical student.

HR1 Explain the effects of an increase in heart rate on the pressure-volume loop.

1. Heart rate,
2. the greater the heart rate,
3. the greater the heart is contracting
4. and therefore the pressure increases.
5. the pressure increases
6. and the volume is also increasing.

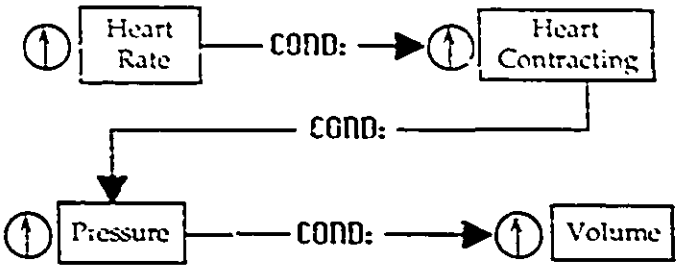


Figure 7: Semantic Network Representation of Premedical Student's Response to Question HR1.

When the pressure is a *back flow* pressure, an increase in pressure results in a decrease in flow because the pressure gradient is narrowed (as in question PGV). When right atrial pressure rises to equal the mean systemic filling pressure, the pressure gradient becomes zero and flow stops. The subject erroneously predicted an increase in flow that propagates throughout the system.

PGV. Predict what would happen if the right atrial pressure rises to equal the mean systemic filling pressure.

Excerpt from the premedical student

1. If the right atrial pressure rises
2. that means that the right ventricular volume is going to increase.
3. Okay,
4. and therefore the right ventricular pressure is also going to increase
5. and get a greater flow of blood,
6. from the right ventricle to the lungs
7. back to the left ventricle
8. to the left atrium,
9. down to the left ventricle,
10. therefore the whole system would increase.

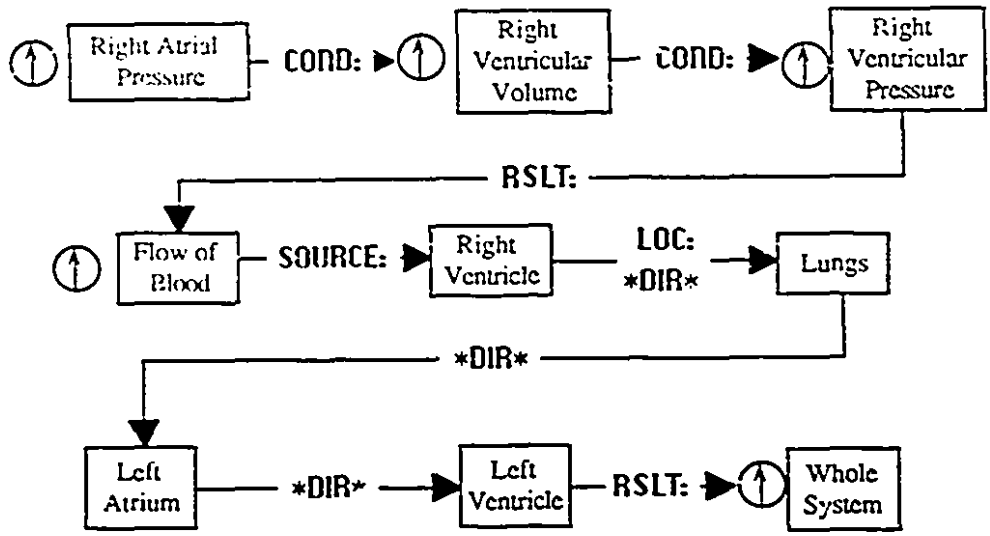


Figure 8: Semantic Network Representation of the Premedical Student's Response to Question PGV.

The above excerpt illustrates that the subject possesses a mental model of the circulatory system and can envision the consequences of the effect of a change in state, however erroneously. Yet, it is possible that he did not understand the functional role of the right atrium or its anatomical position at the end of the venous system. However, it became apparent that this is not the case. This is illustrated below in a network representation of an answer to a venous pressure gradient question (VP3), which asks about the relationships between venous return and right atrial pressure. The subject recognized that the right atrium collects returning blood and that the vena cava carries blood back to the right atrium. Yet, he still reasoned that a decrease in right atrial pressure will lower venous return.

VP3 What happens to venous return when the right atrial pressure is lowered?

Excerpt from the premedical student

1. If the right,
2. the atrial pressure refers to the right atrium,

3. the right atrium collects returning blood,
4. if the pressure in the right atrium is decreased,
5. that means that the pressure in the venous cava,
6. which is carrying the blood back to the right atrium is also decreased,
7. so I would say for that reason venous return would be lower

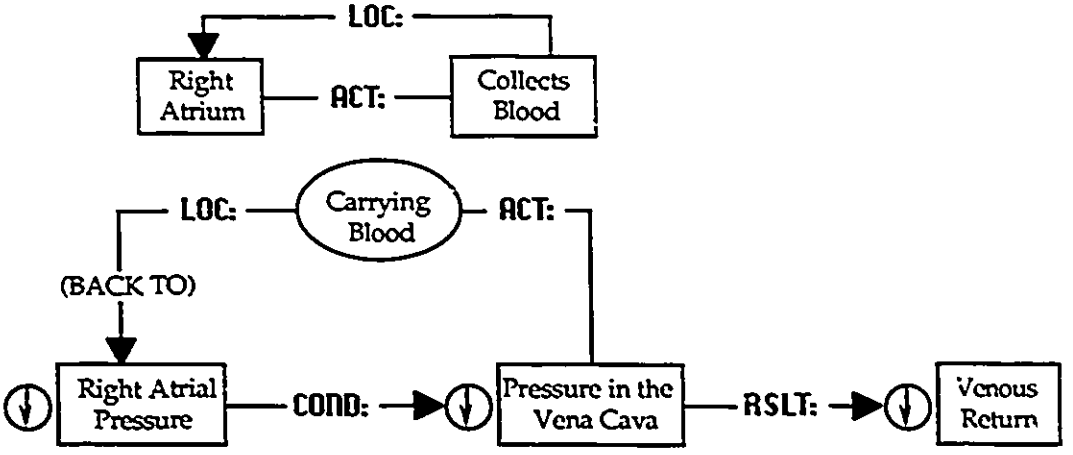


Figure 9: Semantic Network Representation of the Premedical Student's Response to Question VP3.

The question below (RA) addresses the issue concerning what will happen to venous return when the right atrial pressure falls below a critical value, that is zero. The question addresses the issue that there is an asymptotic value in which venous return is at its maximum value and can no longer increase. However, given that the subject has this misconception concerning pressure and volume, he interpreted the situation as a *no pressure* therefore *no flow* situation, resulting in an accumulation of blood.

RA Explain what happens to venous return when the right atrial pressure falls below zero and all other factors are held constant?

Response. You would get an accumulation of blood in the right atrium.

There are invariably multiple sources of converging knowledge that comprise a misconception. In this case, the sources include: the reversal of a directional relationship (increase in volume leads to an increase in pressure); and a failure to differentiate between a driving pressure and a back pressure that opposes flow. In addition, the subject manifests a "cardiocentric bias", which means that the heart or cardiac output is the sole determinant of blood flow and that the venous return merely offers a passive transport system. This very fundamental misconception was not characteristic of any of the other subjects.

Determinants of Cardiac Output

Cardiac output is a product of stroke volume and heart rate. There are three determinants of stroke volume, which is defined as the blood ejected from the heart on a single beat. These factors are *preload*, *afterload*, and *contractility*. This section presents the results related to subjects understanding of these specific concepts.

Preload

The Frank-Starling Law of the heart is one of the fundamental principles of cardiovascular physiology. It defines the regulating effect of the preload and its influence on matching venous return and cardiac output. The law states that the force or tension generated by the contracting muscle is dependent, within physiological limits, on the degree of stretch of the muscle before contraction. All things being equal, a greater inflow of blood will cause the subsequent contractions to be more forceful and will result in a greater stroke volume. The following question addressed that issue:

P1 How does the Frank-Starling mechanism regulate cardiac output?[21]

There are several elements to this question. First is the issue of the length-tension relationship in the cardiac muscle and the analog functional relationship — the pressure-volume relationship in the intact contracting ventricle (or atrium). The Frank-Starling mechanism[22] regulates the heart by producing a proportionate increase in stroke volume for an increase in end-diastolic volume. There is a limit to the heart's ability to distend, and further increase in end-diastolic pressures will not result in further increase in stroke volume or cardiac output.

Table 6 presents subjects' responses coded for particular aspects of the Frank-Starling mechanism. Seven subjects, including every subject up to second year medical school and a fourth year student (4.2), did not recognize or did not remember this mechanism. They were then provided with a prompt to explain the length-tension relationship in terms of the cardiac muscle and to explain its analog with pressure-volume relationships. The subjects who did not need a prompt and most subjects who received the prompt addressed most of the key elements.

[21]This question did not ask for specific predictions and the responses were not included in the tabulations of accurate predictions.

[22]It is commonly referred to as the Starling mechanism.

Table 6

Responses to Frank-Starling Mechanism Question (P1).

Responses	P	1.1	1.2	1.3	2.1	2.2	3.1	3.2	4.1	4.2	R1	R2	Ph	CP	AC	Totals
A*	1	1	1	1	1	1				1						7
B	1	1		1	1		1	1	1	1	1	1	1		1	12
C						1	1	1	1		1		1		1	7
D	1	1			1	1	1	1	1	1	1	1	1	1	1	13
E							1	1	1		1	1	1	1		7
F	1													1		2
Totals*	3	2	0	1	2	2	4	4	4	3	4	3	4	3	3	41

A. Needed prompt

B. An increase in muscle stretch results in a greater force in tension or shortening of the muscle.

C. The increase in tension will result in an increase in muscle contraction

D. This will result in an increase in the ejection of blood or an increase in cardiac output.

E. Regulates the heart by producing a proportionate increase in stroke volume for an increase in end-diastolic volume or preload.

F. Physiological limit of the hearts ability to distend and further increases in end-diastolic pressures will not result in a further increase in stroke volume.

*Totals exclude Response A—that subject needed a prompt.

Twelve out of fifteen subjects spoke of the increase in muscle stretch resulting in greater force, and 13 recognized that this would increase cardiac output. Three of the students were unclear on the concept, including two first year students (1.2, 1.3) and a second year student (2.2). The second year student could not recall if the mechanism was related to preload or contractility. Most of the subjects (7 out of 9) from third year on, stated that this was an important regulating mechanism for producing a proportionate increase in stroke volume for an increase in preload. An understanding of this regulating mechanism is illustrated in a rather colourful excerpt from a third year student (3.1).

Excerpt from a third year student (3.1)

1. The Frank-Starling mechanism says
2. that the fiber that's stretched is,
3. a fiber that's stretched further will contract with a greater force.
4. And it's sort of nice to have your heart working on that principle
5. because I mean,
6. if you happen to with one beat be really overloaded
7. and get in a big volume,
8. your heart will contract more,
9. it will work late
10. and stay on weekends to catch up with the extra work load.
11. So it won't get behind and on your next beat.
12. It will be down to a normal volume
13. and it will be ready to accept an overload again.

This quote clearly exemplifies the central regulating effect of the Frank-Starling mechanism, that of coupling the work of cardiac function with the demands provided by the amount of incoming blood.

Only 2 subjects, including the premedical student and the cardiologist practitioner, explicitly mentioned the physiological limits of cardiac function. This omission is probably an oversight on the part of several subjects. In other subjects it reflects a lack of understanding of cardiac function. This issue is discussed further in the context of the integration questions.

The Frank-Starling mechanism integrates and synthesizes different elements of knowledge. Most students who have not had the course in cardiovascular physiology would not likely be familiar with this concept. This affords us an opportunity to examine the kinds of prior knowledge they bring to bear on the issue. The following excerpts illustrate two students grappling with the problem and trying to produce a satisfactory response. Here is an excerpt from a premedical student.

Excerpt from the premedical student

2. The length-tension relationship of a muscle,
3. I remember.
4. Length and tension increase linearly
5. up to a certain point.

E. Situate it in terms of a cardiac muscle.

6. In terms of a cardiac muscle, okay.
7. As the tension in the cardiac muscle is increased,
8. which I would believe by increasing the volume,
9. the volume increasing the length,
10. the tension should increase as well
11. and that will be true to a certain point.
12. At which point any more increase in tension
13. will not result in any more increase in length.

E. Lets think about the ejection of blood.

16. The ejection of blood would become constant
17. it would not increase after a while.
18. In other words,
19. as the tension increases,
20. and the length of the muscle increases,
21. and therefore the volume of blood ejected would increase,
22. but at a certain point when the length of the muscle will not,
23. as the tension of the muscle will not increase any more.
24. the ejection will remain constant.

The premedical student draws on his knowledge of the length-tension knowledge of muscle and then with prompting from the experimenter is able to generate an appropriate inference concerning the cardiac muscle. In the third passage he is asked to think about the ejection of blood and is able to extrapolate the knowledge to the contracting vessel. It is noteworthy that this student was one of only two subjects to explicitly refer to the limits of stretch of the muscle and the corresponding limitation of the ejection of blood.

A first year student experienced greater difficulty in using his prior knowledge to answer the question. This subject (1.2) has a strong

background in physics, but limited knowledge of the cardiovascular and circulatory system. Here is an excerpt from his response.

Excerpt from a first year student (1,2)

5. When you say length-tension, um,
6. without losing elasticity,
7. so as long as you are still in a range
8. that you still haven't lost it,
9. elasticity of any kind of fiber or metal
10. the more you bend it,
11. the more tension you have.
12. It's linearly proportional,
13. up to the point where you've gone past the elastic limit,
14. in which case you have no, nothing left.

In this first passage, it is evident that he understood the physical principle governing length-tension relationships. However, he drew on the wrong phenomenological category. The appropriate analog is "objects that stretch" rather than "objects that bend". The difference is a subtle one. However, metal objects are typical of objects that bend and generate force. Balloons or elastic bands may have provided a more apt analogy.

Excerpt from a first year student (1,2)

E. Think about what you just told me and try to develop its analog in pressure-volume.

33. How do you relate cardiac output,
34. the more you try to distend something,
35. it's going to take a lot of energy
36. and time to do that,
37. cardiac output,
38. as I been seeing, seems that you've got a time element,
39. such that how many times do you pump
40. and you've got
41. and that affects filling.
42. Also the force with which you are doing it,
43. with which you can pump,
44. so your are somehow going to have to strike a balance
45. between getting a full, the maximum force

52. or liquid molecules come out.
53. But it's taking you a lot of,
54. I mean if cardiac output is the amount of volume per unit time then um,
55. it's taking you a lot of time
56. to get that increase in volume.
57. So you have to strike a balance,

The first year student was then prompted by the experimenter to try to develop the analog in terms of pressure-volume. He struggled with this for some time because his source analogy does not map too well onto his target. In the second and third passages, he related kinetic energy to the time needed to achieve maximum force in the ejection of blood and suggested that the regulating effect is one in which there is a balance achieved between heart rate and the force of contraction.

The responses from these two students (premedical and student 1.2) are typical of the kind of responses provided by first year medical students and the premedical student to many of the other questions. These groups of students are genuine beginners in that they have minimal backgrounds in cardiovascular physiology. Generally, they seem to have an elementary understanding or an acquaintance with most of the concepts. Nevertheless, these subjects appear to be engaged in a learning process during the study. They are drawing on analogies from disparate knowledge domains in an effort to produce satisfactory responses, sometimes failing and sometimes succeeding in their attempted.

There were three questions that asked about the direct effects of preload. They are presented below:

P2 Explain the effects of a decrease in volume on ventricular preload.

There were three questions that asked about the direct effects of preload. They are presented below:

P2 Explain the effects of a decrease in volume on ventricular preload.

P3 Explain the effects of incremental changes in preload on peak systolic ventricular pressure.

P4 Explain the effects of an increase in preload on the area of the pressure-volume loop.

The first question is the most basic one. If one understands the concept of a preload, then it should be clear that a decrease in volume will usually cause a decrease in pressure and a corresponding decrease in preload. To respond to the second and third questions, it was advantageous to consult the pressure-volume loop presented to the subjects. The second question focuses on the effects of incremental changes in preload on peak systolic ventricular pressure. The correct answer is that the ventricle accommodates increase pressure with increases in preload. The third question addresses the effects of an increase in preload on the area of the pressure-volume loop. The area of the loop represents the work of the heart (pressure times volume) and would increase when preload increases, other things being equal. Table 7 presents the correct answers coded against subjects' responses.

The responses indicated that the concept of preload was understood by most subjects. Every subject, except a first year student (1.2), responded correctly that preload is decreased when volume is decreased. The second question proved to be more difficult. Eleven subjects correctly predicted that peak systolic pressure would increase. Eleven subjects also recognized that there would be an increase in the area of the pressure-volume loop. A resident, after studying the loop diagram carefully responded that area would

not change. However, when questioned about the effects an increase in volume would have on the work of the heart, he responded that it would increase.

Table 7

Subjects' Responses to Preload Questions Coded for Correct Answers.

Q	Responses	P	1.1	1.2	1.3	2.1	2.2	3.1	3.2	4.1	4.2	R1	R2	Ph	CP	AC	Totals
P2	Preload is decreased	1	1		1	1	1	1	1	1	1	1	1	1	1	1	14
P3	Peak systolic pressure increases	1	1		1	1	1	1	1		1	1	1			1	11
P4	Increase in Area of pressure-volume loop	1	1			1	1	1	1	1	1		1	1	1	1	12
	Totals	3	3	0	2	3	3	3	3	2	3	2	3	2	2	3	37

Afterload

As discussed previously, afterload refers to the load the muscle must lift, after it begins to shorten. It is closely associated with *aortic pressure*. An increase in afterload will reduce the speed of shortening, lessen the extent of muscle shortening, and thus decrease the stroke volume. There were four questions, involving seven predictions, that addressed the effects of afterload on different variables. They are as follows:

A1 Explain how a decrease in aortic pressure would affect stroke volume.

A2 What effect does an increase in afterload have on ventricular end-systolic pressure and on end-diastolic pressure?

A3 What effect does an increase in afterload have on ventricular end-systolic volume of a single beat?

A4 How does a large increase in afterload influence stroke volume, the left ventricular ejection

fraction, and the work of the heart, on a single beat?

Aortic pressure is often taken to be a measure of afterload. Therefore, stroke volume increases when aortic pressure decreases. The second question asks about the effect of an increase in afterload on ventricular end-systolic pressure and on end-diastolic pressure. The first part of the question is relatively straightforward. An increase in afterload reduces stroke volume and therefore, end-systolic volume increases and end-systolic pressure also rises. The second part of the question is less straight-forward because it involves a time dimension. On the next successive beat of the heart with less volume having been ejected and the same volume coming back, there is an increase in ventricular pressure at the end of the diastolic filling period. The fourth question asks about the stroke volume, the ejection fraction and the work of the heart. The ejection fraction is the ratio of stroke volume to end-diastolic volume. Assuming that end-diastolic volume remains constant and stroke volume is reduced, the ejection fraction should decrease. The work of the heart may stay the same or increase. The subjects' responses coded against the correct answers are presented in Table 8.

In general, most subjects clearly understood the concept of afterload and responded accurately to most questions. However, the premedical student and the first year medical students did not have a sound grasp of what afterload represented. Despite this lack of understanding, they were able to reason about the effects of 'a kind of pressure' impeding cardiac output and could respond correctly to most questions. A fourth year student seemed to understand the concept, but correctly responded to only two of the questions. The reasons for this performance are discussed further on.

Table 8

Subjects' Responses To Afterload Questions Coded For Correct Answers.

Q	Responses	P	1.1	1.2	1.3	2.1	2.2	3.1	3.2	4.1	4.2	R1	R2	Ph	CP	AC	Total
A1	Stroke volume increases			1	1	1	1	1	1		1	1	1		1	1	11
A2	End-systolic pressure increases	1	1		1	1	1	1	1	1	1	1	1	1	1	1	14
A2	End-diastolic pressure increases	1		1			1	1					1	1	1	1	8
A3	End-systolic volume increases	1				1	1	1	1		1	1		1			8
A4	Stroke volume decreases		1	1	1	1	1	1			1	1	1	1	1	1	12
A4	Ejection fraction decreases		1	1	1	1	1	1	1		1	1	1	1	1	1	13
A4	Work of the heart increases/ doesn't change		1	1	1	1		1	1	1	1	1	1	1	1	1	13
	Totals	3	4	5	5	6	6	7	5	2	6	6	6	6	6	6	79

Several of the more advanced subjects based their judgments on clinical analogies. At times these analogies proved to be an effective heuristic for determining an outcome. For example, in determining the effect of a decrease in aortic pressure on stroke volume, the cardiologist practitioner used the following analogy:

Excerpt from a cardiologist practitioner

1. Again without thinking through as a cardiovascular physiologist
2. and just as a clinician,
3. we give vasodilators to increase cardiac output
4. and stroke volume is a measure of cardiac output
5. and they work by decreasing the aortic pressure,

6. the mean arterial pressure.
7. So one would expect that a decrease in aortic pressure
8. would increase stroke volume.

The cardiologist works backward from a common clinical practice for increasing cardiac output, that is providing vasodilators. Since vasodilators decrease aortic pressure, and stroke volume is a measure of cardiac output, "one would expect that a decrease in aortic pressure would increase stroke volume".

There are occasions in which inappropriate clinical analogies are introduced to explain physiological phenomena. A resident (R2) attempted to determine the effect of an increase in afterload on end-systolic volume, on a single beat of the heart, with the following results:

Excerpt from a resident (R2)

9. I guess one way to conceptualize this would be
10. in a situation of aortic stenosis,
11. where you would have an increase in afterload
12. which the heart actually sees at the ventricular level
13. and you get high pressure peak
14. and a hypercontractile state,
15. in which case, your end-systolic volume
16. could conceivably be smaller than normal.

The subject generated the analogy of aortic stenosis that results in an increase in afterload. This causes an increase in pressure leading to a hypercontractile state and a smaller end-systolic volume. The problem with this analogy is that clinical inferences have a *coarser* time dimension. In a given clinical situation, there are many compensatory factors that come into play. The hypercontractile state is a systemic response, on subsequent cardiac cycles, to a greater afterload that causes an increased end-systolic

volume and increased end-diastolic pressure. This results in a greater preload and diminished end-systolic volume. However, on a single beat of the heart, afterload causes a decrease in stroke volume and therefore an increase in end-systolic volume

Two questions presented some difficulty for subjects. Only eight subjects recognized that end-diastolic pressure would increase in response to an increase in afterload and surprisingly, only eight subjects recognized that end-systolic-volume increases. A third year student (3.1) reconstructed the physiological events in sequence when explaining the effects of afterload on end-systolic pressure and end-diastolic pressure (A2). Her response is illustrated below in a semantic network (Figure 10).

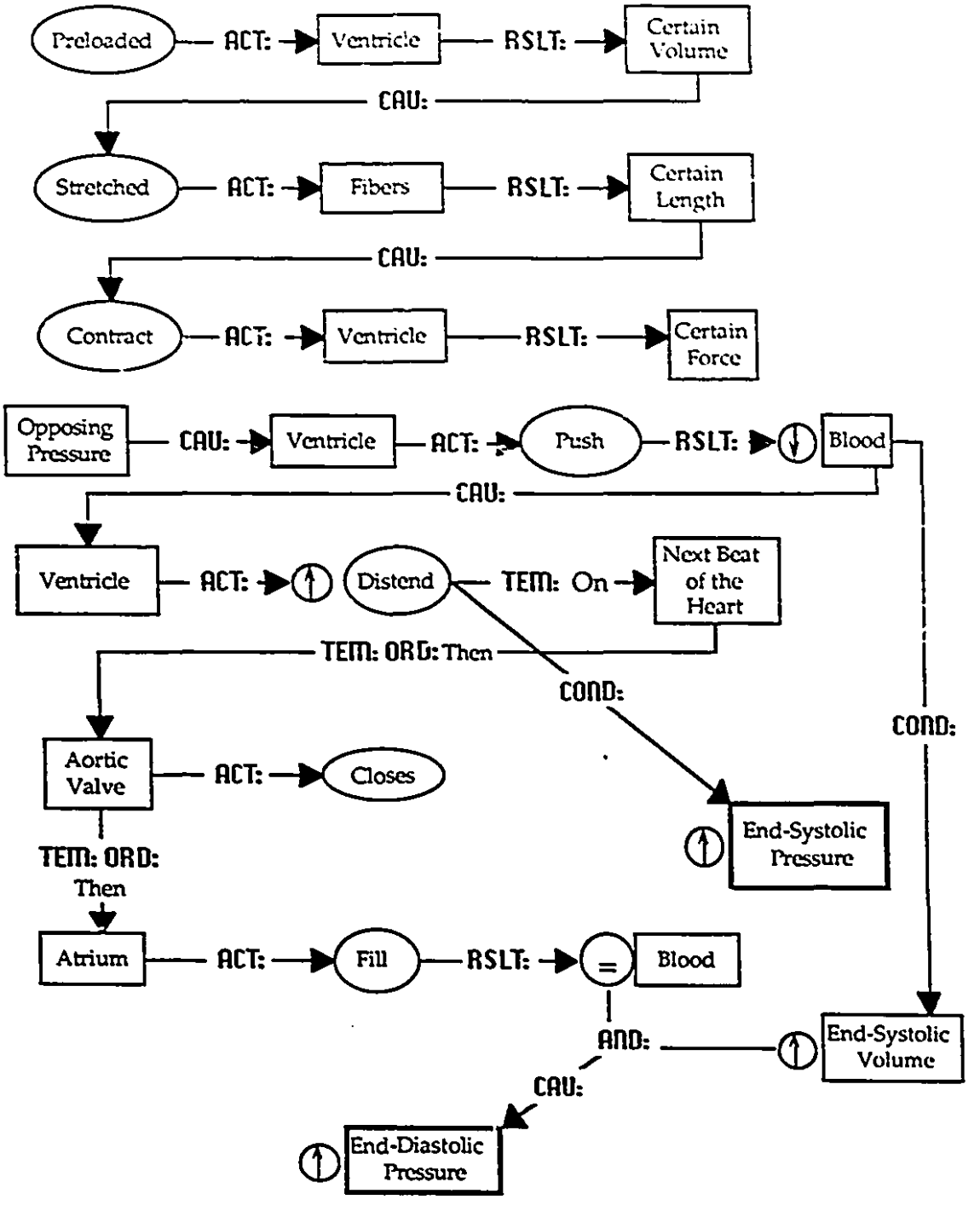


Figure 10. Semantic Network of a Third Year Student's (3.1) Response to Question A2.

The student (3.1) began by asserting that a ventricle is 'preloaded' to a certain volume, and that causes the fibers to be stretched to a certain length

and the ventricle to contract with a certain force. An opposing pressure causes the ventricle to push out less blood, which increases the end-systolic volume and causes the ventricle to distend more on the next beat. Then the aortic valve closes and the atrium fills with the same amount of blood. This volume of blood and the volume left over from the end of systole is going to then increase end-diastolic pressure.

This response illustrates the complexity of propagating the effects of changes in variables on other measures in the circulatory system over time. This student developed a well-constructed chain of inferences beginning with basic premise of pressure-volume and length-tension relationships and proceeded to introduce the perturbation in the system, which is an opposing pressure. The explanation then focused on specific events in the cardiac cycle (e.g., aortic valve closes, atrium fills with blood) and examined the effects on pressure in the ventricle at the end of systole and at the end of diastole.

A fourth year student (4.1) appeared to understand the concept of afterload in that he could articulate its meaning. However, he correctly predicted only 2 out of seven predictions, which was less than any other subject. We can delineate the source of his error. The subject relied extensively on the pressure-volume loop diagram to calibrate his judgments of the effects of the determinants of cardiac output. He repeatedly failed to retrieve correct correspondences between the graph features and the functional relationship. This is illustrated below in a reconstruction of the subject's drawing and explanations of the effects of afterload on the pressure volume loop (Figure 11).

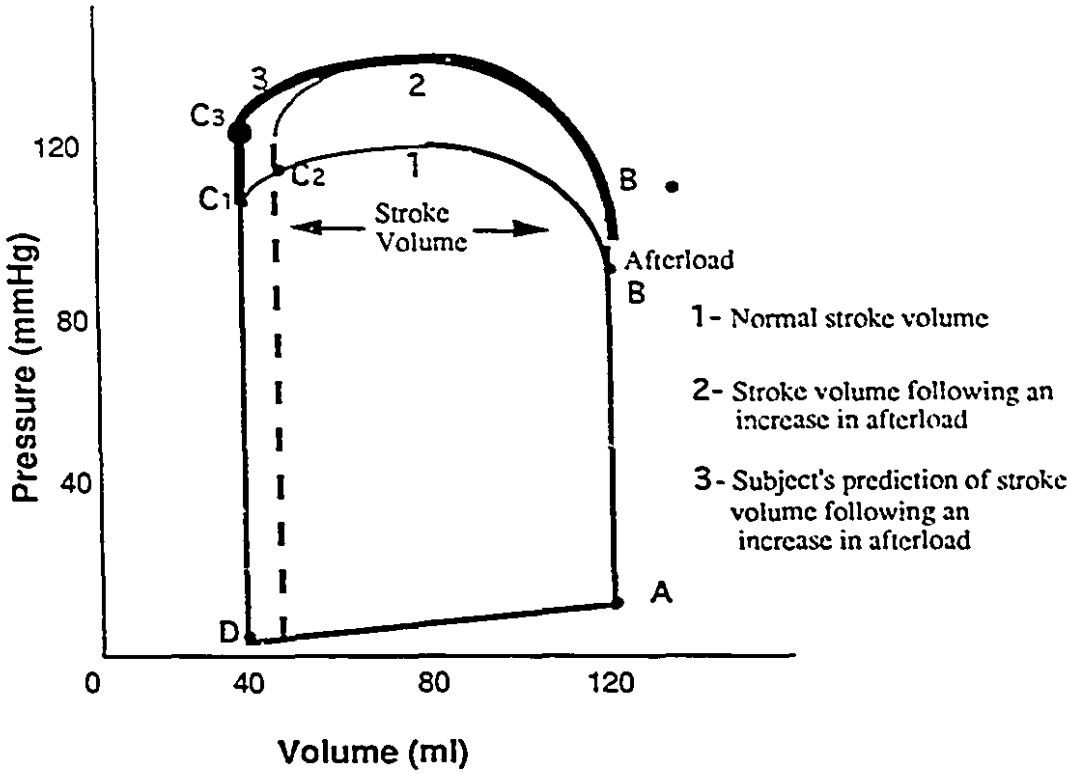


Figure 11: The Predicted Effect of Afterload by a Fourth Year Student (4.1) as Illustrated on the Pressure-Volume Loop Diagram.

The primary inference is that afterload has no effect on stroke volume. Afterload, in fact, decreases stroke volume. Stroke volume is the volume ejected on a single beat of the heart (difference between volume in the ventricle at **B** and volume in the ventricle at **C**). The afterload corresponds to the pressure facing the ventricle once the aortic valve opens (point **B**). If this pressure increases, then the heart will not be able to eject as much blood. Point C_2 indicates that there is more blood left in the ventricle following systolic ejection. This subject correctly identified that the afterload corresponds to point B on the diagram, and that an increase in afterload shifts the curve upward. However, in attempting to reconstruct the curve from memory, he erroneously shifted the curve back to the same level of volume (C_3) and therefore concluded that there is no change in stroke

volume. It is also possible that he did not remember that the end-systolic pressure volume relationship can shift back and forth in response to changes of various kinds. Given this erroneous representation and conclusion of no change in stroke volume, all subsequent inferences, such as no change in cardiac output, are likely to be false.

The cardiologist practitioner made a similar error. However, the error was made only in response to the question concerning the effect of afterload on end-systolic volume. Here is an excerpt from his response:

Excerpt from a cardiologist (CP)

9. I don't think it would have an effect on end-systolic volume,
10. if your contractility and everything else remains the same.
11. Let me see afterload.
12. I'm just starting at point A,
13. when the ventricular contraction begins
14. and uh its an isovolumic contraction
15. and it moves up to B
16. and if the afterload is higher,
17. point B will be higher up the pressure scale.
18. But it seems to me that uh,
19. that the ventricular end-systolic volume stays constant.

This mistake is identical to the one made by the fourth year student. It is noteworthy that this cardiologist correctly predicted that an increase in afterload would reduce stroke volume. End-systolic volume is a function of stroke volume and end-diastolic volume. The cardiologist's analysis, therefore, focused on the variables in isolation, and overall, his response was inconsistent with his other responses.

It is difficult to ascertain whether these errors are conceptual or as a result of an experimentally induced artifact. The student (4.1) was able to recognize his error in subsequent discussions, suggesting his understanding

was affected by the diagram. Graphic diagrams and data plotted on curves, are frequently used by cardiologists and other physicians in decision-making activities. They are important representational devices and valuable resources for explaining phenomena. Nevertheless, they also present a source of possible confusion and error.

Contractility

As discussed previously, contractility is the functional state of the heart muscle, that is defined by the rate and extent of shortening, for a given afterload and preload. An increase in contractility causes the muscle to contract faster and to a higher peak tension. This section focuses on subjects' response to the following question:

C1 Explain the effects of increases in contractility on end-systolic volume, end-systolic pressure, end-diastolic volume, and end-diastolic pressure and on stroke volume.

The question asks for explanations and predictions of the effects of an increase in contractility on five variables: *end-systolic volume*, *end-systolic pressure*, *end-diastolic volume*, *end-diastolic pressure*, and on *stroke volume*.

An increase in contractility is known to increase stroke volume. If stroke volume is increased then, all other things being equal, end-systolic volume will decrease. If the ventricle contracts with greater force, then its peak systolic tension is increased and therefore, end-systolic pressure is also increased. These three explanations require a minimum of inference. However, the effects of end-diastolic volume and end-diastolic pressure are more difficult to determine. They demand that subjects propagate the effects of the increase in contractility over a cardiac cycle or two. Assuming that the

venous return is constant on the next beat, and the ventricle has emptied to a lower volume, there will be less end-diastolic volume and therefore a lower end-diastolic pressure. The subjects' responses coded, against the correct answers, is presented in Table 9.

Table 9

Subjects' Responses to Contractility Question Coded for Correct Answers.

Responses	P	1.1	1.2	1.3	2.1	2.2	3.1	3.2	4.1	4.2	R1	R2	Ph	CP	AC	Totals
End-systolic volume decreases	1	1	1	1	1	1	1	1	1	1		1	1	1	1	14
End-systolic pressure increases		1	1	1	1	1		1				1	1	1		9
End-diastolic volume decreases		1	1									1			1	4
End-diastolic pressure decreases			1									1			1	3
Stroke volume increases	1	1		1	1	1	1	1	1	1		1	1		1	12
Totals	2	4	4	3	3	3	2	3	2	2	0	5	3	2	4	42

The results indicate that fourteen subjects recognized that end-systolic volume decreases with increases in contractility and twelve suggested that stroke volume would increase. Because of the strong correlation between these two variables, it was expected that subjects would respond correctly or incorrectly to both of these questions. The cardiologist practitioner correctly predicted that end-systolic volume would decrease, but suggested that stroke volume would not change. He pursued an interesting strategy, which is illustrated in an excerpt from his response.

Excerpt from the cardiologist practitioner

10. As you increase contractility,
11. your Frank-Starling curve shifts up and to the left.
12. Which I never see in my patients and if,
13. so if you started at a given volume
14. and you increase
15. uh you're going to finish at a given pressure.
16. The stroke volume conceivably does stay the same.
17. I'm sort of starting at the back.
18. If everything else stays the same,
19. your looking at the area under the curves and,
20. I think that the area for the stroke volume would be about the same.
21. Your end-systolic volume, would decrease.

The cardiologist attempted to reconstruct the situation using Frank-Starling curves, which is a common device for examining changes in cardiac output. The analysis focused on each variable in isolation from one another. A problem with graphs and diagrams is that time dimensions can become obscured, if one focuses on Cartesian points in isolation. An analysis of pressure-volume changes needs to be considered in the context of events in the cardiac cycle.

Nine subjects generated 3 or more correct predictions. One resident (R2) correctly predicted all 5 answers, and the other resident (R1) did not predict any of the correct responses. The resident (R1) suggested, without much deliberation, that all five variables would not be affected by an increase in contractility. Only four subjects correctly predicted that end-diastolic pressure would decrease, and three correctly predicted that end-diastolic volume would decrease. This question was somewhat more complex than others in the first section, requiring a longer chain of inferences. In general, most subjects did not undertake the necessary analysis to determine the effects of contractility on these variables.

Heart Rate

Cardiac output is a product of stroke volume and heart rate. Heart rate is defined as the number of beats or cardiac cycles per unit time. It is principally controlled by factors extrinsic to the heart muscle. Heart rate exerts its influence on cardiac performance in three ways. An accelerated heart rate, within limits, can increase cardiac output. An increased heart rate also produces an increase in contractility. Heart rate also interacts with stroke volume. An increase in heart rate decreases diastolic filling time and the compliance of the ventricle which results in a decreased end-diastolic volume. This effect produces a decrease in stroke volumes on subsequent contractions. The net effect of increases in heart rate on cardiac output is dependent on many factors. However, it is assumed that within normal physiological limits, that increases in heart rate will produce increases in cardiac output. Heart rates beyond normal limits (e.g., upwards of 200 beats per minute) will produce decrements in cardiac performance.

Subjects were asked the following three questions:

HR1 Explain the effects of an increase in heart rate on the pressure-volume loop.

HR2 How would an increase in heart rate affect end-diastolic volume, with all other factors held constant?

HR3 Predict what would happen to cardiac output if the heart rate is increased by a pacemaker from a normal value of 60 beats per minute to a) 125 beats per minute, b) 300 beats per minute, and c) decreased to 20 beats per minute.

The first question presented subjects with the opportunity to focus on any of the variables related to the pressure-volume loop. In particular, we wanted to see if subjects would identify end-diastolic volume as being potentially

compromised.[23] The second question directly addressed the issue of heart rate and end-diastolic volume.

The third question presents 3 scenarios in which the heart rate is specifically altered by the use of a pacemaker set to three different values. The pacemaker is employed as a device to provide a controlled situation in which we can examine the mechanical effects of heart rate independent of the various compensatory mechanisms that would otherwise come into play. The first scenario presents a normal increase in heart rate (similar to the increase in heart rate one may expect in moderate exercise) which should achieve a concomitant increase in cardiac output. The second situation, in which heart rate is increased fivefold, is characteristic of the most extreme tachycardia (abnormal high heart rate) and would severely compromise diastolic filling and dramatically reduce cardiac output. The last scenario describes a situation of severe bradycardia (abnormal low heart rate) and one would expect a decrease in cardiac output.

We were also interested in whether subjects recognized that, increases in heart rate results in increases in contractility, although, there was no question that explicitly addressed this issue, there were several questions throughout the study in which this functional relationship was of relevance. Subjects were coded according to whether they acknowledged the effect of heart rate on contractility. The subjects' responses coded against the correct answers is presented in Table 10.

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[23]It was anticipated that subjects might also address the interaction of heart rate and contractility. The end-systolic pressure-volume line, whose slope is a measure of contractility, was omitted from the pressure-volume diagram presented to subjects (compare Figures 2 and 3). As a result, only one subject made any mention of contractility in his response and this was excluded as a coding criteria for this question.

Table 10

Subjects' Responses to Heart Rate Questions Coded for Correct Answers.

Q	Responses	P	1.1	1.2	1.3	2.1	2.2	3.1	3.2	4.1	4.2	R1	R2	Ph	CP	AC	Totals
HR1	End-diastolic volume decreases		1	1		1	1	1	1	1	1	1	1	1	1	1	13
HR2	End-diastolic volume decreases		1	1		1	1	1	1	1	1	1	1	1	1	1	13
HR3	Cardiac output increases—125 beats per minute	1		1	1	1		1			1		1	1	1		9
HR3	Cardiac output decreases—300 beats per minute		1	1		1	1	1	1	1	1	1	1	1	1	1	13
HR3	Cardiac output decreases—20 beats per minute	1	1	1	1	1	1	1	1	1	1		1	1	1	1	14
	Heart rate increases contractility							1	1	1		1	1	1	1	1	8
	Totals	2	4	5	2	5	4	6	5	5	5	4	6	6	6	5	70

Subjects correctly responded to most of the questions concerning heart rate. Thirteen of the subjects identified end-diastolic volume as the measure most likely affected by an increase in heart rate. The same thirteen subjects responded that increases in heart rate decrease end-diastolic filling and volume. Only nine subjects suggested that cardiac output would increase at 125 beats per minute. The other subjects suggested that it would not change. Every subject, except for a first year student (1.3) and the premedical student, recognized that a heart rate of 300 beats per minute would dramatically decrease end-diastolic filling time and decrease cardiac output.

Fourteen subjects recognized that a drop in heart rate to 20 beats per minute would reduce cardiac output.

Two third year students made particular kinds of over-generalizations concerning heart rate. Here is an excerpt from a first year student (1.3):

Excerpt from a first year student (1.3)

5. If the pacemaker increases the value
6. from 60 beats per minute to 125 beats per minute,
7. which is roughly a twofold increase,
8. then the cardiac output should double plus a bit more
9. because it's slightly more than twofold.
10. If it's increased fivefold to 300 beats per minute,
11. then cardiac output should increase fivefold
12. and if it's decreased to 20 beats per minute,
13. which is a decrease of a factor of three,
14. then cardiac output should,
15. the new cardiac output should be one-third of the old cardiac output.

This student knew that cardiac output is equal to heart rate times stroke volume. He interpreted this equation (CO = SV x HR) as describing a positive linear function, suggesting that there is one-to-one relationship between any increase or decrease in heart rate and cardiac output. The student did not consider that stroke volume could be compromised at very high heart rates.

The other first year medical student (1.1) made the opposite over-generalization. She understood that high heart rates can compromise diastolic filling. An excerpt from her response is given below:

Excerpt from a first year student (1.1)

2. If you increase beats to 125 beats per minute,
3. um I think your output,
4. your cardiac output would decrease a heck of a lot.

5. Reason being it's not having time to sort of fill up again.

The response suggested that any increase in heart rate will compromise diastolic filling and decrease cardiac output.[24] This response also discounted the fact that heart rate is a primary determinant of cardiac output and will, within normal limits, increase cardiac output.

Eight subjects discussed the effects of heart rate on contractility, in responding to different questions in the study. Every subject from third year medical school on, except for a fourth year student, recognized that an increase in heart rate can produce an increase in contractility. No subjects prior to third year discussed this functional relationship.

Summary

This section presented results of subjects' responses to the four factors related to cardiac output. These include: heart rate; and the determinants of stroke volume; preload, afterload, and contractility. The questions focused predominantly on the various functional relationships between these variables. There was a tendency towards increased conceptual understanding with expertise, although there were very salient individual differences. Subjects from second year medical school onward had a clear understanding of each concept. The first year students and the premedical students often did not have a clear understanding of the meaning of each concept. However, one first year student (1.1) performed at a high level relative to her peers. She demonstrated a broader knowledge of

[24]It is also possible that the subject did not appreciate that a doubling of heart rate is not an extreme or unusual occurrence.

cardiovascular physiology, and a superior ability to use her prior knowledge to understand novel concepts.

Many of the subjects understood the basis of the Frank-Starling mechanism, although several subjects needed prompts. Only three subjects expressed the fact there was a physiological limit to the heart's ability to distend and to increase stroke volume. Subjects responded with considerable accuracy to the questions concerning preload. The questions, that presented the most difficulty for both afterload and contractility concepts, were those that required temporally distal explanations and predictions. That is, fewer subjects responded correctly to the questions that necessitated a consideration of a behavioural process, such as, propagating the effects of increased pressures over different cycles of the heart. The mechanical effects of heart rate were well understood by most subjects. However, few subjects indicated an understanding of the relationship between heart rate and contractility.

There were many individual differences between subjects at the same level. The premedical student's understanding of these concepts was impaired by a serious misconception related to pressure gradients. This subject, although acquainted with the concepts, tended to view increases in one variable as promoting increases in other variables. As mentioned previously, a first year student acquitted herself particularly well, demonstrating an understanding beyond what would have been expected from a student who had not taken the cardiovascular physiology question. She correctly predicted 76% of the cardiac output questions. The two other first year students did not demonstrate a clear understanding of these concepts. Both students had strong backgrounds in physics and attempted to draw on this physical science knowledge to answer various questions.

Generally, these attempts were less than successful, apparently because their knowledge of the target domain (cardiovascular physiology) was insufficient to access and map the appropriate analogies.

A third year student (3.1) responded with considerable accuracy to the cardiac output questions, correctly predicting 83% of the outcomes pertaining to cardiac output. Her explanations were among the most elaborate and complete of any of the subjects. A fourth year student (4.1), although indicating an understanding of each concept, did not correctly identify many of the functional relationships. This subject predicted only 50% of the correct cardiac output outcomes. One of the reasons for this poor performance was the ineffective use of the loop diagram to calibrate his judgments. This was clearly demonstrated in the questions pertaining to afterload. One of the residents (R2), responded with considerable precision to the cardiac output questions, correctly predicting 86% of the cardiac output outcomes. The other resident only predicted 57% of the correct outcomes. His errors were distributed across question types and there was no discernible pattern of conceptual errors. The academic cardiologist provided the best explanations and had the highest percentage of accurate predictions of the expert subjects, predicting 89% of the cardiac output responses.

Determinants of Venous Return

The output of the heart represents a balance between the demands of the body's metabolism, reflected in the venous return and the ability of the heart to meet the demand (Smith & Kampine, 1990). This section focuses on concepts related to venous return. As discussed in chapter 5, venous return is determined primarily by *vascular compliance, stressed volume, right atrial pressure,* and by *venous resistance.* Vascular compliance refers to the ability

of a vessel to distend to accommodate more blood volume per unit pressure. Vascular resistance is the opposition to blood flow offered by the vessels.

Mean systemic pressure[25] is defined as the static pressure remaining in the vessels if the circulation is stopped (Guyton, Jones, & Coleman 1973). It is the driving pressure for venous return. Mean systemic pressure can be expressed as the ratio of *stressed volume* to systemic vascular compliance. *Stressed volume* is the volume of blood that stretches the elastic walls of vessels and thus produces pressure in the vasculature. It constitutes about 30% of the volume in the vasculature. The downstream pressure for venous return is the pressure at the outflow to the venous system, which is the *right atrial pressure.*

Mean Systemic Pressure Stressed Volume, and Right Atrial pressure

Subjects were presented with two questions that asked about the defining qualities of mean systemic pressure and stressed volume. The question pertaining to mean systemic pressure is as follows:

VP1 What does mean systemic pressure (P_{MS}) refer to?

The reference response for this question includes: the definition that mean systemic pressure is the pressure which distends the vessel when the circulation is stopped; it is the driving pressure for venous return; and it is determined by stressed vascular volume and/or venous compliance. The subjects' coded responses are presented in Table 11. In addition to the correct responses, we also coded for original content.

[25]Mean systemic pressure is sometimes referred to as mean systemic filling pressure. It is also sometimes used synonymously with mean circulatory filling pressure. To avoid terminological confusion, subjects were informed of the multiple terms.

Table 11

Subjects Response to Mean Systemic Pressure Question (VP1).

Responses	P	1.1	1.2	1.3	2.1	2.2	3.1	3.2	4.1	4.2	R1	R2	Ph	CP	AC	Totals
A					1				1	1	1		1		1	6
B															1	1
C					1								1			2
D	1											1		1		3
E		1	1	1		1		1								5
F			1					1								2
Totals*	0	0	0	0	2	0	0	0	1	1	1	0	2	0	2	9

*Only responses A-C are included in the totals.

a. The vessel distending pressure measured when the circulation has been stopped.

b. The pressure that determines venous return.

c. A function of stressed vascular volume and/or venous compliance.

d. The mean arterial pressure

e. An average pressure in the systemic circulation, throughout the body or the circulatory system.

f. Don't know/can't remember.

The results suggest that the concept of mean systemic pressure was not well understood by most subjects. Six subjects, including a second year student (2.1), both fourth year students, a resident (R1), a physiologist, and the academic cardiologist, stated that mean systemic pressure is a distending pressure measured when the circulation has been stopped. Only the academic cardiologist mentioned that it was a pressure that determines venous return. In two previously discussed questions pertaining to pressure gradients for venous return and factors that regulate venous return, both the physiologist and a resident (R1) indicated that mean systemic pressure was a driving pressure for venous return. Only the physiologist indicated that mean systemic pressure is a function of both stressed volume and compliance.

Three subjects, including the premedical student, a resident (R2), and the cardiologist practitioner, equated mean systemic pressure with mean arterial pressure, which is the average pressure in the arteries. This is illustrated in the following two excerpts:

Excerpt from a resident (R2)

1. Typically systemic pressure refers to pressures on the arterial side.
2. A mean systemic pressure would be
3. one-third the pulse pressure
4. added on to the diastolic pressure.

Excerpt from a cardiologist (CP)

1. I would take that to mean mean arterial pressure ah,
2. which is a combination,
3. a mean between the systolic and diastolic pressure
4. being as there is a cyclic nature to the function.

.....

11. It is two-thirds of the diastolic
12. and one third systolic.

Both of these physicians describe mean systemic pressure as being synonymous with mean arterial pressure, which is a very different kind of pressure. Mean arterial pressure is the more commonly used measure in clinical situations.

Five subjects stated that mean systemic pressure reflected an average pressure of some kind, which may reflect a recognition of the term 'mean'. The most novice subjects, including the premedical student and the first year medical students, clearly did not recognize the term mean systemic pressure.

They typically described it as some kind of average pressure in the system. Here is an excerpt from one of the more interesting responses:

Excerpt from a first year student (1,2)

1. Well, if there is this pumping,
2. there is going to be like waves of high
3. and then you go down
4. and you go up
5. and then you go down
6. and if you,
7. I don't know what would happen,
8. if you actually averaged it.
9. I don't know if it's sinusoidal or whatever.
10. But if you took a straight average you might get zero.
11. But if you did something like a root mean square.

The first year student responded to this question by making an analogy to the "pumping motion of waves". This is a student with a strong background in physics. We can speculate that he also understands something about the *pulsatile* and *turbulent* nature of blood flow in the circulatory system. Given that, he produced a reasonably sophisticated response. However, he consistently drew analogies from the physical domain of *kinematics*, which is the study of the description of objects in motion (e.g., velocity, acceleration). Pressure-volume relationships are subsumed under the physical domain of *statics*, which is the study of forces acting on bodies in equilibrium. Pressure-flow relationships characterize the *dynamics* of circulation, which is the domain that describes forces acting on bodies in motion. Therefore, the physical analogs for mean systemic pressure are to be found in the domains of statics and dynamics. Spontaneous analogies are rather difficult to generate when the objects in the target domain are not clearly specified.

Subjects were asked about the defining properties of stressed volume. The question reads as follows:

VP2 What is the difference between stressed volume and unstressed volume? What is the significance of this difference? Estimate the percentage of stressed volume and the percentage of unstressed volume in the human circulatory system.

There are three parts to the question. The first part of the question asks subjects to distinguish between stressed and unstressed volume. The important point is that stressed volume contributes to the pressure within the vasculature, and unstressed volume does not. The significance of the difference is twofold. Stressed volume contributes to mean systemic pressure and unstressed volume acts as a reserve volume in the venous system. Stressed volume, under normal conditions, constitutes approximately one-third to one-quarter of the total vascular volume, with the remainder being unstressed volume. Table 12 presents the subjects' coded responses compared to the correct response. In addition, subjects' original responses were also included in the table.

The results suggest that like the concept of mean systemic pressure, stressed volume was not well understood by most subjects. Five subjects, including a first year student (1.1), a fourth year student (4.2), a resident (R1), the physiologist, and the academic cardiologist, stated that the stressed volume was the volume that contributes to pressure in the vasculature. Only three subjects stated that stressed volume contributes to mean systemic pressure. Five subjects understood that unstressed volume can act as a reserve. Six subjects suggested that stressed volume constituted about one-quarter of the total blood volume. Five subjects, including a resident (R2) and the cardiologist practitioner, indicated that they did not know or could not remember what the term meant.

Table 12

Subjects' Response to Stressed Volume Question (VP2).

Responses	P	1.1	1.2	1.3	2.1	2.2	3.1	3.2	4.1	4.2	R1	R2	Ph	CP	AC	Totals
A		1								1	1		1		1	5
B					1								1		1	3
C				1			1				1		1		1	5
D					1		1		1	1	1				1	6
E		1														1
F	1			1								1				3
G	1															1
H			1													1
I							1							1		2
J									1							1
K				1		1		1				1		1		5
Totals*	0	1	0	1	2	0	2	0	1	2	3	0	3	0	4	19

*Only responses A-D are included in the totals

A. Stressed volume is volume that contributes to pressure within the vasculature.

B. Stressed volume is the volume that contributes to mean systemic pressure.

C. Unstressed volume is a kind of reserve volume.

D. Twenty-five to 30% of total volume is stressed and 70-75% of the volume is unstressed.

E. More stressed than unstressed.

F. Stressed volume is measured while the person is under stress.

G. The heart would have to work harder during stressed period.

H. Stressed volume is blood undergoing kinetic motion.

I. Arterial versus Venous

J. Intravascular Space versus extravascular interstitial space.

K. I Can't remember / I don't know.

There was a considerable range of erroneous responses. Three subjects, the premedical student, a first year medical student (1.1), and a resident (R2), suggested that stressed volume was measured while a person

was under stress or engaging in physical exertion. In keeping with this theme, the premedical student indicated that the significance of this "was that the heart would have to work harder during the stressed period". A third year student and the practitioner alluded to the fact that pressure was higher on the arterial side, and that stressed volume would be measured in the arteries.

The first year students did not recognize the concept of stressed volume. However, they proposed some interesting possibilities. Here is an excerpt from a first year student:

Excerpt from a first year student (1,1)

I presume that stressed volume has something to do
with the amount of blood flow
that would be under direct muscular pushing as it were,

Although lacking the vocabulary ("direct muscular pushing"), the student comes very close to the idea that stressed volume reflects volume under pressure in the vasculature. Below is another excerpt from a first year student:

Excerpt from a first year student (1,2)

Stressed volume might be the part of the blood that experiences some motion
because of pumping.
I'm thinking in terms of water in an ocean,
if you have this wave going through,
not all of the water experiences the wave in the same way.
There is going to be parts of the liquid
that would have greater kinetic motion
and other parts that are not going to move as much.
I'm thinking that the parts that are moving more,
are stressed volume.

This student (the physics major). again proposed an analogy relating to the pumping action of waves and kinetic energy. He suggested that the "parts that are moving more are stressed volume".

E. Can you think of an analog in a closed system, say distensible tubes or pipes.

Excerpt from a first year student (1,2)

12. Okay,
13. if there is a lot of viscosity
14. and your moving something through a pipe,
15. I think, actually
16. that things in the middle will have a greater velocity than things at the edge.
17. And so I'm not sure
18. if that would cause it to be one to be called stress
19. and the other to be called unstressed.

The experimenter then suggested to him that he try to develop an analogy from a closed physical system. He adopted his prior analogy rather literally to this closed system, adding only the idea of the fluid viscosity. There are two things wrong with the analogy. The first is that he was still focused on the kinematics of motion instead of the statics of the system. The second error in the analogy is that he emphasized the *composition of the substance* (its viscosity) rather than *forces acting* on the substance.

There were four questions that required explanations and predictions concerning the pressure-flow relationships related to venous return. They are as follows:

VP3 How does an increase in mean systemic pressure effect the venous return curve?[26]

VP4 Predict what would happen if the right atrial pressure rises to equal the mean systemic filling pressure.

VP5 What happens to venous return when the right atrial pressure is lowered?

VP6 Explain what happens to venous return when the right atrial pressure falls below zero and all other factors are held constant?

The first question asks how an increase mean systemic pressure would affect the venous return. Since it is the driving pressure, an increase in mean systemic pressure would increase venous return. The second question presents a situation in which a pressure gradient is reduced to zero and thus blood flow would cease. The third and fourth questions address the issue of the effects of a decrease in right atrial pressure on venous return. Since right atrial pressure is a back pressure, a decrease will produce an increase in venous return. However, the fourth question (VP4) describes a situation in which venous return is at its maximum and any further decreases will produce a collapse of the vessel. The subjects' responses coded against the correct answers is presented in Table 13.

Twelve out of fifteen subjects correctly suggested that venous return would increase with an increase in mean systemic pressure. It is noteworthy that many of these subjects did not correctly define mean systemic pressure. Several subjects suggested that it was synonymous with mean arterial pressure. In this case, they would have been wrong, since mean arterial pressure has only a minimal direct effect on venous return. A resident (R2)

[26]The question was asked with the intent that subjects would talk about venous return curves. Few subjects were able to do so. Therefore, they were asked to discuss the effects of an increase in mean systemic pressure on venous return.

suggested that it could impede blood flow. A first year student (1.3), after calculating the pressure changes throughout the circulatory system, determined that venous return would decrease. A third year student (3.2) suggested that mean systemic pressure would affect afterload and not change venous return.

Table 13

Subjects' Responses to Venous Return Pressure Questions Coded for Correct Answers.

Q	Responses	P	1.1	1.2	1.3	2.1	2.2	3.1	3.2	4.1	4.2	R1	R2	Ph	CP	AC	Totals
VP3	Venous return increases	1	1	1		1	1	1		1	1	1		1	1	1	12
VP4	Flow stops				1	1		1		1	1	1	1	1	1	1	10
VP5	Venous return increases		1	1	1	1	1	1	1	1	1	1	1	1	1	1	14
VP6	Reaches asymptote									1	1	1		1		1	5
	Totals	1	2	2	2	3	2	3	1	4	4	4	2	4	3	4	41

Ten subjects correctly predicted that flow would cease if the right atrial pressure would rise to equal the mean systemic pressure. Each subject indicated in an earlier question that right atrial pressure was the opposing pressure gradient for venous return. All the other subjects, except for the premedical student, suggested that venous return would decrease. As discussed previously, the premedical student consistently predicted that any increase in pressure resulted in increased blood flow. This would also explain why he correctly predicted that venous return would increase in response to an increase in mean systemic pressure. All subjects except for the premedical student recognized that a decrease in right atrial pressure would increase venous return. However, only five subjects, including both fourth year students, a resident (R2), the physiologist, and the academic cardiologist,

predicted that venous return would reach an asymptote in which further decrease in right atrial pressure would not result in further increase in venous return. The academic cardiologist argued that the true asymptotic level was closer to -5 mm Hg (millimeters of mercury). The finding that subjects tended to discount the physiological limits of increases to blood flow is consistent with the pattern of responses to several other questions.

Compliance

The venous system typically holds three to four times as much blood as the arterial system. However, venous capacitance can increase to accommodate 18 times as much as blood as in the arteries. Vascular compliance refers to the ability of a vessel to distend to accommodate more blood volume per unit pressure. The veins, particularly, the small veins and venules, are highly compliant vessels.

There were two questions that examined subject's understanding of the concept of compliance. They are as follows:

CM1 Explain the relationship between the compliance of a vessel and its ability to store volumes of blood?

CM2 Morphine is a drug known to increase venous capacitance.[27] What effect do you think it would have on cardiac output?

The first question asks about the defining quality of compliance. If a vessel is more compliant then it can distend to accommodate larger volumes of blood. The second question states morphine can increase venous capacitance and asks subjects to predict what would happen to cardiac output. When the

[27]Morphine can have various effects on cardiac output including the reduction of afterload. This question focuses only on its effect on capacitance.

capacitance of the venous system is increased, the veins can store more blood, and therefore venous return will decrease.[28] Consequently preload will decrease and cardiac output will decrease.

Every subject correctly asserted that the more compliant a vessel is the more blood it can store. Twelve out of fifteen subjects, except for the premedical student, a first year student (1.2), and a fourth year student (4.1), accurately predicted that an increase in capacitance would decrease cardiac output. Both the premedical student and the first year student (1.2) suggested that an increase in blood accumulation would increase pressure and increase the return of blood to the heart. The first year student struggled with two "theoretical models" of venous capacitance. An excerpt is illustrated below.

Excerpt from a first year student (1.2)

6. I'm thinking it would go down
7. because you pump into this huge well.

Model one, the well analogy, correctly predicted that flow would decrease. The second model is illustrated with another excerpt from his response.

Excerpt from a first year student (1.2)

8. But see one perspective I'm thinking
9. it could go up because
10. you'd definitely be filling all the time
11. and so when you pump,
12. you know you're always having,
13. you're having this greater pressure of venous blood
14. filling into the outflow
15. because there is more there.
16. So I would think that the pressure to fill up the ventricles would be more

[28]Compliance is defined as the ratio of a change in volume to a change in pressure. Capacitance refers the total volume over the total pressure.

17. based on that it seems like you'd have a greater cardiac output

The second model predicted that there would be a build up of pressure from venous blood that would increase output. Ultimately, he was not able to resolve the issue. Another first year student (1.1) grappled with the same issue, but was able to resolve the dilemma with better results. Here is an excerpt from her protocol:

Excerpt from a first year student (1.1)

3. To increase venous capacitance means
4. is it increases the amount of blood
5. that can be pushed into the veins.
6. Therefore, your increasing venous return,
7. and therefore you'd probably be increasing cardiac output,
8. so you'd have a ...(pause)
9. Does morphine do that?,
10. Is there something wrong with my logic?
11. I assume that it would increase cardiac output.
12. No hang on a second,
13. if I think back to the physics definition of capacitors,
14. it's the storing of energy,
15. uh by increasing the venous capacitance,
16. you'll increase how wide the veins can be,
17. how much they can actually keep in there
18. without returning to the heart first,
19. so you may actually decrease cardiac output.
20. That makes more sense in what I know about morphine.

The student began with the same premise that an increase in volume is going to increase forward flow. The subject then accessed an analogy to the physics term 'capacitors', which are energy-storing devices. This led her to reconsider the initial response and to conclude that blood will be stored in the veins, reducing cardiac output. There are two converging sources of evidence that caused her to change her response. The analogy with capacitors and the belief that morphine is unlikely to increase cardiac output.

Venous Resistance

Vascular resistance is the opposition to blood flow offered by the vessels. It is determined by the radius of the vessel and, to a lesser extent the viscosity of the blood. The pressure in the venous system is only about 10% as much as that of the arterial system. However, even small changes in venous resistance can strikingly impede venous return because it controls the outflow from the large compliance region of the circulation.

The subjects were presented with the following general knowledge question related to vascular resistance:

R1 Explain what is meant by vascular resistance?

The essential properties of vascular resistance are: it is an opposition to blood flow; it can be measured by the ratio of driving pressure to flow; and it is determined by the radius or diameter of the vessel. Subjects' response to this question coded against the correct responses are presented in Table 14.

The responses indicate that most subjects identified a core of the defining properties of resistance. Ten subjects suggested that the radius is the most important factor in determining resistance. Seven subjects stated that resistance can be defined as the ratio of driving pressure to flow. The most complete response was provided by a resident who detailed various systemic and pharmacological agents that influence resistance. Although, the other responses varied in their completeness, it is reasonable to assert that all subjects from the second year level onward understood the concept of resistance. The premedical student and a first year student (1.3) claimed that resistance reflects the resistance of the vessels to stretching or the distensibility of the vessel. This statement suggests that the subjects are

confusing compliance with resistance. A third year student (3.1) added the notion that resistance affects the compliance of a vessel. These concepts are related but conceptually distinct and physiologically independent.

Table 14

Subjects' Responses to Vascular Resistance Question Coded for Correct Answers.

Responses	P	1.1	1.2	1.3	2.1	2.2	3.1	3.2	4.1	4.2	R1	R2	Ph	CP	AC	Totals
A		1						1		1	1		1			5
B					1			1	1	1		1		1	1	7
C		1	1		1	1	1			1	1	1	1		1	10
D					1		1					1	1	1	1	6
E								1	1		1	1	1	1		6
Totals	0	2	1	0	3	1	2	3	2	3	3	4	4	3	3	34

A. An opposition to blood flow.

B. The vascular resistance is represented by the ratio of driving pressure to flow.

C. Determined by the radius or diameter of the tube or cross-sectional area.

D. It is determined by the viscosity or density of the fluid,

E. It is determined by the rigidity or elastic properties of the vessel.

Two questions addressed the issue of how venous resistance would affect venous return and cardiac output. They are as follows:

R2 How does a marked increase in venous resistance affect venous return and cardiac output?

R3 Predict what would happen to cardiac output, if the veins leading to the heart are suddenly compressed.

As mentioned previously, even small changes in venous resistance can impede venous return. Therefore, a marked increase in venous resistance will dramatically reduce venous return, and through the Starling mechanism,

reduce cardiac output. The veins *leading to the heart* (downstream from the compliance region) are resistance vessels and are most sensitive to changes in resistance. Venous return and cardiac output should drop precipitously when the veins are compressed. Subjects' response to this question coded against the correct responses are presented in Table 15.

Table 15

Subjects' Responses to Venous Resistance Question Coded for Correct Answers.

Q	Responses	P	1.1	1.2	1.3	2.1	2.2	3.1	3.2	4.1	4.2	R1	R2	Ph	CP	AC	Totals
R2	Venous return decreases		1	1	1		1		1		1		1	1	1	1	10
R2	Cardiac output decreases		1	1	1		1		1		1		1	1	1	1	10
R3	Cardiac output decreases	1	1	1	1			1	1	1	1	1		1	1	1	12
	Totals	1	3	3	3	0	2	1	3	1	3	1	2	3	3	3	32

Ten out of fifteen subjects correctly predicted that venous return and that cardiac output would consequently decrease if there was a marked increase in venous resistance. Twelve subjects correctly predicted that compressing the veins leading to the heart would result in a decrease in cardiac output.

There was a very interesting and consistent error pattern evident in the answers provided by the subjects who responded incorrectly. These subjects' suggested that venous resistance would diminish compliance and therefore increase blood flow. This misconception is illustrated in an excerpt and a semantic network (Figure 12) of a second year student's (2.1) response to the question concerning the effect of a marked increase in venous resistance on venous return and on cardiac output.

Excerpt from a second year student (2.1)

1. If you're talking about venous resistance,
2. it's going to increase venous return.
3. Um the large veins in front of the heart are capacitance vessels.
4. So they mainly store blood.
5. So if you increase the resistance of these vessels,
6. you're going to get more blood
7. flowing back to the heart.

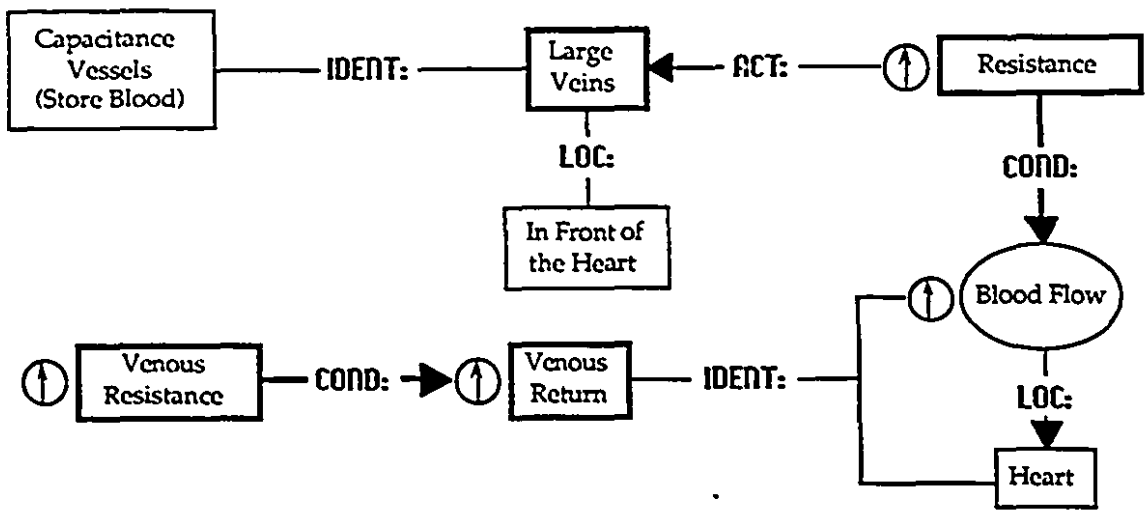

Figure 12: **Semantic Network Representation of a Second Year Student's (2.1) Response to Question R2.**

The subject reasons that since the large veins are storage vessels, an increase in resistance would diminish storage capacity and increase blood flowing back to the heart. The most significant error here is that the *large veins are storage vessels.* It is commonly taught that veins are "storers of blood". The large veins are downstream from the capacitance vessels, (the small veins and venules are compliance vessels) and are, in effect, resistance vessels that are critically important in determining blood flow. This misconception was evident in the responses of six subjects, including the premedical student, both fourth year students and both residents. However, in these subjects the misconception reflected a tension between two

competing forces, *venous resistance* and *venous compliance.* The second year student continually maintained that compliance was the determining factor and venous resistance can only affect compliance.

A third year subject (M3.1) clearly expressed the nature of the misconception in an excerpt below.

Excerpt from a third year student (3.1)

1. If your veins become more resistant,
2. I presume that means there scrunched down,
3. they become less compliant.
4. So for one thing their holding less blood.
5. It's at a slightly greater pressure.
6. So this will increase venous return.

The tension between resistance and compliance in the veins is most clearly reflected in response of a fourth year student (4.2) to the same question.

Excerpt from a fourth year student (4.2)

1. It sort of depends on how you take venous resistance.
2. Your venous vessel are very compliant,
3. and so that initially,
4. if you were to increase venous resistance,
5. you could perhaps look at it
6. as a loss of compliance of the vessels,
7. because you can't be compliant
8. and resistant at the same time.
9. They're inversely related.
10. so in that case,
11. I would think that initially,
12. perhaps, if you increase venous resistance
13. you would in fact increase venous return
14. and improve cardiac output.
15. But I think if your increasing venous resistance
16. enough to actually impede flow back to the heart,
17. which is perhaps what is meant
18. by a marked increase in venous resistance,
19. then I would say you would decrease venous return
20. and you would decrease cardiac output.

In this case, the *forces of resistance* triumph over the *forces of compliance* and the subject makes the correct predictions. The term *marked*, emphasizing the magnitude of the resistance, is the deciding factor in this subject's reasoning. Since the pressures are very small relative to the arterial system, even a small increase in resistance can substantially reduce cardiac output. The tension between these forces is suggested in the phrase "you can't be compliant and resistant at the same time, they're inversely related." Venous compliance and resistance are physiologically independent.

Both residents responded to some of the questions in a manner that would indicate that they could not disambiguate the effects of compliance from venous resistance. This is in evidence in the response of a resident (R2) to the question concerning the compression of veins leading to the heart, as illustrated in an excerpt and a semantic network below (Figure 13).

Excerpt from a resident (R2)

1. I think cardiac output would,
2. if the veins were suddenly compressed would increase...,
3. Certainly this would an extreme of a physiological mechanism,
4. whereby the ascent of the diaphragm
5. compresses the abdominal structures,
6. reducing venous capacitance,
7. which would increase the return of blood to the heart
8. and hence if increasing preload,
9. cardiac output.
10. I'm taking it that you don't compress the veins to such a degree
11. that the heart is flooded,
12. overwhelmed
13. and unable to give an increment of cardiac output flow,
14. doesn't increase beyond that.

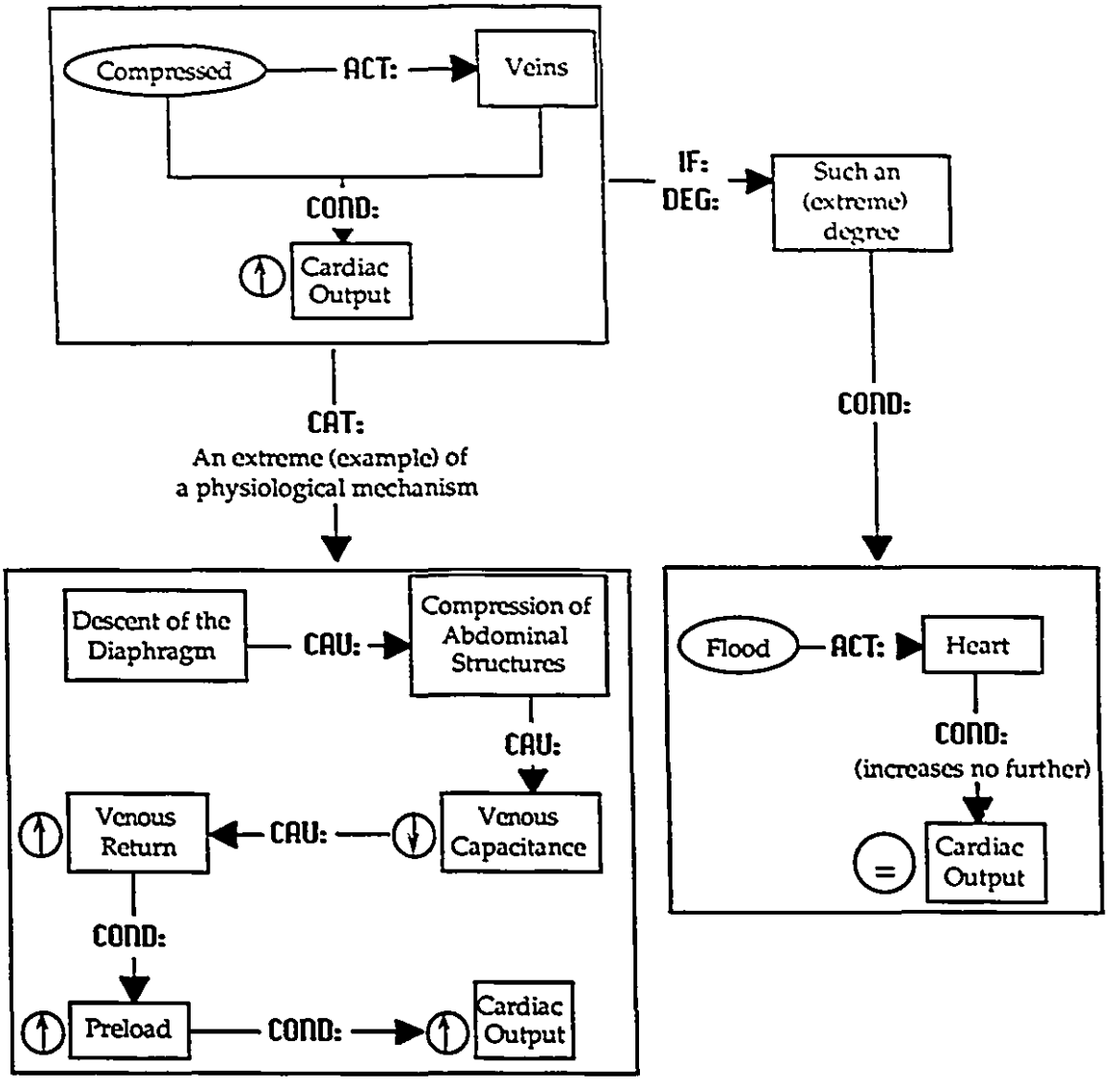

Figure 13: **Semantic Network Representation of a Resident's (R2) Response to Question R3.**

In the above example, the resident applied an inappropriate analogy from a common clinical situation whereby the diaphragm compresses the abdominal structures. This situation is typical of many medical conditions, such as asthma, where the lung inflation increases, and one can observe a sudden increase in respiratory rate and an increased blood flow. Extreme exercise is another example where the diaphragm would compress abdominal

structures and increase venous return. The second possibility suggested was that the compression of the veins could be so extreme so as to flood the heart, in which case, cardiac output could no longer increase. Compressing the veins leading to the heart would dramatically increase resistance and severely reduce venous return.

It should be noted that this misconception concerning the effect of resistance was specifically tied to venous resistance. Each of the subjects, who exhibited this misconception, correctly pointed out that an increase in *arterial resistance* would increase the afterload and therefore reduce cardiac output. There are several bits of erroneous knowledge and beliefs that contribute to this misconception: 1) the belief that venous resistance and compliance are inextricably intertwined resulting in a composite schema where the effects of one can not be differentiated from the other; 2) the notion that the large veins are storage vessels, when they are, in fact, resistance vessels; 3) a malprioritization of factors resulting in a misjudgment concerning the primary effect of resistance; and 4) the use of inappropriate clinical analogies.

Many misconceptions are grounded in experience and reflect an acceptance of the primacy of experience and intuition over counter-intuitive formal teachings. However, formal learning can also result in the development of significant misconceptions. Resistance is a concept that is well grounded in experience. It corresponds to what diSessa (1983) refers to as a phenomenological primitive or p-prim. People have a sense that resistance refers to the slowing down or interference with some process (diSessa, 1983). Given that these subjects (with the exception of the premedical student) clearly understand the concept of resistance, it is almost certain that this misconception is a function of formal learning experiences.

Summary

This section presented the results of subjects' responses to questions pertaining to venous return. The questions focused on the concepts related to pressure-volume and pressure-flow relationships on the venous side, specifically, *mean systemic pressure*, *stressed volume*, and *right atrial pressure*. In addition, the section presented subjects' response to questions concerning the primary determinants of venous return: *venous compliance* and *venous resistance*.

The results suggest that, in comparison to the cardiac output concepts, subjects did not understand the primary concepts related to venous return very well. In particular subjects experienced difficulty explaining the concepts of mean systemic pressure and stressed volume. Only six subjects were able to characterize the defining properties of mean systemic pressure. Several subjects, including a resident (R2) and the cardiologist practitioner, equated this pressure with mean arterial pressure. Similarly, only six subjects could define stressed volume as the volume that contributes to pressure within the vasculature.

This pattern is consistent with the responses provided by subjects in responding to the open-ended venous return question. Few subjects were able to discuss at any length the factors that affect blood returning to the heart. Subjects were generally able to discuss the factors that regulate cardiac output at greater length. To some extent that was also evident in the accuracy of subjects' predictions. Ten out of fifteen subjects, including all subjects at each level up to and including third year medical students, predicted a higher percentage of correct cardiac output questions than venous return question.

The advanced group predicted a considerably higher percentage of correct venous return outcomes (80%) than the medical students (67%). However, two physicians, a resident (R2) and the cardiologist practitioner, experienced considerably more difficulty in responding to the venous return questions than the cardiac output questions. The physiologist and academic cardiologist provided the best explanations and predicted the highest percentage of correct responses. A fourth year student predicted every venous return outcome, even though his explanations indicated a couple of conceptual errors.

A misconception was evident in the responses of six subjects, including both fourth year students and both residents. It was related to a confounding of venous resistance and venous compliance. The notion is that since an increase in venous resistance is associated with a decrease in compliance, then the net effect of resistance would be to increase venous return. If one considers, the meaning of resistance, which most of these subjects clearly understood, then it appears quite counterintuitive that resistance can facilitate blood flow. However, consider the vessels in Figures 14a and 14b. It becomes apparent how one can see resistance and compliance as competing forces, with a net effect of a flatter and less distensible vessel, as suggested by Figure 14c. A less compliant vessel would then increase the return of blood to the heart.

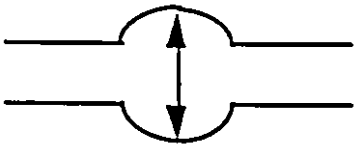

Figure 14a. Compliance Vessel

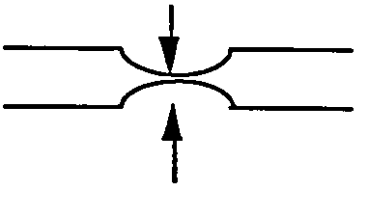

Figure 14b. Resistance Vessel

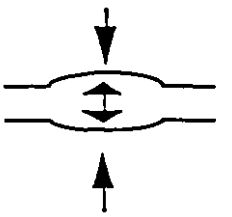

Figure 14c. The Effects of Resistance on a Compliant Vessel

Integration Questions

The circulatory system is a closed system and therefore, cardiac output must inevitably match venous return. These two major segments of the circulatory system are affected by different factors. The right atrium is considered the anatomical point where both circuits converge. The right atrial pressure serves as the back pressure for venous return and as well, the forward flow pressure (the pressure that determines the preload) for cardiac output. Right atrial pressure is influenced by the amount of blood returned to the heart, and the contractile state of the heart. This pressure mechanically couples cardiac function to the systemic circulation by directly affecting the pressure gradient for venous return (Green, 1987). An increase in cardiac function produces a decrease in right atrial pressure, which allows for a greater venous return. A greater venous return increases cardiac output through the Starling mechanism.

There are four primary integration themes considered in this section. They include the fact that changes in cardiac function affect the determinants of venous return, only through changes in right atrial pressure. Changes in cardiac function can change right atrial pressure and affect venous return. A second theme that emerges is that changes in venous return exert a more immediate effect through the Starling mechanism. A third theme is that there is an upper boundary to which the heart cannot further increase output

in response to additional volumes of blood. The final theme considered is that right atrial pressure mechanically couples cardiac output and venous return.

Cardiac Factors Affecting Venous Return

As discussed previously, changes in cardiac function affect the pressure gradient for venous return by changing the right atrial pressure. Changes in arterial pressure are not easily transmitted across the arterioles (small arteries). Changes in pressure on the arterial side typically do not have any immediate consequence for venous return because of the large compliance region upstream from this pressure which attenuates pressure changes.

There were five questions related to issues of how cardiac function affects venous return. They are as follows:

INT1 Explain how a decrease in left ventricular contractility affects venous return.[29]

INT2 How does a marked increase in arterial resistance affect venous return and cardiac output?

INT3 What is the immediate effect of a significant decrease in cardiac output on mean systemic pressure?

AP1 If the heart suddenly became hypoeffective, without any significant changes in the systemic blood vessels, what would immediately happen to venous return prior to any reflex adjustments (within the first few heart beats)?

INT4 Explain what would happen to blood entering the right atrium if the arterial flow into the systemic circulation were suddenly stopped (e.g., by clamping the aorta).

[29]There are two questions which initially appeared in different sections of the study, INT1 appeared in the basic physiology section, and Ap1 was included in the applied section. Both questions are included in this analysis because they address issues related to integration.

These questions all deal with the existing mechanisms that ensure that venous return matches cardiac output. The first question asks about how a decrease in left ventricular contractility affects venous return. The correct response would identify that a decrease in left ventricular contractility results in a higher left atrial pressure, which will be transmitted through the pulmonary veins and arteries and eventually effect the right heart. This results in a downward shift of the cardiac function curve. This results in a higher right atrial pressure and a decrease in venous return.

The second question asks about the effects of a marked increase in arterial resistance. A correct response would include the information that an increase in arterial resistance results in an increase in afterload on the left ventricle, which will result in a transient decrease in output from the left heart, and in higher diastolic filling pressures on the left heart, and eventually on the right heart. This increase is represented by an increase in right atrial pressure that will decrease the gradient for venous return.

The third question asks about an immediate effect of a significant decrease in cardiac output on mean systemic pressure. An accurate response would indicate that a decrease in cardiac output has no effect on mean systemic pressure. Mean systemic pressure is solely determined by stressed volume and vascular compliance. The fourth question (Ap1) presents a scenario in which cardiac function is diminished. If the heart suddenly became hypoeffective, then it would be unable to pump at full capacity and right atrial pressure would rise until the pressure gradient reduced venous return to match cardiac output. The final question (INT4) presents a very similar scenario in which flow out of the left ventricle is completely obstructed. The question focuses on what happens to venous return. The answer is that compliant vessels on the venous side have a reserve of blood

and flow would continue for some time (at least 30 seconds) until right atrial pressure rises to equal venous return.

Subjects' response to these questions coded against the correct responses are presented in Table 16. Ten subjects responded to the first question by suggesting that venous return decreases. Nine subjects described an intervening process in which mechanical events induce a change, reducing venous return. Five other subjects, including the premedical student, two first year students (1.1 & 1.3), a second year student (2.1) and the cardiologist practitioner predicted a more immediate effect. Four subjects, including a second year student (2.1), a fourth year student, a resident (R1), and a physiologist stated that venous return would be unaffected by changes in left ventricular contractility.

The subjects responded to the question concerning the effects of a decrease in left ventricular contractility on venous return (INT1) by alluding to the backup of volume and raised pressures resulting in a decrease in venous return. A third year student (3.1) erroneously suggested that a decrease in arterial pressure would propagate across the venous system and you would have less forward pressure driving venous return.

Table 16

Subjects' Responses to Integration Questions Pertaining to Cardiac Output Factors Affecting Venous Return Coded For Correct Answers.

Q	Responses	P	1.1	1.2	1.3	2.1	2.2	3.1	3.2	4.1	4.2	R1	R2	Ph	CP	AC	Totals
Int1	Intervening process						1	1	1	1	1	1	1	1		1	9
Int1	Venous return decreases	1	1		1	1		1	1		1		1		1	1	10
Int2	Cardiac output decreases		1	1		1	1	1	1		1		1	1	1	1	11
Int2	Venous return eventually decreases		1	1				1	1		1		1	1	1	1	9
Int3	No immediate change in mean systemic pressure					1	1				1			1			4
Ap1	Intervening process	1				1	1	1	1	1	1	1		1		1	10
Ap1	Venous return Increases	1	1	1	1				1		1	1	1	1	1	1	11
Int4	Flow would continue for a period of time.		1	1	1		1	1	1	1	1	1	1	1	1	1	13
Int4	Flow would trickle down to zero		1	1	1	1	1	1	1	1	1	1	1	1	1	1	14
	Total	3	6	5	4	5	6	7	8	4	9	5	7	8	6	8	91

The academic cardiologist provided a response that suggested different ways in which the venous and arterial systems achieve integration. This illustrated in a semantic network in Figure 15.

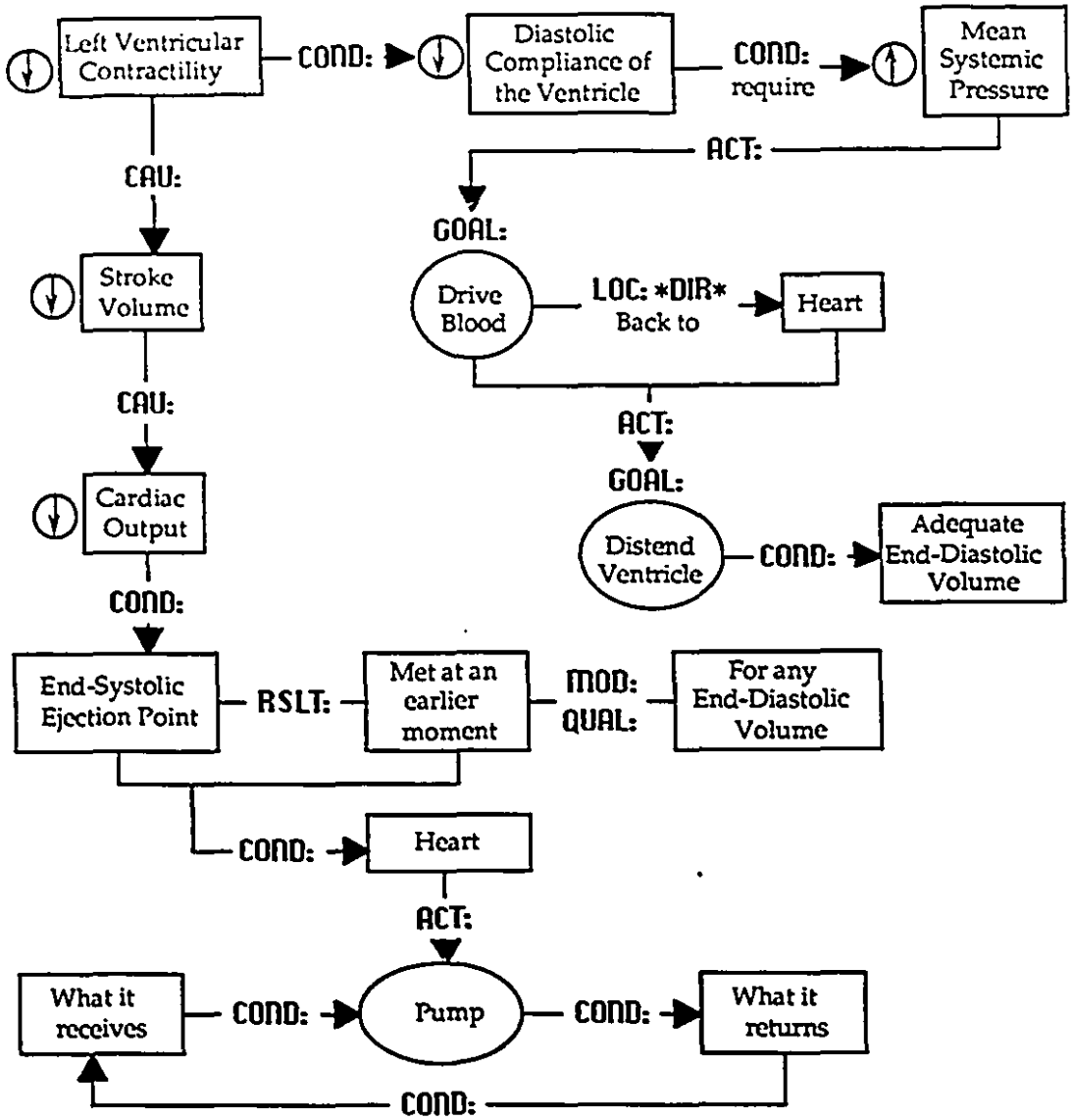

Figure 15. **Semantic Network Representation of Academic Cardiologist's Response to Question INT1.**

The cardiologist proposed two alternative mechanisms in which venous return would match cardiac output. The first mechanism results from the fact that an increase in contractility leads to a decrease of the compliance of the left ventricle. This requires an increase in mean systemic pressure to drive blood back to the heart and distend the ventricle to an adequate end-

diastolic volume. In this way, venous mechanisms compensate by delivering a larger load to the heart, presumably to increase cardiac output.

The second mechanism is suggested by the relation between contractility and stroke volume. A decrease in contractility causes a decrease in stroke volume which in turn causes a decrease in cardiac output. This leads to a shift in the end-systolic ejection point, which will be met at an earlier moment for any end-diastolic volume. One can conceptualize this as a downward shift in the slope of the end-systolic pressure-volume line that is a measure of contractility. This decrease results in a smaller loop with less volume getting out and initially, the same volume coming back.

Another way to interpret this chain of inferences is in terms of cardiac function and venous return curves. The intersection for cardiac output and venous return are routinely plotted on curves. This technique provides a method for examining the effects of various determinants of both venous return and cardiac output and determine at which output value they intersect. This format is commonly presented in textbooks, and is also used as a research tool. This cardiologist (AC) frequently represented information in these questions and problems in terms of cardiac function and venous return curves. Other advanced subjects used this approach as well, although somewhat less successfully.

The second question addresses the effects of an increase in arterial resistance on cardiac output and venous return. Eleven subjects correctly predicted that there is a decrease in cardiac output. Nine of these subjects suggested that venous return would also decrease. The premedical student maintained that increases in resistance lead to increases in pressure which always result in increased output. A fourth year student (4.1) suggested that arterial resistance produces an increase in afterload. On the basis of his

previous analysis of the pressure-loop diagram, suggesting that afterload did not affect cardiac output, he also indicated that arterial resistance does not affect output. A third year student gave the correct answer, but also considered two other models. The following is an excerpt from her response:

Excerpt from a third year student (3.1)

1. Arterial resistance increase,
2. increased arterial resistance increases afterload,
3. so your cardiac output goes down,
4. therefore your venous return also goes down

In the first analysis, she correctly predicted a decrease in both cardiac output and venous return due to afterload. She then considered another possibility, as illustrated in the following excerpt:

Excerpt from a third year student (3.1)

5. Well if your arterioles,
6. if your smooth muscle all went into spasm
7. and your arterioles squished down,
8. then the pressure would be increased
9. and the blood would be driven towards the capillaries
10. and may end up in the veins
11. and your venous return would increase.

The second analysis introduced the possibility that an increase in pressure would propel blood into the venous system and increase venous return. She suggested yet a third possibility in the excerpt below.

Excerpt from a third year student (3.1)

12. It would still depend on the capillaries in between.
13. And if your capillaries transmit the pressure
14. then the venous pressure will increase.
15. Whereas if they buffer it
16. and just take it in the stomach
17. and dilate then nothing will happen.

The third analysis, suggested that the increase in pressure would likely be buffered by the capillaries, which is essentially correct, although the buffering really takes place in the arterioles. The last possibility reflects a tension between two competing models of whether pressure is transmitted across the capillaries from the arterial side to the venous side or vice-versa. Pressure is not transmitted across the capillaries. This issue is central to arterial-venous integration. Several subjects struggled with this issue in responding to questions in this section.

The third question asks about the immediate effect of a significant decrease in cardiac output on mean systemic pressure. Only four subjects suggested that there would be no immediate change in mean systemic pressure. Ten subjects predicted that mean systemic pressure would decrease. Their responses suggest a misunderstanding of the concept. Two excerpts are illustrated below.

Excerpt from a first year student (1.1)

1. Immediate effect of a significant decrease,
2. If you decrease cardiac output,
3. you're immediately going to drop mean systemic pressure,
4. since the first pressure that you're taking to measure mean systemic pressure drops,
5. which is the amount of blood going to the arteries.

Excerpt from a third year student (3.1)

1. Mean systemic pressure would go down.
2. It will go down
3. because blood will come out
4. of the other end of your arterial tree
5. into your capillaries
6. and less will come in.
7. So your pressure gradient will be lower
8. across your arterial system.

The first excerpt suggested a general notion that mean systemic pressure is an average pressure, and a drop in arterial pressure would immediately shift the average downward. The second excerpt, from a third year student, indicated that mean systemic pressure is equated with mean arterial pressure.

In an earlier section, we had determined that six subjects were able to characterize the defining properties of mean systemic pressure. Three of these subjects, including a second year student (2.1), a fourth year student and the physiologist correctly predicted that there would be no effect. One other subject (2.2) also predicted the correct outcome. The two subjects who were able to define mean systemic pressure, proposed interesting predictions. The resident (R1) suggested that mean systemic pressure would rise, but did not elaborate. We can speculate that he was suggesting that mean systemic pressure would rise to maintain cardiac output. The academic cardiologist initially predicted that it would rise, but his analysis failed to yield a conclusive answer.

Question 4 (Ap1) presented a situation in which the heart suddenly becomes hypoeffective and subjects are asked to predict what would happen to venous return prior to any reflex adjustments. This question is analogous to the question concerning the effects of a decrease in left ventricular contractility. Eight of the subjects responded using a similar line of reasoning to the contractility question (INT1), that is to say, there is an intervening process that eventually results in a reduction of venous return to match cardiac output.[30] Four of these subjects responded correctly to both

[30]There are two inherent difficulties with these questions. One has to do with whether the time frame suggested by the question corresponds with subjects' understanding of events

questions, including both third year students, a fourth year student (4.2), and the academic cardiologist. Six subjects answered this question correctly, four subjects suggested that there would be no change to venous return and another five indicated that there would be an immediate reduction in venous return.

The question posed some trouble for the resident and the cardiologist. Here is an excerpt from his response:

Excerpt from a Resident (R2)

1. Well I think within the first couple of heart beats,
2. I think you would see a decrease in your venous return,
3. assuming a lot of things are constant
4. because the gradient for venous return is reduced.
5. This is very physiological,
6. holding things constant within that time.

The subject raises the issue that the question "is very physiological", alluding to the fact that it is somewhat contrived and not typical of what one may see in a clinical situation. Other questions that describe experimentally controlled situations tended to present some difficulties for both residents and the academic cardiologist. Patients can present with a myriad of problems, and one cannot easily isolate a single aspect of their physiological state from all the other complicating reactions.

The last question presented relatively few difficulties for the subjects. Thirteen out of fifteen subjects recognized that there is a reserve of blood in the venous system and sufficient pressures to maintain flow for some time. The flow of blood would then come to a stop as the vascular system emptied,

---

within the cardiac cycle. The second problem has to do with coding the subjects' response. It is sometimes difficult to ascertain whether they are describing an *intervening process.*

and the mean systemic pressure would equal right atrial pressure. The physiologist suggested a rather interesting response as illustrated in the following excerpt:

Excerpt from the physiologist

1. You'd still have blood entering into the right atrium,
2. So long as you maintain the stressed volume
3. in the systemic part of the circulation
4. independent of whether anything is coming out or not.
5. Once you went down below the point
6. where there is no more stressed volume.
7. if you didn't have any kind of compensatory mechanisms,
8. then your return would fall off.
9. It would fall to nothing.
10. As long as you maintained the stressed volume
11. and mean systemic pressure,
12. so I think within the first beat
13. you wouldn't see anything,
14. but thereafter,
15. gradually as the mean systemic pressure fell,
16. you'd have less and less return.

The idea that the pressure gradients would equalize is correct. The subject suggested that a minimal level of stressed volume would maintain a certain mean systemic pressure and as stressed volume would continue to decrease, so would mean systemic pressure. However, as the vascular system emptied, stressed volume might even go up slightly, since the left ventricle is prevented from pumping blood into the peripheral circuit. It is right atrial pressure that would continue to rise and eliminate a pressure gradient for venous blood flow.

Venous Return Factors Affecting Cardiac Output

Changes in venous return tend to have an immediate and profound effect on cardiac output. An increase in flow into the right atrium produces an increase in preload within physiological limits. However, the pressures in the venous system are not transmitted back to the arterial system for two reasons: the pressures are minimal in the venous system relative to the arterial system, so that a very significant change in venous pressures represents only a very marginal increase in arterial pressures; secondly, in the arterioles, vessels act as *waterfalls* which collapse under critical pressures. Therefore, changes in downstream characteristics do not affect the arterial pressure-flow relationship.

There were five questions related to issues of how venous return affects cardiac output. They are as follows:

R2 How does a marked increase in venous resistance affect venous return and cardiac output?

CM2 Morphine is a drug known to increase venous capacitance. What effect do you think it would have on cardiac output?

INT5 Explain the effect a sudden increase in mean systemic pressure from its normal value of 7 mm Hg to 12 mm Hg would have on cardiac output.

R3 Predict what would happen to cardiac output, if the veins leading to the heart are suddenly compressed.

INT6 When the amount of blood flowing into the heart is unlimited (i.e., from large reservoirs with no collapsible tubes), what happens to cardiac output?

Two of these questions were previously discussed in the section on venous resistance (R2 and R3), and another was discussed in relation to venous compliance (CM2). The analysis of responses to these questions, presented

here, focus only on the issue of integration. In brief, factors that decrease venous return, as described in these three questions, cause a decrease in cardiac output. A sudden increase in mean systemic pressure (INT5) would cause an increase in the pressure gradient of venous return and therefore increase the preload.

The final question in this section (INT6) presents a hypothetical situation in which an unlimited supply of blood is available to be pumped by the heart. The question is to determine what happens to cardiac output. The correct answer is that cardiac output will rise until it reaches a plateau of the cardiac function curve, as suggested by the Starling mechanism. At this point, right atrial pressure continues to rise but cardiac output can no longer increase. Subjects' response to these questions coded against the correct responses are presented in Table 17.

The responses indicated that subjects clearly understood the immediacy of a change in venous return on cardiac output. When subjects correctly determined the effect of a change in a variable, such as resistance or compliance on venous return, they were able to correctly predict the outcome for cardiac output. For example, the same ten subjects who predicted that an increase in venous resistance would reduce venous return, also predicted that cardiac output would decrease.

Table 17

Subjects' Responses to Integration Questions Pertaining to Venous Return Factors Affecting Cardiac Output Coded For Correct Answers.

Q	Responses	P	1.1	1.2	1.3	2.1	2.2	3.1	3.2	4.1	4.2	R1	R2	Ph	CP	AC	Totals
R2	Venous return decreases		1	1	1		1		1		1		1	1	1	1	10
R2	Cardiac output decreases		1	1	1		1		1		1		1	1	1	1	10
Cm2	Cardiac output decreases		1		1	1	1	1	1		1	1	1	1	1	1	12
R3	Cardiac output decreases	1	1	1	1			1	1	1	1	1		1	1	1	12
Int5	Cardiac output increases		1		1	1	1	1				1		1	1	1	9
Int6	Cardiac output decreases	1	1	1	1	1	1	1	1	1	1	1	1	1	1	1	15
Int16	Cardiac output reaches a plateau						1	1		1				1	1	1	6
	Total	2	6	4	6	3	6	5	5	3	5	4	4	7	7	7	74

There was an interesting conceptual error expressed by a fourth year student (4.2) in answering question R3. This is illustrated in the following excerpt:

Excerpt from a fourth year student (4.2)

1. Okay, if you compress the veins going to heart,
2. you decrease venous return,
3. therefore you cannot put out as much blood on the next beats,
4. by the same token
5. your increasing afterload,
6. therefore giving the heart a bigger gradient to pump against
7. and that will also affect cardiac output.

The subject correctly predicted a decrease in both cardiac output and venous return. However, he erroneously explained that an increase in venous resistance would be transmitted across the capillaries and would therefore increase the afterload. This line of reasoning was also evident in his responses to at least two other questions.

Nine subjects predicted that an increase in mean systemic pressure would result in an increase in cardiac output. It is noteworthy that twelve subjects had suggested that a decrease in mean systemic pressure would affect venous return. Four out of the six subjects who were able to define the essential properties of mean systemic pressure correctly predicted this outcome. The two fourth year students who had expressed an understanding of the concept, correctly predicted that an increase in mean systemic pressure would affect venous return, but not cardiac output. This is exemplified in the following two excerpts:

Excerpt from a fourth year student (4.1)

1. Cardiac output is stroke volume times heart rate,
2. well an increase in mean systemic pressure,
3. ya a decreased cardiac output.

Excerpt from a fourth year student (4.2)

1. A sudden increase in mean systemic pressure,
2. I don't see why it should have an effect at all on cardiac output.

The responses from both subjects suggest that although they can define mean systemic pressure, they did not really understand that it represents a significant pressure in the venous system. This is the most likely conclusion, since both subjects appeared to understand the Starling mechanism and as well, the relationship between changes in venous return and its effects on cardiac output.

Each of the 15 subjects correctly predicted that there would be an initial increase in cardiac output in response to an unlimited flow of blood into the heart. The experimenter would question whether the subject believed that there would be a continuing linear increase, if subjects did not qualify their responses, by suggesting that there was an upper limit to this increase. Six subjects, including both cardiologists, the physiologist, a fourth year student (4,1), a third year student (3.1), and a second year student (2.2), predicted that cardiac output would plateau at a given point. Only the cardiologist practitioner specifically discussed the Frank-Starling mechanism. He provided the most complete explanation. The following is an excerpt from his response.

Excerpt from the cardiologist practitioner

1. Well initially you're going to increase your
   cardiac output um.
2. I presume this is one of the only places
3. where you'd ever get up to the plateau of the
   Frank-Starling mechanism.
4. Your cardiac output increases
5. with increasing left ventricular diastolic
   volume.
6. But there is a plateau
7. and uh I take it that at some point in time
8. it reaches that plateau
9. and it doesn't increase anymore
10. and you probably
11. The consequence is you'd eventually would have a
    backup
12. an increased pressure in the right atrium
13. which will decrease the flow from this unlimited
    source
14. and you will reach some new equilibrium
15. at the plateau of the Frank Starling curve.

The subject recognized that this a situation in which one reaches the plateau of the Frank-Starling mechanism and cardiac output no longer increases. This will cause an increase in right atrial pressure and this will decrease flow.

Five subjects, including a resident (R1), three first year students and the premedical student, insisted that cardiac output would keep increasing linearly. Four subjects, including a resident (R2), a fourth year student (4.2), a third year student (3.2) and second year student (2.1) predicted that cardiac output would increase until a point at which it will go into failure. This illustrated by the following excerpts:

Excerpt from a third year student (3.2)

6. Well, there is probably a cut off point beyond
7. which the pump will fail.

Excerpt from a fourth year student (4.2)

7. Now if your giving a great deal of blood to the heart,
8. your going to reach the point
9. where you start coming down off your
10. your myocardial contractility curve.
11. As you increase and increase and increase the stretching of the heart,
12. you get to the point .
13. where the heart's systolic function is compromised
14. and you'll find that you will decrease cardiac output.

Excerpt from a resident (R2)

3. But at some point you would overwhelm the capacity of the heart
4. to such a large preload
5. and it would start failing.
6. In which case you would see a drop in your cardiac output.

The common element in these excerpts is that these subjects are suggesting that cardiac output does not reach a level at which it plateaus, rather it gets to a point in which the pump is compromised and the heart could go into

failure.[31] The fourth year student suggested that the heart stretches to a point "where the heart's systolic function is compromised". This would appear to be a clear example of the 'mechanical overstretching' misconception documented by Feltovich and colleagues (1989) and discussed in detail in chapter 4. The focal point of the misconception is that heart failure results from an overstretching of the fibers. The critical elements in the misconception, as documented by Feltovich (1989) is the belief that the behaviour of skeletal muscle is isomorphic to the behaviour of cardiac muscle and that individual isolated muscle fiber is an accurate reflection of the behaviour of an intact ventricle (chamber of the heart). Feltovich has showed that students reason that the length-tension relationship is isomorphic to the pressure-volume relationship in a contracting ventricle.

This observation led to a re-evaluation of subjects' responses to previously considered questions related to preload and the Frank-Starling mechanism. There was clear evidence that this misconception characterized the reasoning of both the fourth year student (4.2) and the resident (R2). This misconception was particularly apparent in the responses to the following two questions.

P1 How does the Frank-Starling mechanism regulate cardiac output?

P3 Explain the effects of incremental changes in preload on peak systolic ventricular pressure.

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[31]It is noteworthy that the original experiments by Frank and Starling suggested that there was a downward slope to the cardiac function curve. However, subsequent research has indicated that this result was a due to a methodological flaw in those previous experiments (Sagawa, 1978).

The misconception is illustrated in the following excerpt from the fourth year student (4.2) to question P1:

Excerpt from a fourth year student (4.2)

3. The way the cardiac muscle works is that,
4. as you stretch the cardiac muscle,
5. you actually get, um,
6. initially, you would get an increase in contractility,
7. which will actually improve your cardiac output.
8. Once you get to the point where your,
9. where your muscle sarcomere is no longer overlapping very well,
10. you start compromising cardiac output.
11. So initially it will improve it.
12. But as you increase the wall tension,
13. you will start decreasing cardiac output,
14. after a certain point.

E. Can you relate that to pressure-volume.

15. Okay,
16. as you get to an increase volume,
17. uh at a certain,
18. as you increase and increase the volume,
19. for example,
20. as you increase and increase the preload,
21. you will eventually start compromising cardiac output.
22. Although initially you will certainly improve cardiac output.

The student stated that an increase in the stretch of the cardiac muscle beyond a certain point result in a situation in which the sarcomere (subcellular structures that are the contractile units of a myocardial cell) no longer overlap. This characterizes the down side of the length-tension relation in an isolated strip of contracting muscle. However, there is no such analog in the intact heart. The subject clearly suggested that length-tension relationship in an isolated strip is isomorphic to the pressure volume relationship in the intact heart. A similar response to the same question was suggested by the resident as indicated in the following excerpt:

Excerpt from a resident (R2)

16. Well if the loading pressure is reduced,
17. um what would happen?
18. You would travel along the curve towards the left
19. and I guess down just along the length of the curve
20. and this would result
21. result in a decreased cardiac output.
22. This curve is notable in that
23. it tends to have a decrease in slope,
24. as it approaches higher loading conditions.

The excerpt suggests that the cardiac output curve is seen as having a decrease in the slope at higher loading conditions. The implication of this misconception in the intact heart is indicated in the resident's response to question P3, as illustrated in the following excerpt:

Excerpt from a resident (R2)

1. We know from the Starling mechanism that initially,
2. depending on which part of the curve you are on,
3. for a normal contractile ventricle,
4. changes, changes at the lower end,
5. at the left end of the curve,
6. which will show a more marked increase in your peak ventricular pressure.
7. I take that back.
8. I think what happens with that initially as increased output
9. with stroke volume
10. and hence an increased systolic ventricular pressure
11. generated up to a certain point,
12. beyond which it becomes detrimental
13. and then systolic ventricular pressure
14. may actually fall.
15. As the myocardium starts failing
16. to accommodate the larger preload.

The focal issue here is that there is an upper boundary for the Starling mechanism and that the cardiac function curve has a downward slope. The subject also asserted a connection between this part of the slope and heart

failure. This connection was indicated by the statement that at larger preloads, the myocardium starts failing. In a clinical situation, one may actually observe a downward slope of a curve, where cardiac output decreases with increased filling. However, it is not a consequence of changes in loading conditions, rather it is a function of ischemia or deterioration of the cardiac muscle wall.

These findings very closely mirrors the misconception documented by Feltovich and colleagues, albeit in a very different context. In this section, we are interested in the integration of venous return and cardiac function and particularly the Starling mechanism. It is probable that this misconception, the belief that the heart begins to fail at higher preloads, is intertwined with a lack of understanding of the coupling of venous return and cardiac output.

Right Atrial Pressure as a Coupling Mechanism

As discussed previously, the right atrial pressure plays a pivotal role in the coupling of venous return and cardiac output. It is convenient at this point to introduce a graphic analysis of the intersection of cardiac output and venous return. Many textbooks (e.g., Green, 1987) use this kind of graphical analysis to explain the functional relationship between various factors and systemic blood flow. This diagram, as illustrated in Figure 16, can be used to elucidate the function of right atrial pressure.

Right atrial pressure is represented on the X-axis (millimeters of mercury), and flow is represented on the Y-axis (liters of blood per minute). On the left side of the Y-axis are three possible venous return curves and on the right hand side are three possible cardiac outputs. The intersecting curve represents the actual flow for a given right atrial pressure. At any one time, there can only be a single value for return and output. Different venous and

cardiac factors can shift the curve to the right or left or move the slope up or down.

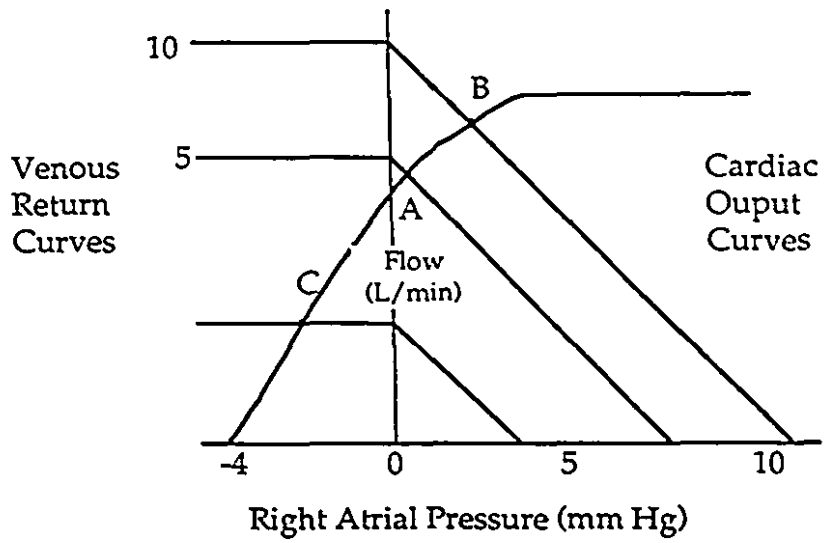


Figure 16. **Graphic Analysis of the Intersection Between Venous Return and Cardiac Output. Adapted from Green (1987, p.90).**

Subjects were asked the following question concerning the nature of right atrial pressure:

RAP `What role does right atrial pressure play in the interaction between cardiac output and venous return?`

The questions suggests that right atrial pressure plays a role in integrating cardiac output and venous return. The range of correct answers include the fact that right atrial pressure: is the intersection point between the cardiac output and venous return curves; it is the back pressure for venous return; it is a measure of the preload on the right ventricle; and when cardiac function is increased, right atrial pressure is decreased, allowing a larger

gradient for venous return. Table 18 presents a list of the correct answers coded against subjects' responses.

Table 18

Subjects' Responses to Right Atrial Pressure Question to Coded for Correct Answers (RAP).

Responses	P	1.1	1.2	1.3	2.1	2.2	3.1	3.2	4.1	4.2	R1	R2	Ph	CP	AC	Totals
A		1	1	1	1	1	1	1	1	1	1	1	1	1	1	14
B		1	1		1	1	1	1		1	1				1	9
C																0
D													1		1	2
E										1					1	2
Totals	0	2	2	1	2	2	2	2	1	3	2	1	2	1	4	29

A. The right atrial pressure is the back pressure for venous return.

B. It is a measure of the preload of the right ventricle.

C. As cardiac function is increased, right atrial pressure is decreased.

D. Right atrial pressure is the intersection point between the cardiac output and venous return curves.

E. Starling mechanism

Every subject, except for the premedical student, stated that the right atrial pressure is a back pressure for venous return. Nine subjects, including, two first year students (1.1 & 1.2), both second and third year students, a fourth year student (4.2), a resident (R1), and the academic cardiologist, suggested that it is a measure of preload on the right ventricle. Four subjects, including a first year student (1.3), a fourth year student (4.1), and a resident (R2), focused on the fact that right atrial pressure affects the venous return gradient, which in turn affects cardiac output. The cardiologist practitioner discussed the issue of right atrial pressure as the back pressure for both systemic circulation and venous return. A resident used a clinical

analogy to answer the question. The following excerpt was taken from his response:

Excerpt from a resident (R2)

1. The venous return does depend on right atrial pressure
2. to facilitate this return.
3. Assuming if you have a stimulus
4. which elevates your right atrial pressure
5. such as tricuspid stenosis,
6. too such a high degree
7. that you won't have an adequate venous return
8. because of uh decreasing
9. your pressure differential,
10. and edema peripherally.
11. It could cause a drop in your cardiac output.

The subject alluded to a clinical condition, tricuspid stenosis, which is characterized by a constriction of the valve between the right atrium and the right ventricle. This situation provided an exemplar in which a change in right atrial pressure affects both cardiac output and venous return. However, the focus in the subjects' response was exclusively on the role of right atrial pressure as an opposing pressure for venous return.

Not a single subject mentioned that an increase in cardiac function would decrease right atrial pressure. Only two subjects, the physiologist and the academic cardiologist, made any reference to the intersection between cardiac function curves and venous return curves (see Figure 16). Although, most of the advanced subjects, including each of the physicians and the fourth year students would make reference to these curves in responding to other questions. What is most surprising is that none of the second or third year students used the curve as a basis for explaining the role of right atrial pressure because these subjects had most recently received instruction in this subject. Both the lectures presented to these subjects and the textbook used

for the course, Green's Cardiovascular and Pulmonary Physiology (1987), made ample use of these diagrams as explanatory constructs. One third year student attempted to recall the graph, but could not. This might suggest that either these graphic representations were not understood or that they failed to make a significant impact on subject's thinking about the domain content.

The physiologist focused entirely on these curves in his explanation of the role of right atrial pressure. The following excerpt provides an illustration:

Excerpt from the physiologist

1. Um, right atrial pressure um,
2. are you looking for the intersection of the two curves?
3. Because where they cross depends on the slope of the relationship,
4. of the venous return curve
5. and as the right atrial pressure changes
6. the slope of that line changes
7. the intersection point between
8. the venous return curve and the cardiac output
9. will move up or down.
10. So for example if right atrial pressure became lower,
11. moved to the left that is,
12. the intersection would move down
13. and your cardiac output would go down.
14. Whereas if right atrial pressure would move up to the right
15. and your cardiac output would go down.

This excerpt provides an interesting juxtaposition to the resident's (R2) response. The physiologist, who engages in basic science resea: .h, would very likely make frequent use of similar diagrammatic representations in analyzing functional relationships. The resident, who is a practicing physician, is perhaps more inclined to retrieve a clinical exemplar, to explain the role of right atrial pressure. These are two instances where one's understanding of basic concepts are shaped by their functional utility.

The following excerpt from a first year student (1.1) illustrates the difficulty they have in developing an integrated model of the system.

Excerpt from a first year medical student (1.1)

1. Right atrial pressure,
2. The right atrium is where the venous return goes into.
3. If you have decreased right atrial pressure,
4. you're going to increase the venous return
5. because it's going to want to go into there.
6. If you can increase the amount of blood going into the heart,
7. the that will increase the cardiac output.
8. So um,
9. basically the pressure of the right atrium um
10. acts as a kind of,
11. I guess beginning of a cycle,
12. or circle or a link between cardiac output and venous return.
13. If something happens to the right atrial pressure,
14. um, if it increases,
15. then that will result in venous return increasing,
16. therefore cardiac output increasing.
17. If it decreases,
18. then that will result in both decreasing.
19. I guess that's the main part of the feedback system.

This response indicates that many of the correct knowledge elements are in place. Specifically, the subject knows that the right atrium is a pressure gradient for venous return and cardiac output, that it is at the end of a segment of one circuit and at the beginning of another. She also suggested the notion of a feedback system, in the sense that changes in right atrial pressure affects changes in blood flow, but also reacts to these changes. However, there is an evident contradiction in this model. The subject first stated that a decrease in right atrial pressure will increase venous return (segments 3 and 4) and at a later point (segments 13 to 16) suggested that an increase in right atrial pressure will increase venous return and cardiac

output. She attempted to represent a causal model in terms of "a cycle", in which venous return results in an increase in cardiac output, and errs when she suggested that this in turn leads to a further increase in venous return. It is possible that the subject has several correct but irreconcilable pieces of knowledge. The idea that right atrial pressure is the back pressure for venous return and a decrease in this pressure increases blood returning to the heart; and the notion that right atrial pressure is a measure of preload, and an increase in this pressure increases stroke volume and cardiac output. Her mental model of the system has not been fully consolidated into an integrated model, even though many of the elements are in place and this led her into this pattern of circular reasoning.

Restoring the Equilibrium

The circulatory system is a closed system and except for a few transient states, cardiac output has to equal venous return. There are a host of mechanisms that restore and maintain this equilibrium. The subjects in the study were asked the following question:

> INT7 Explain why cardiac output and venous return never remain significantly out of equilibrium for more than a few seconds. Discuss the mechanical factors that restore this equilibrium when a) cardiac output temporarily exceeds venous return; and b) venous return temporarily exceeds cardiac output.[32]

The question presents two scenarios in which there is a temporary disequilibrium between venous return and cardiac output. In the first case,

[32]The focus of this study is on mechanical factors. However, it is difficult to restrict answers for this question to mechanical factors because baroreceptors (sensory nerves) are primarily responsible for sensing changes in blood pressure and flow throughout the circulatory system, and play a role in restoring the equilibrium.

cardiac output exceeds venous return, and in the second case, venous return exceeds cardiac output. In either case, there is going to be a shift of blood volume and the resulting increases in pressure in vessels and chambers of the heart. In the first situation, either venous return could increase as a result of a change in pressures and/or venous compliance, or cardiac output could decrease. In the second situation, there would most likely be an increase in preload, although, there could also be a change in venous capacitance.

Subjects responses were coded for content. The coding for the situation in which cardiac output temporarily exceeds venous return is presented in table 19.

The most common response was a general statement concerning a change in pressure leading to an increase in venous return. Six subjects, including the premedical student, each of the first year students, and a third (3.1) and a fourth year student (4.2) suggested this as the mechanism that would restore the equilibrium. Four subjects, including both second year students and a fourth year student (4.1), focused on a decrease in preload as the primary means in which cardiac output would be reduced to match venous return. Four subjects, including a third (3.2) and a fourth year (4.2) student, a resident (R2), and the academic cardiologist, also suggested that there would be an increase in afterload due to changes in arterial resistance. The last two responses places the locus of change on the cardiac function side. Seven subjects, including both third year students, a fourth year student (4.2), both residents, the physiologist, and the academic cardiologist, referred to changes in venous vessels resulting in a change in compliance and thereby increasing venous return. A resident (R1) and the physiologist also discussed the recruitment of stressed volume from the reserve of unstressed volume.

Table 19

Subjects' Responses to Cardiac Output Exceeding Venous Return (INT7).

Responses	P	1.1	1.2	1.3	2.1	2.2	3.1	3.2	4.1	4.2	R1	R2	Ph	CP	AC	Totals
A	1	1	1	1			1			1						6
B			1		1	1			1							4
C															1	1
D								1		1		1		1		4
E								1		1	1	1				4
F											1		1			2
G							1	1			1				1	4
H								1					1			2
I												1			1	2
J														1		1
K															1	1
Totals	1	1	2	1	1	1	2	4	1	3	3	3	2	2	4	31

A. Accumulation of blood in the venous system cause shifts in volume and pressure increasing venous return

B. A decrease in preload and/or stroke volume decreases cardiac output.

C. Blood shifts from the pulmonary circulation and heart into the systemic circulation.

D. Afterload or arterial vasoconstriction increases diminishing cardiac output.

E. Venous constriction would increase venous return.

F. Unstressed volume is recruited to increase venous return.

G. Changes in elastic/compliance/capacitance properties of the vessels.

H. Redistribution of blood from capillaries to venous system.

I. Baroreceptors would sense a pressure difference.

J. Increase in mean venous pressure.

K. Decrease in heart rate due to changes in sympathetic activity.

The coding of the responses for the situation in which venous return temporarily exceeds cardiac output is presented in Table 20. Nine subjects, including two first year students (1.1 and 1.2), both second year and both fourth year students, a third year student, the physiologist, and the academic

cardiologist stated that there would be an increase in preload to increase cardiac output. Four subjects, including the premedical student, a third (3.2) and a fourth (4.2) year student, and a resident (R1) suggested that there would be an increase in contractility. A resident and the cardiologist also considered the effects of changes in venous capacitance to reduce venous return in order to match the level of output.

Table 20

Subjects' Responses to Venous Return Exceeding Cardiac Output (INT7).

Responses	P	1.1	1.2	1.3	2.1	2.2	3.1	3.2	4.1	4.2	R1	R2	Ph	CP	AC	Totals
A			1							1						2
B		1	1		1	1	1		1	1			1		1	9
C	1							1		1	1					4
D	1										1					2
E				1												1
F							1		1				1			3
G											1				1	2
H												1		1		2
I															1	1
Totals	2	1	2	1	1	1	2	1	2	3	3	1	2	1	3	26

A. Increased right atrial filling.

B. As the filling of the heart is increased, then the preload and/or stroke volume are increased.

C. Increase in contractility.

D. Increase in heart rate.

E. Reduced pressures in the venous system.

F. Frank-Starling mechanism

G. Shift of venous blood from stressed to unstressed and/or increase in venous capacitance.

H. Decrease in afterload would increase cardiac output.

I. Decrease in mean systemic pressure decreases venous return

A third year student (3.1) initially described the factors that would restore the equilibrium in terms of a rather nonspecific compensatory mechanism. She was then asked to focus on mechanical factors and provided the following rather colourful analogy:

Excerpt from a third year student (3.1)

43. Okay,
44. well the mechanical factors,
45. if you think of this vascular circuit out side the heart
46. as a rubber hose the
47. if you inject a bolus of water into a hose
48. then the elastic properties of the hose will cause that bolus to travel
49. to travel down the hose,
50. I guess.
51. it won't stay in one end of the hose.
52. It will fill the whole hose
53. and that will increase your return.
54. If you suddenly decrease your beat to beat output into the hose,
55. then you will have blood piling up at the other end of the hose.
56. I don't know how that would happen if that was a hose.

E. Lets move from the hose to the heart.

57. Okay,
58. so if the heart pumps less blood on one beat into the vascular system
59. then that means
60. you've got some extra blood in the heart somewhere.
61. It's got to be still in the heart
62. and the heart
63. by the Frank Starling mechanism
64. is going to deal with that extra load
65. and pump it out on the next beat to compensate.

She developed this analogy of a water hose with elastic properties that distends and develops pressure which propels water into the other end of the hose. A drop in water pressure will cause blood (water?) to pile up at one end of the hose. When asked by the experimenter to focus on the heart, the subject was able to map the objects from the source domain (the hose) to the

problem. She proceeded to explain how the Frank-Starling mechanism can deal with the extra volume of blood. This response suggests a process of self-explanation via analogy. This is a situation in which the subject was not lacking in the prerequisite knowledge or understanding, but rather needed a means to express or access an appropriate explanation to account for the problem. The analogy provided the vehicle for this explanation.

Summary

This section addressed subjects responses to four issues related to questions where the integration of venous return and cardiac function were considered. The first issue concerned the effects of cardiac function on venous return, and the second issue was related to how changes in venous return factors affected cardiac output. The third matter discussed in this section pertained to the physiological limits of cardiac function. The last issue examined subjects' understanding of the role of right atrial pressure as a coupling mechanism for cardiac output and venous return.

There was a trend towards an accuracy of response with increasing expertise. This was evident in the accuracy of subjects' predictions pertaining to the integration section.[33] The more advanced group had predicted 80% of the correct responses (SD = 20.92), even though both residents did not respond very well in this part of the study. The medical students correctly predicted 69.44% of the correct responses (SD = 15.02). The 3 most expert subjects predicted the highest percentage of correct responses.

Most subjects understood that changes in cardiac function did not have an immediate impact on the various determinants of venous return, such as

[33]This section included several questions that were not considered integration questions and were not coded for predictive accuracy in this category.

venous resistance. Only four subjects responded that mean systemic pressure is not immediately affected by changes in cardiac output. This is not surprising, since it was already established that this concept was not very well understood (see Table 11). Several subjects erroneously assumed that pressure and resistance was transmitted from the arteries across the capillaries into the venous system. Almost all of the subjects, (13 out of 15) recognized that an immediate cessation in cardiac output would not cause an immediate stop to blood flow in the venous system.

The majority of subjects clearly understood the effects of an increase in venous return on cardiac output. However, most subjects did not appreciate the fact that there are significant limitations to the heart's ability to pump increasing supplies of blood. Two subjects, a fourth year student (4.2) and a resident (R2), suggested answers that were indicative of a conceptual error related to the upper boundary of the length-tension and pressure-volume relationship. The misconception, first documented by Feltovich and colleagues (1989), reflects a belief that the heart can go into failure when volume and pressure loads exceed an acceptable limit.

In general, subjects did not appreciate the multi-faceted function of right atrial pressure. Every subject, except for the premedical student, understood that right atrial pressure is a pressure gradient for venous return and many subjects also asserted that it is a measure of preload. However, no subjects indicated that cardiac function affects the gradient for venous return by altering right atrial pressure. Subjects are routinely taught about venous return-cardiac output integration with cardiac function and venous return curves. Only two subjects, the physiologist and the academic cardiologist alluded to the intersection between these curves.

Responses to the questions about restoring the equilibrium between venous return and cardiac output indicated that most subjects considered only a narrow range of factors. Several subjects placed the locus of control entirely within the scope of cardiac function. The more advanced subjects tended to discuss changes in venous capacitance/compliance as a means for restoring the equilibrium in blood flow between input and output.

Mental Models

This chapter has examined subjects' understanding of concepts related to pressure-volume and pressure-flow relationships in the circulatory system. In particular, we have investigated subjects' understanding of the determinants of cardiac output, venous return, and their integration. The emphasis has been on the explanation and prediction of individual concepts. This section presents analyses that synthesize the results from the prior sections, and attempts to characterize subjects' mental models of the circulatory system. Specifically, we are interested in examining the effects of conceptual difficulties on subjects' understanding of the system as a whole. The section also summarizes the results of the first three parts of the study, open-ended questions, basic physiology, and integration questions.

Mental Models of Cardiac Output and its Regulation

A significant aspect of the biological system considered in the study can be characterized as sets of (partial) functional relationships or components of functional relationships that hold between the variables of interest. Functional dependency diagrams are a method for representing sets of causal relationships. It can be used to characterize the way subjects' models deviate from an ideal model (the reference model).

Figure 17 illustrates a functional dependency diagram generated from the protocol of the most novice subject, a premedical student. The correspondences between relations were generated from the subjects' predictions and explanations. The subject correctly predicted only 42% of the correct responses. This result is indicated in the diagram by the numerous incorrect correspondences, as well as, connections between concepts that were omitted. For example, there is no connection between afterload and aortic pressure.

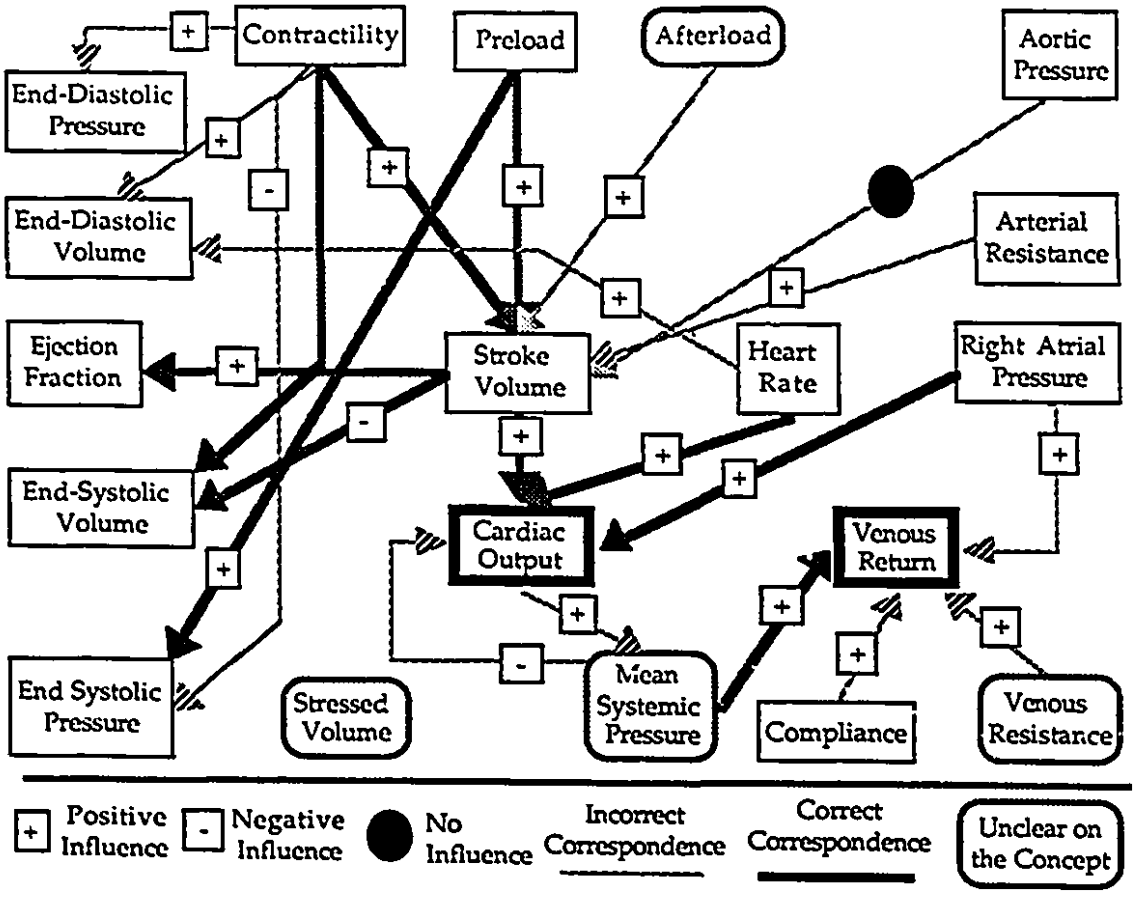


Figure 17: Functional Dependency Diagram of a Novice Premedical Student.

Several of the variables, particularly those related to venous return were not clearly understood. The effect of the misconception concerning the nature of pressure-volume relationships is evident in many of the incorrect connections between variables. Generally, any of the variables that are suggestive of an increase in tension, resistance, or pressure (e.g., contractility, afterload) are believed to propagate an increase in volume or in flow. Almost all relationships are viewed as being positively correlated. This line of reasoning invariably led to errors in prediction and explanation.

The analyses suggest a trend towards an increase in conceptual understanding with expertise. Most subjects were able to determine the effects of changes in quantities to variables. With the exception of the 3 first year medical students, subjects showed considerable inter-response consistency in their predictions and explanations. The majority of subjects exhibited particular conceptual errors in their mental models. A functional dependency network of a second year student (2.1) is illustrated below (Figure 18).

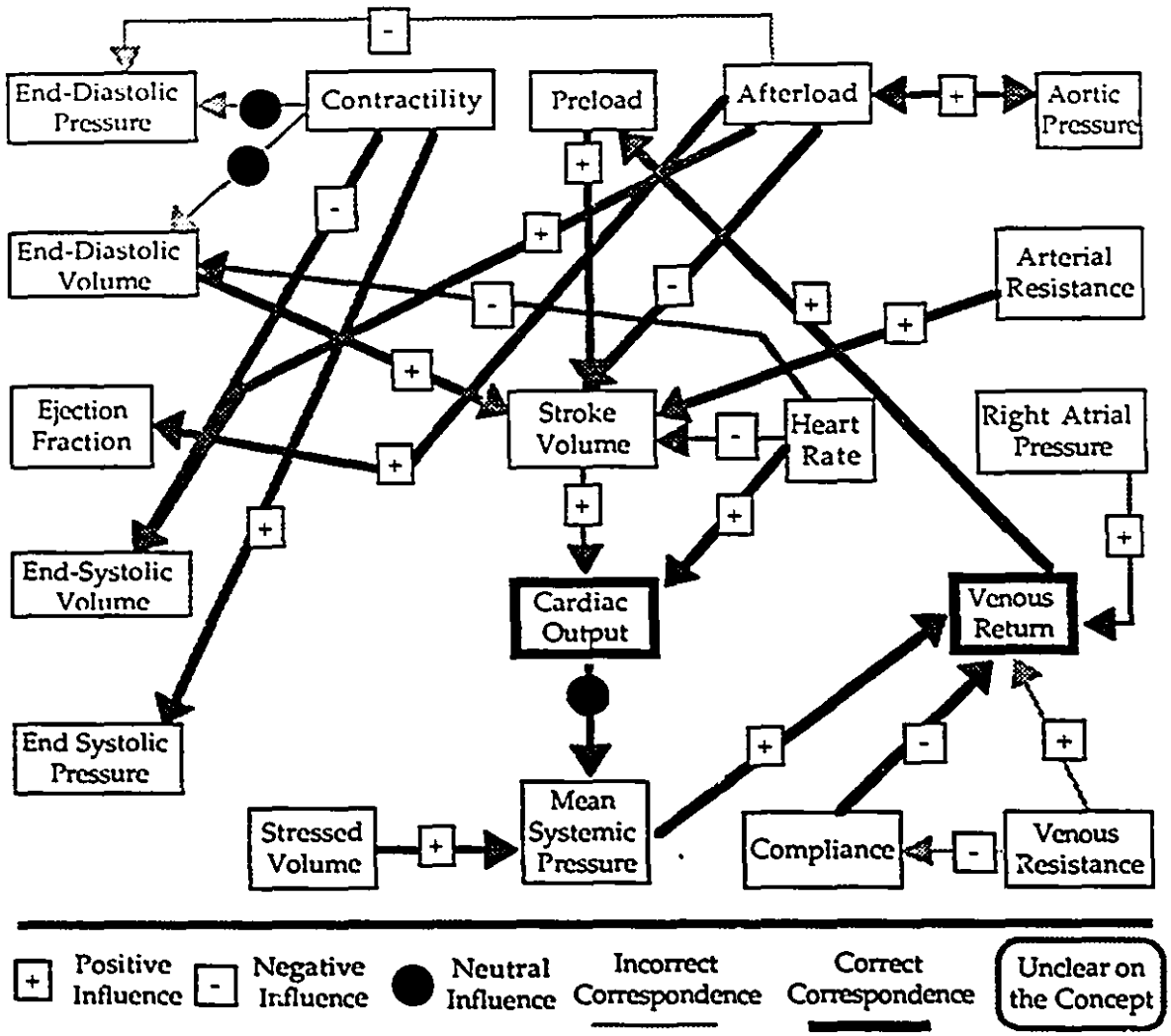


Figure 18: Functional Dependency Diagram of a Second Year Medical Student (2.1).

This student was able to articulate an understanding of each concept. and predicted 70% of all responses, including 71% of the cardiac output predictions and 64% of the venous return predictions. There are three errors evident in the subjects' model.[34] Two are relatively minor errors, including

[34]There are other sources of error that are not represented in the functional dependency diagram. An example is reflected in the failure to recognize the circumstances when an enabling condition is not present.

one relating *end-diastolic pressure* to *afterload*, and another relating contractility to end-diastolic pressure and end-diastolic volume. The third error was a function of a misconception concerning resistance and compliance, discussed in considerable detail in a previous section. This misconception can largely account for the relatively low rate of correct venous return predictions. Many of the other relations expressed in the model are correct and the model is internally consistent. This consistency serves to propagate errors that stem from this misconception.

What is not immediately apparent from the diagram is the lack of differentiation in the integration aspect of the subject's mental model. He predicted 81% of the correct basic physiology outcomes, but only 50% of the integration outcomes. This lack of differentiation is also apparent in the subjects' explanations of the role of right atrial pressure and to the factors that restore an equilibrium between venous return and cardiac output.

The functional dependency network of a fourth year medical student (4.1) is presented in Figure 19. This student correctly predicted 53% of the correct outcomes. Only the premedical student predicted fewer correct outcomes. However, it was evident from the subject's explanations that he understood most of the concepts and could apply them in more complex situations.

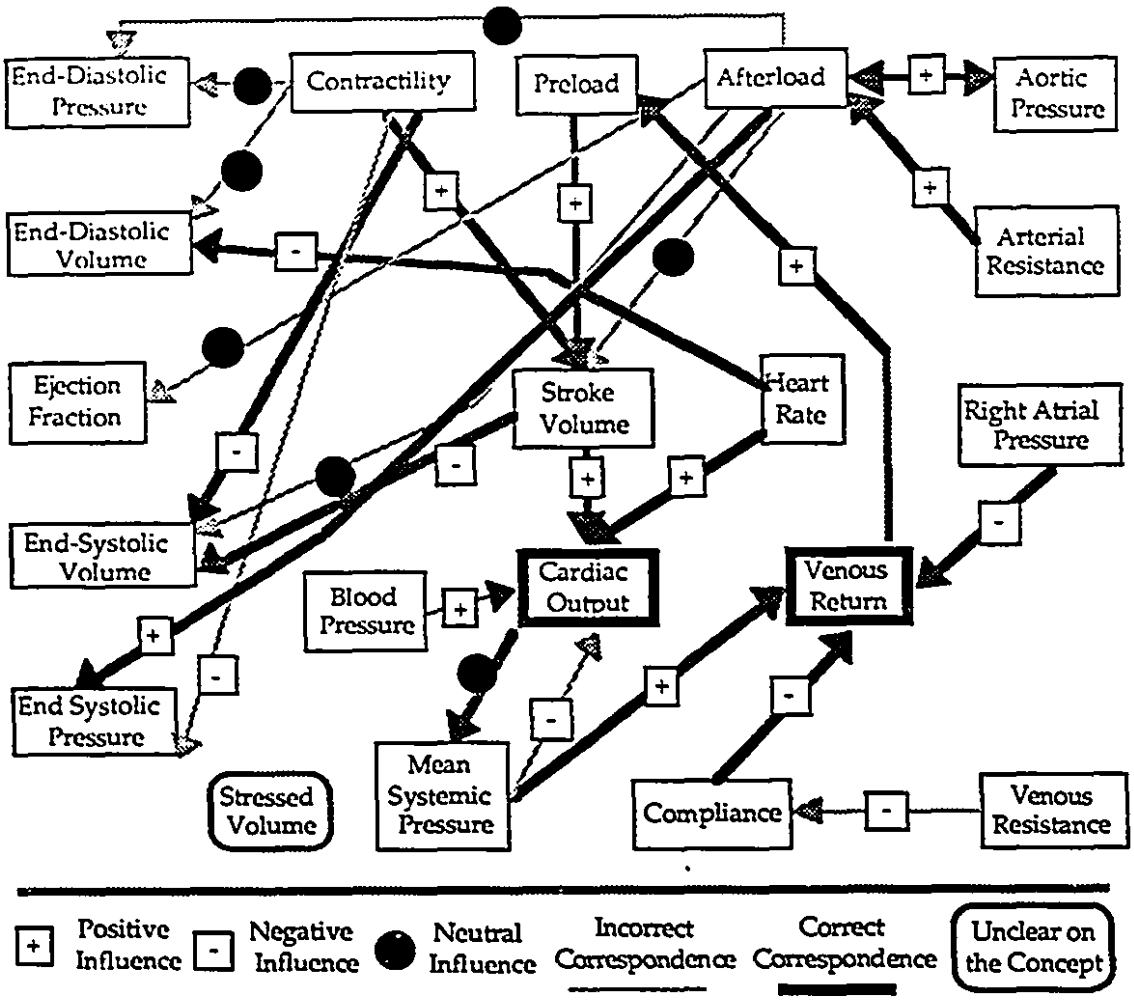


Figure 19: **Functional Dependency Network of a Fourth Year Medical Student (4.1).**

There are a number of sources of errors in this students' model, including a lack of differentiation in integration and in determining the effects of contractility on pressures and volume in the system. One error, in particular, is the source of many of the subject's conceptual difficulties. It is related to the effects of afterload, which is one of the critical determinants of cardiac output. An excerpt from the influence network makes the relationship explicit (Figure 20).

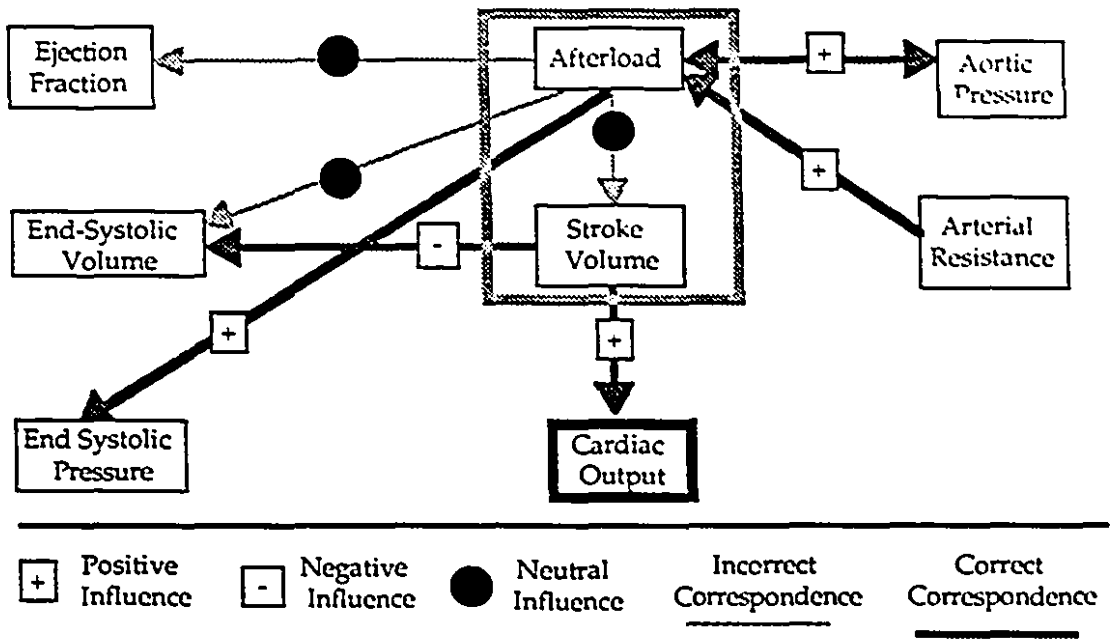


Figure 20: Excerpt from a Functional Dependency Network of a Fourth Year Medical Student (4.1), Indicating the Error Relating Afterload to Stroke Volume.

The problematic inference was that afterload has no effect on stroke volume. Afterload, in fact, decreases stroke volume. The fact that the subject's model is largely coherent, and that he correctly represented the relationship between stroke volume and all other variables, serves to propagate errors throughout the system, when a question involved either *afterload*, *aortic pressure* or *arterial resistance* as causal agents. As discussed previously, the subject relied extensively on the pressure-volume loop diagram to calibrate his judgments of the effects of the determinants of cardiac output. However, he repeatedly failed to retrieve correct correspondences between the graph features and the functional relationship. The error can be best characterized as an error in analysis, rather than as a misconception.

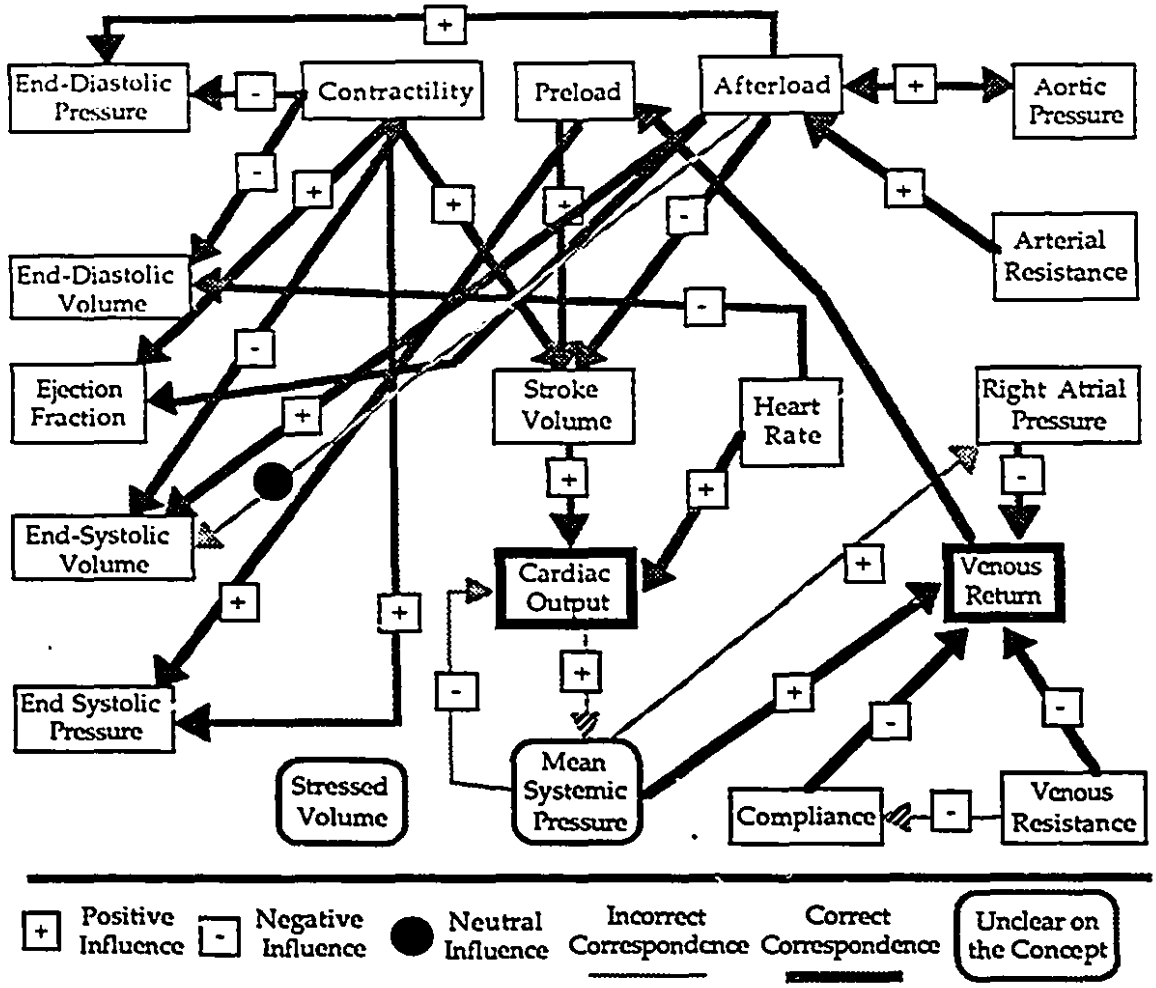


Figure 21: Functional Dependency Network of a Resident Physician (R2).

Figure 21 illustrates the functional dependency network of a resident physician. He predicted 81% of the correct outcomes. The diagram indicates that most of the proposed causal influences are correct in relationship to cardiac output. Eighty-six percent of the cardiac output predictions were correct. The predictions were supported by elaborate and precise explanations that suggested a thorough understanding of cardiac function. However, as represented in the lower right hand corner of the diagram, the venous return concepts were not well understood, and many of the inferences

concerning these relationships were incorrect. The subject predicted 71% of the venous return outcomes. In particular, *mean systemic pressure*, a pressure gradient of venous return, was interpreted as *mean arterial pressure*. This resulted in a number of errors in prediction and explanation. The subject's model was also deficient regarding the integration of venous return and cardiac output.

Component Models

It is possible to partition the conceptual model into various components. This section focuses on subjects' understanding of the components of the conceptual model. Figure 22 presents a reference diagram of the determinants of cardiac output and venous return.

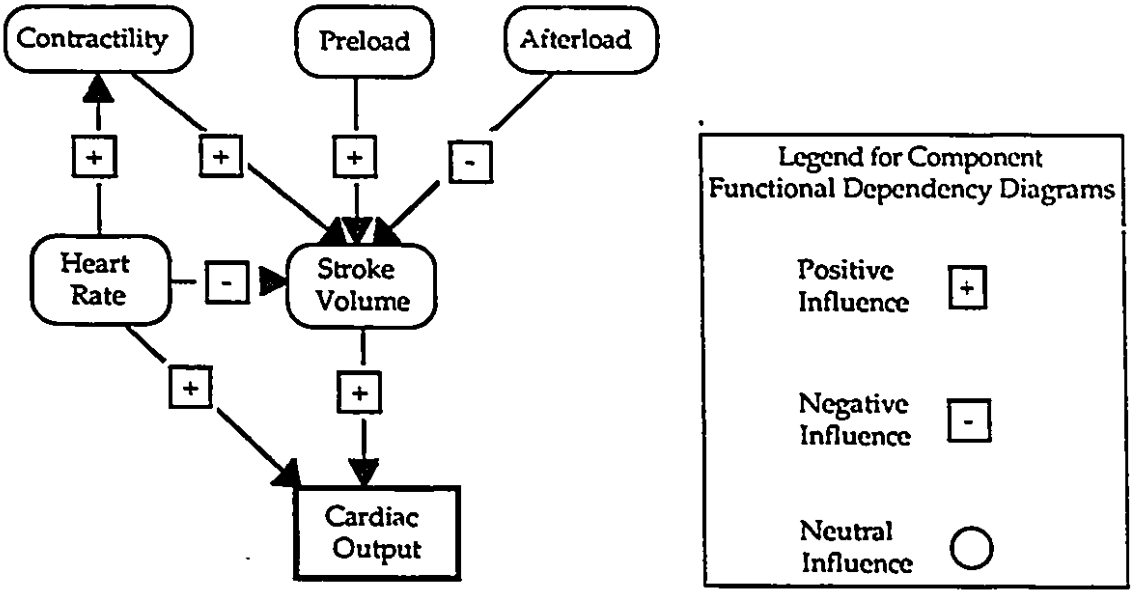


Figure 22. Basic Determinants of Cardiac Output.

The primary determinants of cardiac output were well understood by most subjects. The correct relationship between heart rate and contractility was recognized by subjects from third year medical school to experts.

However, subjects experienced some difficulty in reasoning about the effects of afterload and contractility on changes in pressure and volume in the ventricle. This was most apparent in subjects' predictions and explanations of the effects of increases in contractility on end-diastolic volume and on end-diastolic pressure. It is possible that these problems represent difficulties in analysis, rather than conceptual errors.[35]

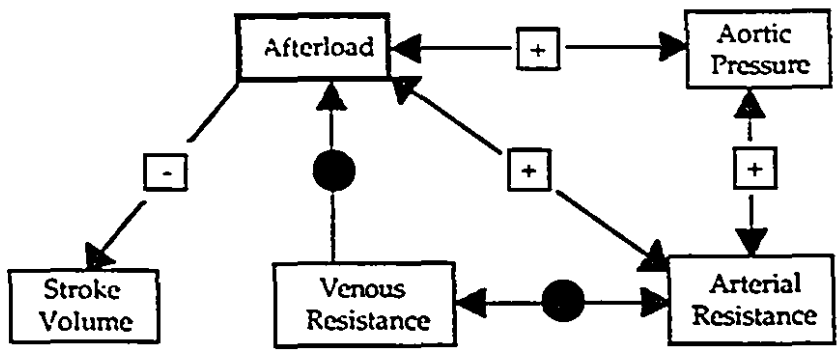


Figure 23. Variables Affecting Afterload.

A diagram of the variables that affect and are affected by afterload are presented in a dependency diagram in Figure 23. Subjects' responded with considerable accuracy to questions pertaining to afterload. As discussed previously, a notable exception was a fourth year student (4.1) who erred in his analysis of the relationship between afterload and stroke volume. The premedical student and first year medical students did not have a sound grasp or clear understanding of afterload as judged by their explanations. Despite this lack of understanding, they were able to correctly predict most of

[35]It is sometimes difficult to delineate a conceptual error from an error in analysis. A conceptual error is one that emanates from a subjects' knowledge-base (e.g., venous resistance and compliance are interdependent). An error in analysis reflects mistakes that can arise from flawed procedures (e.g., as in interpreting a graph) or an error in representing a problem (omitting a variable).

the effects of afterload. A fourth year student incorrectly suggested that an increase in venous resistance would increase afterload.

Two clinicians, a resident (R2) and the cardiologist practitioner used clinical analogies to explain the effects of afterload. These analogies yielded incorrect solutions because changes in afterload result in various compensatory mechanisms coming into play. The map between clinical explanations and physiological explanations proved to be rather complex in the context of understanding afterload.

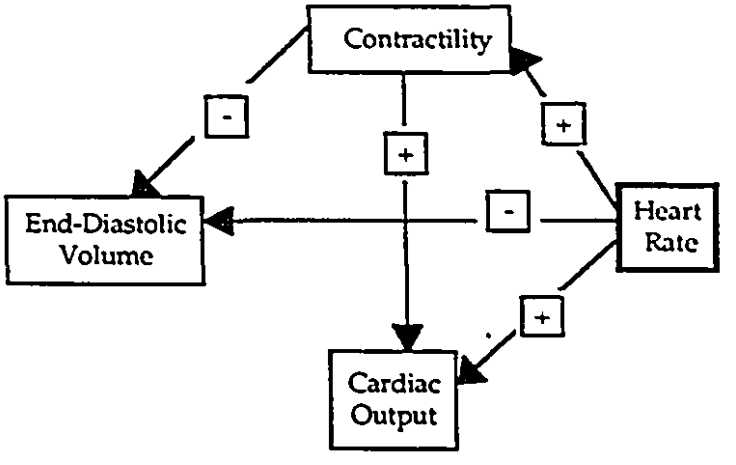


Figure 24. Variables Affecting and Affected by Heart Rate.

A diagram of the variables that relate to heart rate is presented in Figure 24. Most of the questions related to heart rate presented relatively few problems for subjects to respond to correctly. In particular, they were cognizant of two of the primary effects that changes in heart rate can have on cardiac output. They understood that cardiac output is a function of stroke volume and heart rate and that increases in heart rate diminishes stroke volume by compromising diastolic filling. Only the more advanced subjects appreciated the effect of heart rate on contractility.

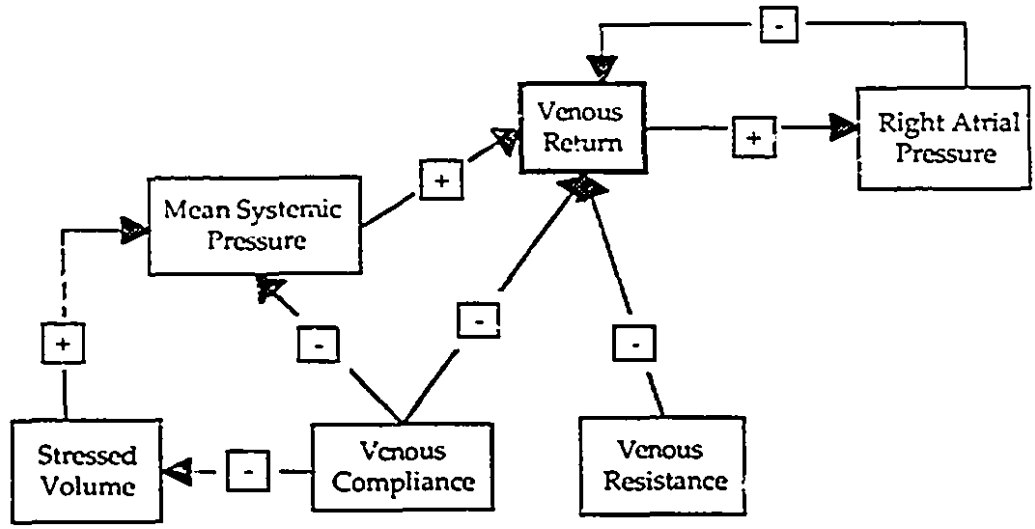


Figure 25. Basic Determinants of Venous Return.

A diagram of the basic determinants of venous return is presented in Figure 25. The concepts pertaining to venous return were not as well understood by most subjects. In particular, the concepts of mean systemic pressure and stressed volume were not known to most participants in the study. Only six subjects including, a second year student (2.1), both fourth year students, a resident (R1), the physiologist, and the academic cardiologist, could identify the defining properties of these concepts. The effects of compliance and right atrial pressure as a back pressure for venous return were recognized by all subjects. The effects of venous resistance were recognized by most subjects. However, several subjects exhibited a particular misconception that can be examined more closely in Figures 26a and 26b.

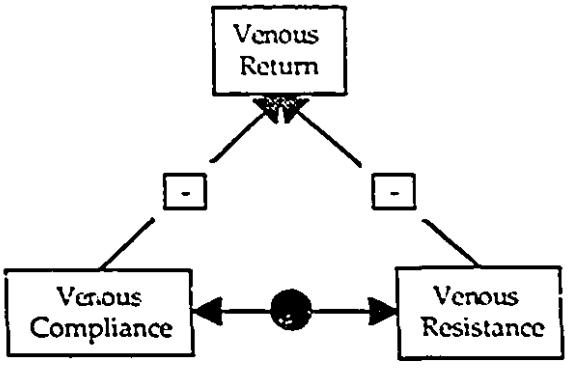


Figure 26a Correct Model of Compliance and Resistance.

In the venous system, compliance and resistance are independent of each other. The venules are the primary compliance vessels and the larger veins, closer to the right atrium, are resistance vessels. There is a clear dissociation. Increases in either compliance or resistance tend to decrease venous return. The model suggested by certain subjects is illustrated in Figure 26b.

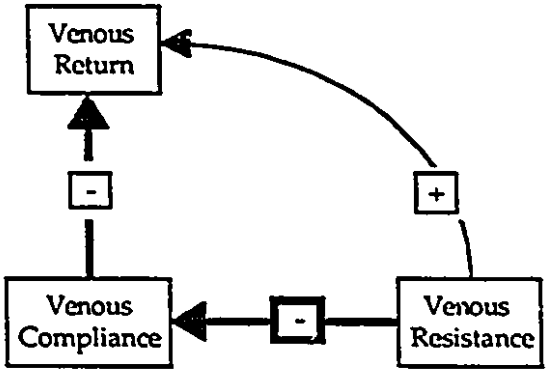


Figure 26b. Interdependence of Venous Compliance and Resistance.

The model illustrated in Figure 26b, suggests an interdependence between compliance and resistance. The effect of compliance on venous return is viewed as the priority relationship and venous resistance exerts an effect only

by acting on the compliance vessels. Six subjects exhibited this misconception, including a second year medical student (2.1), a third year student (3.1), both fourth year students, and both residents. In the second year student's mental model, compliance has clear priority in the venous system. In the models of the five other subjects, compliance and resistance were seen as opposing forces, and the independent effects of venous resistance were viewed as being difficult to extricate from its effects on compliance. It should be pointed out that each of these subjects clearly understood the meaning of resistance. The misconception was unique to venous resistance.

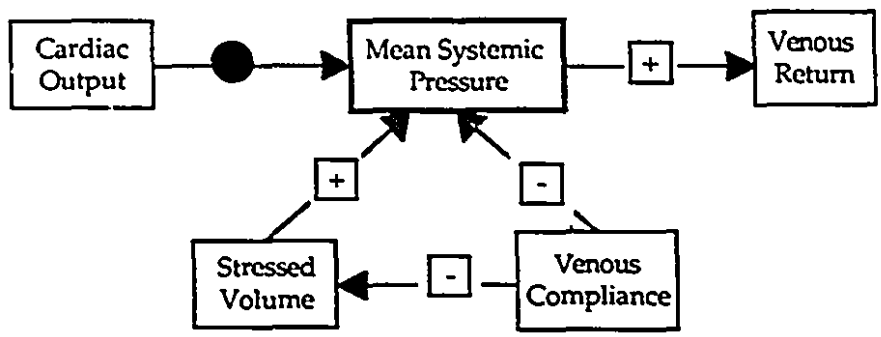


Figure 27. Variables Affecting and Affected by Mean Systemic Pressure.

A functional dependency network of the variables that affect and are affected by mean systemic pressure is presented in Figure 27. As discussed previously, mean systemic pressure and stressed volume were concepts that were not very familiar to most subjects. Therefore, it is not surprising that few subjects appreciated the causal relationships involving these concepts. Only a second year student, the physiologist, and the academic cardiologist made reference to the relationship between mean systemic pressure, stressed volume, and compliance. Four subjects recognized that changes in cardiac output have no immediate impact on mean systemic pressure. A part of this

problem is due to terminological confusion. Terms, such as, mean systemic arterial pressure are more commonly used in clinical situations.

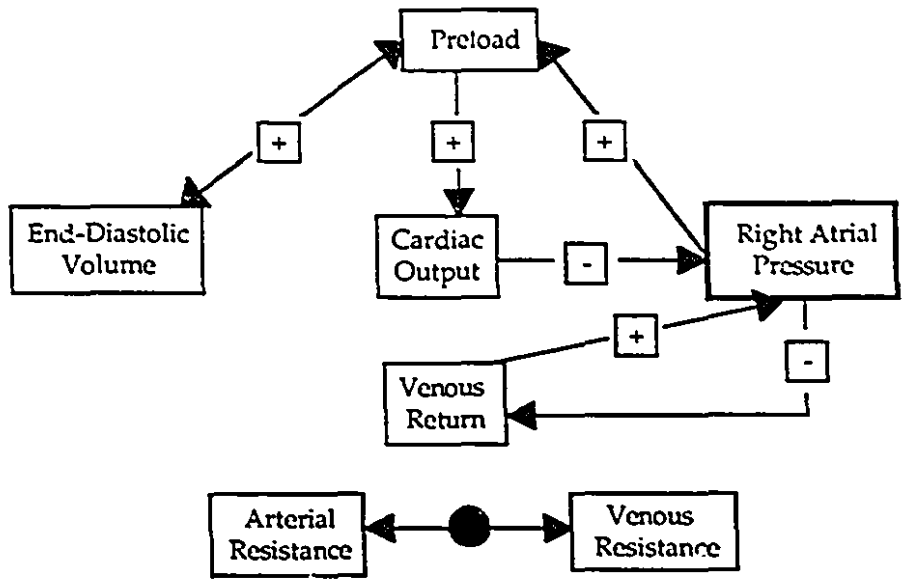


Figure 28. The Integration of Cardiac Output and Venous Return.

The variables that are related to the integration of cardiac output and venous return are illustrated in Figure 28. The factors integrating cardiac output and venous return were partially understood by most subjects. Specifically, subjects recognized that changes in right atrial pressure affect venous return, which in turn affects the preload on the right ventricle and cardiac output. However, the multidimensional role of right atrial pressure was not appreciated by subjects. In particular, no subject stated that changes in cardiac function directly affect right atrial pressure and the pressure gradient for venous return.

There is a dissociation between arterial and venous resistance in that changes in either resistance does not impact on the other. Several subjects, notably, a third year student (3.1), fourth year student (4.2), and a resident (R1) incorrectly suggested that there is an interaction between venous

resistance and arterial resistance. Although, this was not a focal issue in the study, subjects appeared to have difficulty in reasoning about the effects of differential pressures in specific segments (e.g., arteries, arterioles, and venules, etc.) of the circulatory system.

The physiological boundary of the heart's ability to pump blood is also a delimiting factor in the integration of cardiac output and venous return. Subjects' ascribed to one of three models. The first model suggested that there is no apparent limitation on a healthy heart's ability to pump blood. This model was consistent with the responses of five subjects, including the premedical student, three first year students, and a resident (R1). The second and correct model, indicated that there is a plateau to the cardiac function curve, at which point further increases in venous return can be matched with further increases in output. This line of reasoning was evident in the responses of a second year student (2.2), a third year student (3,1), a fourth year student (4,1), the physiologist, and both cardiologists. The third model suggested that given very large preloads, the heart is likely to go into failure. This model is in keeping with the responses of four subjects, including a second year student (2.1), a fourth year student, and a resident (R2). This pattern of reasoning was most prominent in the responses to various questions by the fourth year student and the resident to various questions.

In summary, the results support a noticeable progression in mental models with increasing expertise, as judged by subjects' predictions and explanations. In addition, there were many individual differences between subjects at any given level. Conceptual errors were present in the mental models' of subjects at all levels of expertise. These errors that were evident in the thinking of physicians were bolstered by rather elaborate justifications,

including the use of clinical exemplars. The clinical exemplars were used as analogies to explain physiological situations. Frequently, the exemplars accessed were not appropriate analogies for the physiological situation under consideration.

Based on the analyses of the accuracy of prediction and explanations for the basic physiology and the integration questions, the physiologist and the academic cardiologist had the most complete and robust mental models' of the circulatory system. The first year medical students and the premedical student did not have much of the prerequisite knowledge to respond to some of the questions, since they had not taken a course in cardiovascular physiology. It was interesting to observe the kinds of prior knowledge and strategies they brought to bear on the problems. It seems likely that a certain minimum of physical science knowledge is a prerequisite for understanding in this physiology domain. The lack of such knowledge was an apparent problem for the premedical student. However, physical science knowledge is not sufficient if the students do not know how to select and make the appropriate correspondences. This was a source of difficulty for one of the students, who had drawn liberally on his understanding of *kinematics* to respond to a question that required a knowledge of *statics* and *dynamics*. One of the first year students (1.1) had a greater knowledge of physiology and was able to construct coherent and accurate explanations beyond what would have been expected from a first year student.

The responses of the second through fourth year students' showed considerable variation. The second year students had completed the cardiovascular physiology course less than a year earlier and could recall specific causal relationships, but often could not elaborate in their explanations. One of the third year students (3.1) responded with

considerable accuracy and provided detailed explanations for the cardiac output questions, but was not as accurate in her predictions and explanations for the venous return questions. A fourth year student (4.2) predicted every venous return response correctly. However, his explanations suggested particular conceptual errors. Teleological reasoning was quite prominent in the responses of the more novice subjects. The first year student responses, probably reflected the lack of a process vocabulary (e.g., lacking terms such as, contractile state). In the more advanced subjects, teleological reasoning represented a means for reducing complexity.

The mental models developed by subjects were generally internally consistent. Conceptual errors, and to a lesser extent procedural errors (e.g., errors in analysis), tended to produce patterns of misunderstanding. In a complex domain such as this one, concepts are embedded in networks of interacting concepts. As Feltovich and colleagues (1989) suggest, elements of knowledge, in themselves partly correct or wrong in specific aspects, interact with each other to create large-scale robust misconceptions. As the mental models develop and become increasingly robust knowledge structures, these misconceptions become entrenched in subjects' thinking and are used to explain a wide range of phenomena.

Applied Problems

In the previous sections, we considered results pertaining to the understanding of basic cardiovascular physiology concepts. In this section, the focus is on subjects' responses to problems that require the application of these concepts in different contexts. Specifically, we examine the responses of subjects to four types of problems, applied physiology problems, problems

describing pathophysiological disturbances in structure or function, medical disorders with hemodynamic consequences, and brief clinical problems.

Applied Physiology Problems

The first problem (Ap2) asks about the factors that contribute to an increase in cardiac output during extreme exercise. Exercise activity results in a complex series of neuromuscular, respiratory, circulatory, and metabolic events (Guyton, Jones, & Coleman, 1973). Medical and premedical (health science) students are taught exercise physiology in different courses and they all have some understanding of the effects of exercise. The focus here is on the mechanical factors in the circulatory system that come into play. All cardiac factors and all venous return factors (e.g., resistance) contribute to a highly integrated cardiovascular response to exercise.

Ap2 Extreme exercise can increase the cardiac output from the normal resting value of 5 L/min. to well over 25 L/min. Discuss the factors that contribute to this increase.

During exercise, there is an increase in *contractility* and an increase in *heart rate*. The heart rate can triple during extreme exercise. This results in an upward shift of the cardiac function curve, so that you get a higher cardiac output for any given right atrial pressure. This curve then intersects the venous return curve at a higher point than it would under normal conditions. A significant increase in cardiac output relies on active changes (caused by neurohumoral changes) on the part of the systemic vessels in the form of a decrease in *venous resistance*. There is *redistribution of blood flow* from the splanchnic circuit (vessels supplying the spleen) to the faster circuits, which has a lower time constant for emptying blood. There is a decrease in *vascular*

capacitance and an increase in *stressed volume* that allows for a higher flow for any given right atrial pressure. The increased metabolic rate, the decreased peripheral resistance in the exercising muscles, and the venoconstriction all contribute to an increased atrial filling and a larger *preload*. In addition, the tensing of exercising muscles, including the abdominal muscles can quadruple the *mean systemic pressure*, thereby increasing the *pressure gradient* for venous return. The responses to the problem relating extreme exercise to cardiac output is presented in Table 21.

All subjects were able to respond to this problem with, at least, a partially correct answer. In general, the subjects' responses improved with expertise in the breath of concepts applied and in the coherence of the explanations. Every subject except for a first year medical student suggested that heart rate would increase. Ten subjects suggested that there would be an increase in stroke volume and nine subjects suggested that venous return would have to increase.

Table 21

Subjects' Responses to Factors That Increase Cardiac Output During Exercise (Ap2).

Factors	P	1.1	1.2	1.3	2.1	2.2	3.1	3.2	4.1	4.2	R1	R2	Ph	CP	AC	Totals
Increased Stroke Volume			1			1	1	1	1	1	1	1	1	1		10
Increased Heart Rate	1	1	1		1	1	1	1	1	1	1	1	1	1	1	14
Preload						1							1	1		3
Increased Contractility				1		1		1	1	1	1	1	1			8
Decreased Afterload					1	1				1		1		1	1	6
Increased Venous Return	1	1	1	1						1	1		1	1	1	9
Increased Mean Systemic Pressure											1		1			2
Increased Stressed Volume										1	1				1	3
Decrease Right atrial pressure											1					1
Decreased vascular resistance						1			1	1		1	1		1	6
Venous Compliance		1							1	1	1		1		1	6
Neurohumoral Factors					1	1		1		1		1	1		1	7
Redistribution of Blood											1	1	1		1	4
Increased Metabolic/ Oxygen Demands	1	1	1	1												4
Totals	**3**	**4**	**4**	**3**	**3**	**7**	**2**	**4**	**5**	**9**	**9**	**7**	**10**	**5**	**8**	**83**

The premedical student and the first year medical students focused primarily on the *teleological* or the *demand* characteristics of the heart in their explanations of the effects of exercise. This is illustrated in the following three excerpts:

Excerpt from the premedical student

2. One factor for uh increasing cardiac output
3. and therefore for increasing the amount of blood
4. that's being ejected out.
5. it is because the muscles require more oxygen.
6. Cause when your exercising
7. the muscles are using up the oxygen
8. and therefore blood that's carrying oxygen,
9. must also increase
10. and as a result you are getting a greater output.

Excerpt from a first year medical student (1.1)

5. Well, first of all when you're doing a lot of exercise,
6. your blood automatically beats,
7. your heart automatically beats faster
8. because the metabolic needs of your body,
9. it needs more oxygen,
10. it needs more blood,
11. it needs that system to go faster
12. to take away lactic acid,
13. take away carbon dioxide,
14. bring in oxygen.

Excerpt from a first year medical student (1.3)

1. When you're exercising like that,
2. your metabolic rate is increasing.
3. So you need energy a lot faster
4. and that obviously is a key thing involved in cardiac output,
6. you need a lot of oxygen and so on
7. and that's why cardiac output would increase like that
8. to be able to deliver more oxygen per unit time,
9. which is what happens when the output increases.
10. So that's one thing that is happening,
11. the energy demand is very high.

The common thread in these responses is that cardiac output increases to fulfill a need. That is to say, there is an increase in output because of the metabolic needs of exercising muscle tissue. The responses are essentially correct. This response pattern was unique to the premedical student and first year medical students. This result is in keeping with the previous

findings that suggest that teleological reasoning is a common mode of reasoning used by novices in thinking about biological systems.

The second and third year students predominantly focused on the cardiac output determinants. The fourth year students, the physicians and the physiologist consider a broader range of cardiac and venous factors. In particular, four subjects, a fourth year student (4.2), a resident (R1), the physiologist, and the academic cardiologist were able to correctly apply a broad range of the pertinent factors that affect cardiac output under conditions of extreme exercise.

Perhaps the most complete analysis of this problem was provided by a fourth year student (4.2). The strategy he used to approach the problem is most interesting. Here is an excerpt from his response:

Excerpt from a fourth year medical student (4.2)

1. If cardiac output is equal to stroke volume times the heart rate,
2. you could look at the factors that contribute to both.
3. If you look at the heart rate,
4. exercise causes an increase in sympathetic discharge.
5. So you get an increase in heart rate,
6. in terms of the stroke volume,
7. stroke volume will be affected by increased venous return.

In the first excerpt, he began by reiterating the fact that cardiac output is equal to stroke volume times the heart rate, and proceeded to explain that heart rate would increase. He then suggested that an increase in venous return would promote an increase in stroke volume. In the next excerpt, he developed an explanation for why venous return would increase.

Excerpt from a fourth year medical student (4.2)

8. Your venous return will be increased
9. because for several reasons.
10. One, on exercising your actually squeezing the veins themselves,
11. and so you are increasing venous return in a very mechanical way
12. because of the valves in the veins on exercising
13. on using your leg muscles for instance
14. or your arm muscles
15. your causing blood to stop pooling
16. and to get back into active circulation,
17. going back into the heart.
18. so increasing venous return,
19. Increasing venous return will therefore
20. will cause an increase in end-diastolic filling pressure
21. which will increase contractility, um
22. and which will cause an increase in stroke volume.

The primary emphasis in this explanation is on the constriction of veins reducing venous capacitance (causing blood to stop pooling) and increasing stressed volume (active circulation). This increase venous return produces an increase in end-diastolic filling pressure. The student then suggested that this increase in filling pressure would produce an increase in contractility. This last statement is incorrect. Contractility would not be affected by filling pressures. Rather, an increase in end-diastolic filling pressure would increase the preload. In the last excerpt, he has come full circle and is once again focusing on the effects of exercise on cardiac function.

Excerpt from a fourth year medical student (4.2)

23. Contractility increases of its own accord
24. by the sympathetic discharge
25. that occurs during exercise itself,
26. and so by increasing contractility,
27. the slope of the contractility curve,
28. you therefore increase stroke volume on your pressure-volume loop,
29. For example the output of adrenaline in your system on exercising,
30. you can get a decrease in arterial
31. in your arterial resistance in your muscles,

32. um you get you get arterial dilatation,
33. which will have an effect of decreasing the afterload on the heart.
34. Which by doing that will increase the cardiac output as well.

In this excerpt, the explanation began with the argument that a sympathetic discharge would increase contractility and stroke volume. It would also produce a decrease in arterial resistance that would decrease afterload and lead to a further increase in cardiac output. Exercise produces a highly integrated physiological response and the problem provides a means for assessing the degree of coherence in subjects' models of the circulatory system. In this case, the subject began with a clear starting point and worked his way through the system, describing how a particular variable would react under these circumstances, how this reaction provokes another reaction and so forth. Despite the error concerning contractility, his response is indicative of a highly integrated mental model.

The responses of the two cardiologists differ considerably from one another. Figures 29 and 20 display the semantic network representations of the two cardiologists.

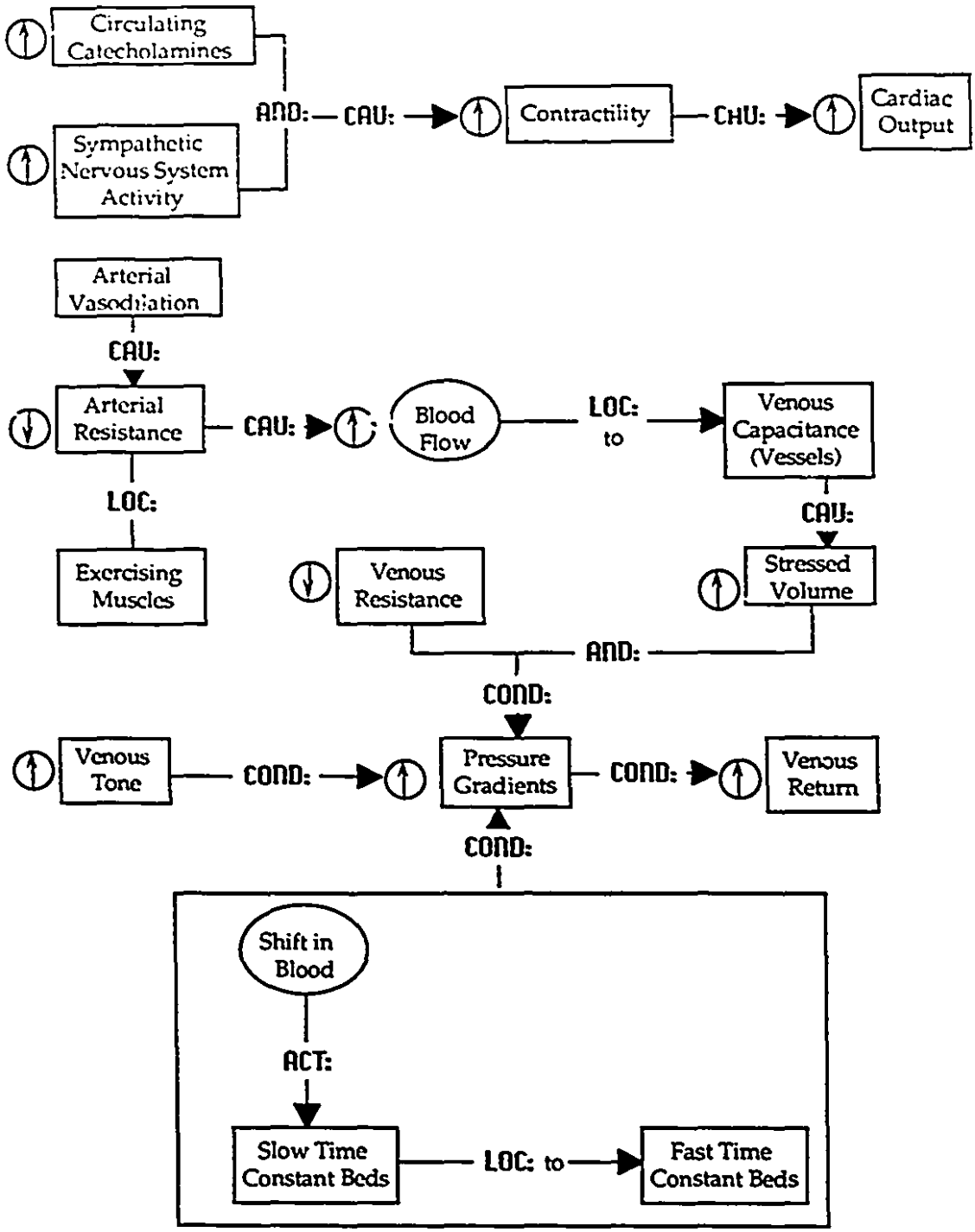


Figure 29: Semantic Network Representation of the Academic Cardiologist's Response to the Exercise Problem (AP2).

The academic cardiologist considered a number of factors. In terms of cardiac factors, he focused on how an increase in sympathetic activity causes an increase in contractility leading to an increased cardiac output. He then developed an explanation that includes four factors that would increase the pressure gradient for venous return: an increase in *stressed volume*; a decrease in *venous resistance*; an increase in *venous tone* (which decreases compliance); and, a *redistribution of blood* to a faster circuit, thereby increasing blood flow.

The cardiologist practitioner approached the problem from a very different perspective. He attempted to account for the fivefold increase in cardiac output by reasoning deductively. He began with the premise that cardiac output is equal to heart rate times stroke volume, and since that heart rate can triple, exercise must cause stroke volume to double. This is a reasonable heuristic, except for the fact that a dramatic increase in heart rate also decreases stroke volume. The practitioner then proceeded to explain the increase in stroke volume as being primarily a function of a decrease in afterload caused by an increase in systolic blood pressure, which in turn causes a decrease in diastolic pressure. He suggested that an increase in venous return would contribute to increasing cardiac output by increasing left ventricular diastolic filling, but did not discuss any of the determinants. The explanation is cardiocentric in that the venous system is viewed as playing only a secondary role. The locus of change is within the variables that affect cardiac function.

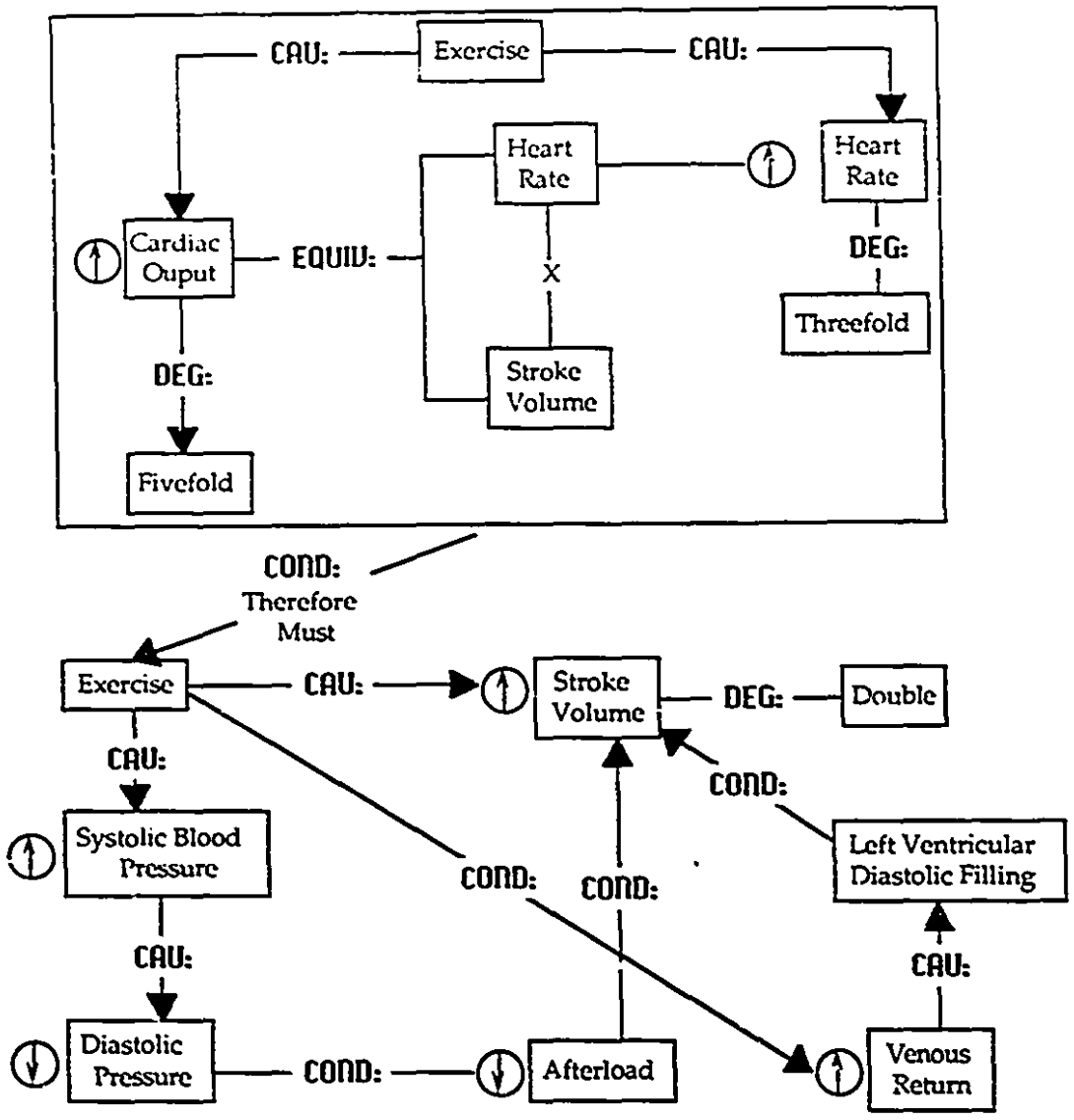


Figure 30: **Semantic Network Representation of the Cardiologist Practitioner's Response to the Exercise Problem (Ap2).**

Structure- Function Problems

Most topics in physiology involve explicit relationships between structure, function, and behavioural processes. The subject matter of cardiac output and the mechanics of the circulatory system tend to be more abstract, involving changes in quantities that produce changes in behaviour (e.g., pressure-flow). The structure-function relationships are not always as

explicit. Several of the primary factors, such as venous resistance and venous compliance, reflect attributes that are distributed across structures. In this section, we consider a set of problems that involve alterations in structure that have particular functional and behavioural consequences.

As described in chapter six, in order to respond to the applied problems there are a set of abstract procedures to follow: 1) Determine if the problem is predominantly affecting cardiac factors, venous return factors or both; 2) Identify the particular factor(s) (e.g., afterload) and their direction of change (increase, decrease); 3) If necessary, construct a mechanistic account (e.g., changes in pressure-volume relationships in the right heart); 4) If necessary, propagate the changes in state through the relevant parts of the circulatory system; and 5) Generate an explanation that accounts for the end-state (e.g., increased cardiac output) described in the problem statement.

The following three problems are indicative of perturbations in the venous system.

Ap3. An experiment is performed in which a box is placed securely around a subjects lower extremities, and negative pressure is created in the box which causes blood to pool in the legs. What would be the consequences for cardiac output.

Ap4. In severely acute hemorrhage (a loss of 700 ml of blood), the cardiac output can be dramatically reduced. Explain the reasons for the low cardiac output.

Ap5. Speculate on how a large tumor in the liver can dramatically reduce cardiac output.

The first problem describes an experiment which causes pooling of blood in the legs. The situation was analogous to a sudden change in posture from supine position (lying down) to an erect position, or to adjusting to conditions of zero gravity (Smith & Kampine, 1990). This causes blood to pool in the

lower extremities and reduces stressed vascular volume, mean systemic pressure, and cardiac output. The subjects' responses to this problem are presented in Table 22.

Table 22

Subjects' Responses to Problem Concerning Pooling of Blood in the Extremities (Ap3).

Responses	P	1.1	1.2	1.3	2.1	2.2	3.1	3.2	4.1	4.2	R1	R2	Ph	CP	AC	Totals
Decreased venous return	1	1	1	1		1	1	1	1		1	1	1	1	1	13
Decreased stressed vascular volume						1					1					2
Decreased mean systemic pressure						1										1
Accumulation of blood in legs	1	1														2
Decreased Cardiac output	1	1	1	1		1	1	1	1		1	1	1	1	1	13
Increased unstressed vascular volume						1							1			2
Increasing venous capacitance							1	1	1							3
Decreased preload						1	1					1				3
Totals	3	3	2	2	0	6	4	3	3	0	3	3	3	2	2	39

The subjects found the problem to be rather facile.[36] Thirteen subjects recognized that venous return would be decreased, and each of these subjects suggested that this would decrease cardiac output. Typically, subjects did not elaborate very much on the mechanisms. A second year student (2.2) provided the most complete response, identifying each of the significant changes in venous factors. A fourth year student (4.2) suggested that cardiac output would initially fall and then rise. The other second year student (2.1)

[36]In retrospect, the problem should not have indicated that blood would pool in the extremities.

stated that there would be an increase in venous return. Here is an excerpt from his response:

Excerpt from a second year medical student (2.1)

1. Well that's going to force blood
2. from the veins in the leg up
3. and so you will have an increase in venous return
4. and an increase in cardiac output.

E. Can you elaborate a little bit on that?

5. You increase the pressure on the veins.
6. The legs veins are large storage volume.
7. So if you increase the pressure around them,
8. you're going to decrease their volume
9. and blood has to go somewhere
10. and it can't flow backwards because of the valves.
11. So it has to flow forward into the heart.

This a most interesting response because the subject misrepresented or disregarded significant aspects of the problem, namely that a *negative pressure* is induced and blood pools in the lower extremities. This student had a misconception concerning the effects of resistance and compliance discussed in a previous section and this misconception is evident in his response. He suggested that pressure on the veins is going to reduce the storage of volume (less pooling of blood!) and this would increase venous return and cardiac output.

The second problem asks subjects to explain how a severe hemorrhage can dramatically reduce cardiac output. Hemorrhage causes a loss of circulatory volume and a drop in stressed volume which produces a decrease in mean systemic pressure and a reduced venous return.[37] The subjects' response to this problem is summarized in Table 23.

[37]This is followed by a number of systemic compensatory responses. Subjects were instructed to focus on the changes prior to compensation.

Table 23

Subjects' Responses to Hemorrhage Problem (Ap4).

Responses	P	1.1	1.2	1.3	2.1	2.2	3.1	3.2	4.1	4.2	R1	R2	Ph	CP	AC	Totals
Reduced venous return	1	1		1	1	1			1	1	1		1	1	1	11
Reduced stressed vascular volume					1								1		1	3
Loss of circulating volume/ hypovolemia	1	1	1	1		1	1	1	1		1	1		1		11
Reduced mean systemic pressure					1	1									1	3
Loss of pressure	1	1	1	1										1		5
Decreased Preload	1									1						2
Decreased mean arterial pressure							1									1
Compromised stroke volume										1						1
Totals	4	3	2	3	3	3	2	1	2	3	2	1	2	3	3	37

Each subject understood that a loss of circulating volume could reduce cardiac output. The responses of most subjects indicates that they followed a simple pattern of inferences including: a loss of circulatory blood volume causing a drop in venous return, and as a result, a reduced cardiac output. Three subjects, including both second year students and the academic cardiologist, suggested that stressed vascular volume and mean systemic pressure would be reduced. The two residents described the compensatory effects in considerable detail, even though that was not of immediate interest in this study.

The third problem asks subjects to speculate on how a large tumor in the liver can dramatically reduce cardiac output. There are several ways in which a tumor in the liver can reduce cardiac output. The most likely possibility is that the tumor compresses a major vessel, such as, the inferior

vena cava and/or the hepatic portal vein. Both of these veins are situated in close proximity to the liver. This compression could produce a rather large increase in resistance to venous return. Another possibility is that the tumor is very vascularized and blood accumulates in the liver reducing venous return. The subjects' responses to this problem are summarized in Table 24.

Table 24

Subjects' Responses to Tumor in the Liver Problem (Ap5).

Responses	P	1.1	1.2	1.3	2.1	2.2	3.1	3.2	4.1	4.2	R1	R2	Ph	CP	AC	Totals
Reduced venous return	1	1	1	1	1	1	1	1	1	1	1	1	1	1	1	15
Compresses the inferior vena cava				1	1		1	1	1	1	1	1		1	1	10
Compresses the hepatic portal vein		1		1	1	1		1		1			1		1	8
Pooling of blood in the liver	1	1	1								1	1	1	1		7
Increase Venous resistance			1										1		1	3
Obstruct arterial blood flow	1		1			1				1						4
Increased afterload										1						1
Increase pressure flow/output to compensate	1		1	1												3
Decreased preload						1										1
Hepatic hypertension						1					1					2
Decreased circulating volume									1			1				2
Shunting of blood						1					1	1				3
Hypercalcemia decreases contractility and heart rate												1				1
Totals	4	3	5	4	3	6	2	3	3	5	5	6	4	3	4	60

The subjects responded with considerable accuracy and insight to this problem, identifying a range of possible ways in which a tumor in the liver can reduce cardiac output. Each subject suggested that venous return would likely be reduced. Thirteen subjects identified either the inferior vena cava or the hepatic portal vein as the vessels most likely to be affected by a tumor in the liver. Five subjects, including a first year medical student (1.3), both second year medical students, a third year student (3.2), and the academic cardiologist, suggested both vessels. Seven subjects, including the premedical student, two first year students (1.1, 1.2), both residents, the physiologist and the cardiologist practitioner, suggested that the pooling of blood in the liver as one reason for a decrease in cardiac output. Three subjects, including a first year medical student, the physiologist and the academic cardiologist, stated that there would be an increase in venous resistance. Both residents raised a wide range of possibilities, only a subset of which are included in Table 24. These residents were able to use their clinical expertise to good advantage in this problem.

The premedical student responded to this problem with some skepticism. This is reflected in an excerpt from his response:

Excerpt from a premedical student

4. I would think it would be the opposite in terms of mechanical factors.
5. A large tumor would block the arteries.
6. So I would think we would need a greater pressure of blood
7. and a greater outflow to overcome that.
8. I would think the opposite would happen.
9. if we are talking in terms of mechanics,
10. I would think that if a large tumor is obstructing an artery or a vein,
11. you would have more outflow
12. to overcome this obstruction,
13. to increase the pressure,
14. so it could flow more easily through the liver.
15. I don't know if I could answer any better.

16. The question seems to imply that cardiac output is decreased,

The response to this problem provides some further insight into the nature of the pressure-volume misconception that was evident in nis earlier responses. The subject suggested that a pressure buildup requires a greater force, more specifically, a greater outflow of blood. The implication of this is that the heart responds to obstructions by increasing flow to compensate. The response suggests a kind of teleological reasoning. This pattern of thinking was also evident in some of the responses provided by the first year medical students.

The two ventricles are the primary pumps in the heart. The right ventricle propels blood through the pulmonary artery to the lungs (pulmonary circulation). The left ventricle pumps blood through the aorta and systemic arteries to the remainder of the body (systemic circulation). Normally, both ventricles eject about 5 liters of blood per minute. To maintain an even flow, the ventricles need to remain in synchronization. However, in the event of sudden unilateral heart failure, there can be a disequilibrium. The subjects were presented with the following problem:

Ap6 In acute left ventricular failure, the left ventricular output is reduced to 1.5 liters per minute and the right ventricle still pumps at a rate of 5 liters per minute. Explain the events that tend to restore the equilibrium. Do not consider nervous system or hormonal factors.

The problem suggests an acute disturbance in function, the sudden failure of the left ventricle, resulting in a disequilibrium between the two ventricles. The situation results in a series of behavioural changes in the system and provides an opportunity to determine the extent to which subjects

can propagate the effects of the disturbance throughout the circulatory system. A simple schematic of the circulatory system is presented in Figure 31.

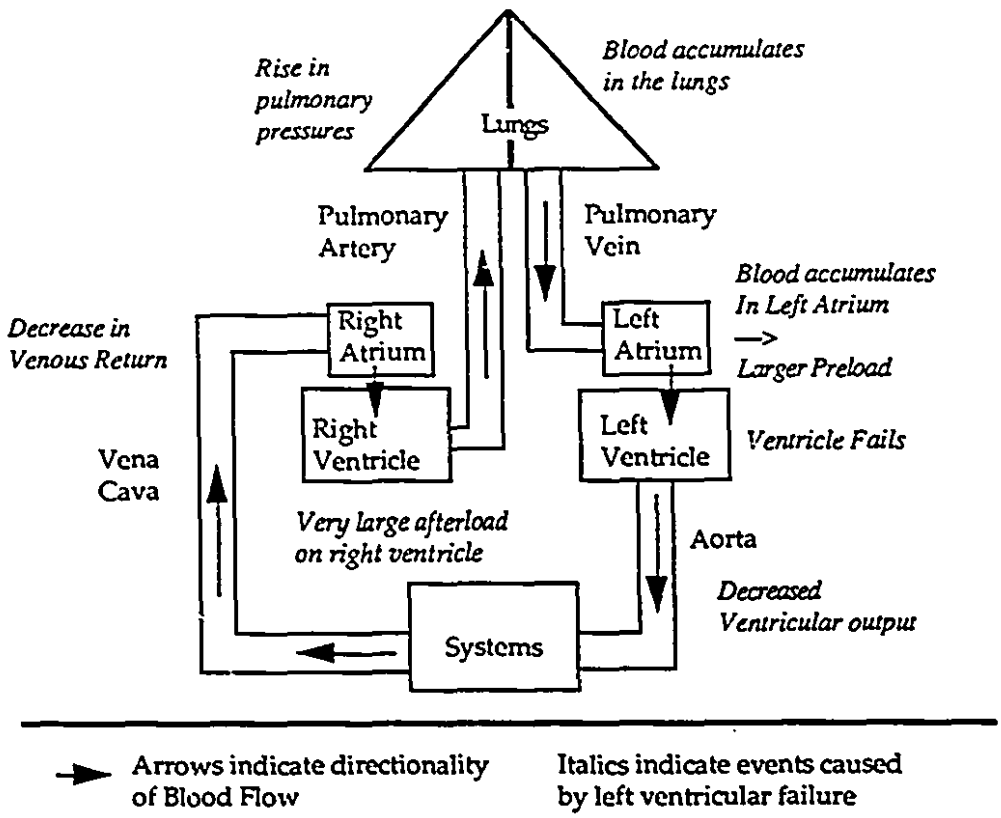


Figure 31. Schematic Representation of the Circulatory System Following Acute Left Ventricular Failure.

Following a fall in left ventricular failure output, there will be a rise in the end-systolic volume and therefore, a rise in end-diastolic volume and a rise in end-diastolic pressure in the left heart. This will result in a rise in pulmonary pressures. A rise in pulmonary pressures will increase the afterload on the right ventricle. A rise in afterload on the right ventricle will result in a rise in right ventricular end-systolic volume and eventually, right ventricular end-diastolic volume and therefore, a rise in right atrial pressure.

A rise in right atrial pressure will decrease the gradient for venous return and therefore decrease cardiac output. However, the other factor to take into account is the accumulation of fluids through all the veins with the backup of end-systolic volume. The ensuing increase in end-diastolic volume will help improve cardiac output to the extent that the failing left ventricle is capable of delivering an increase in blood ejected. So the end-result would be a much lower cardiac output with a higher right atrial pressure, and the output will be higher than the initial 1.5 liters per minute. The subjects' responses are summarized in Table 25.

Each subject, with the possible exception of a first year student (1.2), was able to construct a plausible mechanistic account of the situation and provide some kind of explanation of the events that would restore the equilibrium. Every individual was able to situate the source of the difficulty and predict some of the subsequent effects. Thirteen subjects suggested that right ventricular output would decrease. Twelve subjects, excluding the premedical student, a first year student (1.2), and a second year student (2.1), stated that there would be considerable build up of pressures throughout the system, resulting in changes in pressure-flow relationships in various segments of the circulation.

Table 25

Subjects Responses to Acute Left Ventricular Problem (Ap6).

Responses	P	1.1	1.2	1.3	2.1	2.2	3.1	3.2	4.1	4.2	R1	R2	Ph	CP	AC	Totals
Rise in LV end-diastolic pressures												1	1	1	1	4
Rise in pulmonary pressures		1				1	1		1	1	1	1	1			8
Rise in right atrial pressure						1		1	1		1					4
Rise in left atrial pressure		1					1		1	1	1	1		1		7
Increased afterload on the RV				1			1		1		1	1	1		1	7
Decreased venous return		1	1	1	1	1					1			1		7
Accumulation of blood in the lungs							1	1		1				1	1	5
A decrease in RV output		1		1	1	1	1	1	1	1	1	1	1	1	1	13
A slight increase in LV output	1	1				1	1		1		1	1	1		1	9
Accumulation of blood In LV	1							1								2
Decreased LV afterload						1							1			2
Increased LV stroke volume						1							1			2
Right heart failure								1							1	2
Decrease in mean systemic pressure															1	1
Totals	**2**	**5**	**1**	**3**	**2**	**7**	**6**	**5**	**6**	**4**	**7**	**6**	**7**	**5**	**7**	**73**

LV—Left Ventricle RV—Right Ventricle

Subjects were able to exploit their knowledge of the anatomical structure of the system and propagate the effects of the sudden disturbance in function. The following excerpt from a first year student illustrates this point.

Excerpt from a first year medical student (1.1)

3. Okay, you've got a build up of blood in the left ventricle.
4. The right ventricle basically pumps it to the pulmonary system.
5. The pulmonary system brings it back into the left atrium,
6. pumps into the left ventricle
7. which then pushes it into the system.
8. If your right ventricle is pumping at a rate of 5 liters per minute,
9. um, quite a lot of blood going to the pulmonary system
10. and it's going to come to the left atrium.
11. But it could get blocked up
12. because the ventricle is failing
13. and less blood is being pumped out.
14. So eventually what is going to happen is
15. there is going to be a build up of pressure in that whole area
16. in the pulmonary system
17. and in the left atrium
18. and the right ventricle.
19. However,
20. essentially because the blood backs up,
21. because you have a decreased flow going through the system.
22. You'll have a decreased venous return
23. and you'll have a decreased amount of blood
24. that is going through the right atrium,
25. right ventricle,
26. pulmonary system,
27. left atrium.

.....

The student constructed an explanation based on her understanding of the structure-function relationships. As was evident in her previous responses, the student lacked a process vocabulary (terms such as afterload) for describing changes in the system. Nevertheless, she was able to convey the sequence of changes in pressure-flow relationships, from a buildup of blood in the pulmonary system resulting in pressure changes to a decrease in venous return to the right atrium. A more precise account of the mechanical events that restore the equilibrium was provided by a third year student (3.1).

Excerpt from a third year medical student (3.1)

1. You're going to have blood piling very soon
2. somewhere.
3. Well, you have blood accumulating
4. somewhere between the left ventricle and the right ventricle
5. at a rate of 3.5 liters per minute,
6. between the right ventricle
7. and the left ventricle
8. is in the lungs,
9. pulmonary arteries,
10. pulmonary veins
11. and left atrium.
12. And the more blood you put in there
13. the higher the pressure gets
14. and that is going to maybe
15. increase the output of the left ventricle
16. a little bit.
17. And it's also going to decrease the output of the right ventricle
18. because it's going to be seeing a much greater afterload.
19. So what's going to happen is
20. bloods going to pile up,
21. the blood vessels it's in are going to stretch
22. until the pressure is at a reasonable gradient.
23. At equilibrium, the right ventricle will be pumping out
24. the same as the left ventricle is.
25. But the left ventricle is going to be pumping out less
26. because it's failed.
27. Whereas the right ventricle is going to be pumping out less
28. because it's seeing a humongous afterload.

....

37. Because if there is a big backup of blood in the lungs,
38. it maybe be able to give it a bigger preload.
39. So it might increase its output a little bit.

The analysis began with the recognition that blood is accumulating in the vessels and structure between the right ventricle and the left ventricle at a rate of 3.5 liters per minute. This causes a 'humongous' afterload that impedes right ventricular output. The student also suggested that forward pressures on the left side might be able to give a bigger preload and increase left ventricular output.

The response to this problem suggests that subjects were able to demonstrate an understanding of pressure-flow and pressure volume relationships in a specific context. The basic physiology questions, which tended to require fewer inferences, but were more decontextualized, presented considerably more difficulty for some subjects than this applied problem.

Specific pathophysiological conditions can produce dramatic increases in venous return. An arteriovenous (A-V) fistula is an abnormal communication between the artery and vein that results from a congenital abnormality or from injury. The subjects were asked the following problem concerning an A-V fistula:

Ap7 Discuss the effects of creating an A-V fistula (an abnormal communication between artery and vein due to trauma or a surgical procedure) on venous return.

An A-V fistula allows the high pressure arterial system to empty directly into the veins, bypassing the arterioles and the capillaries which would normally buffer arterial pressure. There is a very large increase in the pressure gradient for venous return and a simultaneous reduction in venous resistance. In addition, there could also be a reduction of afterload which would increase cardiac output. These factors combine to produce a very substantial increase in venous return. The subjects responses for this problem are presented Table 26.

Table 26

Subjects Responses to the A-V Fistula Problem (Ap7).

Responses	P	1.1	1.2	1.3	2.1	2.2	3.1	3.2	4.1	4.2	R1	R2	Ph	CP	AC	Totals
Increased venous return	1	1		1	1	1	1	1		1	1	1	1	1	1	13
Decrease venous resistance						1				1			1	1		4
Increased cardiac output										1	1	1				3
Bypass capillaries and/or arterioles		1		1		1				1		1	1		1	7
Increased forward pressure on venous side		1			1	1	1	1		1	1		1	1	1	10
Increased preload/Stroke Volume					1		1					1				3
Decreased afterload										1						1
Equilibration of mean arterial pressure and mean systemic pressure											1					1
Hyperdynamic circulating conditions												1				1
Totals	1	3	0	2	3	4	3	2	0	6	4	5	4	3	3	43

Subjects had relatively little difficulty in explaining this problem. Thirteen subjects correctly predicted an increase in venous return. Only a first year student (1.1) and a fourth year student (4.1) did not seem to understand the problem. Twelve out of fifteen subjects suggested that either the blood flow would bypass a circulatory segment such as the arterioles in which there is a buffering of pressure and/or that the fistula would produce an increase in forward flow pressure.

The premedical student erroneously suggested that venous return would increase due to the fact that body tissues are not getting sufficiently oxygenated. A first year student (1.2) predicted that the drop in pressure

would cause a drop in venous return. A fourth year student (4.1) suggested that venous return would decrease because "the vein that is probably contributing to venous return before is now connecting in the opposite direction". All of these responses indicate a misrepresentation of the nature of the structural defects produced by an A-V fistula and the ensuing behavioural consequences.

Medical Disorders

The following two problems describe medical disorders that have particular hemodynamic consequences. In both problems the disorder and its effects on cardiac output are presented to the subjects. The subjects were asked to provide a causal explanation accounting for the effects on cardiac output. The problems are as follows:

Ap8 Explain how it is possible for a patient to be suffering from congestive heart failure and yet have essentially normal cardiac output.

Ap9 In hyperthyroidism, the metabolism of all the tissues of the body become greatly increased and cardiac output often increases by 40 to 80%. Discuss the mechanical factors in the circulation that could lead to this unusual increase in cardiac output.

Congestive heart failure (CHF) is a syndrome in which the heart is unable to pump blood at a rate commensurate with systemic metabolic requirements (Smith & Kampine, 1990). Among the many causes of CHF are a degeneration of cardiac muscle and an enlarged heart due to high pressure overloads (e.g., as in renal failure). Typically, CHF results in a marked decrease in cardiac function. However, ancillary effects and compensatory mechanisms can maintain a normal cardiac output. Fluid retention and pulmonary edema are commonly associated with CHF. This produces an

increased left ventricular end-diastolic volume and end-diastolic pressure, which results in a larger preload. This increased diastolic stretch results in an increased cardiac function, with a normal stroke volume, a larger end-systolic volume, and a diminished ejection fraction (the ratio of stroke volume to end-diastolic volume). Overtime, the heart can become rather enlarged and dilated which makes it capable of holding large volumes of blood. Until a certain point of deterioration, the heart can continue to eject a normal volume of blood. The subjects' responses for this problem are presented Table 27.

The responses to this problem suggest a more prominent expertise effect. The problem of congestive heart failure provides an opportunity for subjects to draw on their clinical knowledge. The four clinicians, including the two residents and the two practitioners, provided the most complete and accurate responses. Five subjects, including a second year student (2.1), a fourth year student (4.2), both residents, and the academic cardiologist, suggested that there would be an increase in end-diastolic volume, implying an increase in preload. Seven subjects, including both third and fourth year students, both residents, and the cardiologist practitioner, indicated that pulmonary edema would lead to high pulmonary venous pressures. Five of these seven subjects also indicated that high pulmonary pressures would produce raised left atrial pressures. Seven subjects, including a first year student (1.2), both second year students, a third year student (3.2), a fourth year student (4.1), the physiologist, and the academic cardiologist, suggested that heart rate would increase to compensate for CHr. Four subjects, including a second year student (2.1), a fourth year student, a resident (R2), and the academic cardiologist, recognized that despite the normal stroke volume, the ejection fraction would be diminished.

Table 27

Subjects Responses to the Congestive Heart Failure Problem (Ap8).

Responses	P	1.1	1.2	1.3	2.1	2.2	3.1	3.2	4.1	4.2	R1	R2	Ph	CP	AC	Totals
Increased end-diastolic volume					1					1	1	1			1	5
Increased preload							1			1	1					3
Increased heart rate			1		1	1		1	1				1		1	7
A decrease in afterload						1									1	2
Pulmonary edema leading to high venous pressures							1	1	1	1	1	1		1		7
High EDP and ESP in left atrium							1			1	1	1		1		5
Large dilated heart with large volume capacity					1					1		1				3
Contractility goes up enough													1			1
Normal cardiac output at rest but not on exertion														1	1	2
CHF maybe caused by diastolic filling problems														1		1
Larger end-sysstolic volume															1	1
Decreased ejection fraction					1					1		1			1	4
Totals	**0**	**0**	**1**	**0**	**4**	**2**	**3**	**3**	**2**	**4**	**5**	**5**	**2**	**4**	**6**	**41**

The premedical student and first year medical students could not suggest any plausible explanations to this problem. The following excerpts illustrate this point.

Excerpt from a first year medical student (1.1)

8. It could be passing through the same amount of blood,
9. but just not doing it in an all rhythmic fashion.
10. It could be blooping it out in little bits,
11. instead of doing it in a nice.

Excerpt from a first year medical student (1.3)

5. Are the ventricles behaving normally?
6. They might not, right?
7. So there must be something else accounting for the normal output.
8. (long pause).
9. I'm thinking that there is something
10. that is accounting for this output.
11. That is bypassing the heart or something.
12. (long pause).
13. Maybe, I don't know,
14. maybe it's,
15. Could essentially normal cardiac output under congestive heart failure
16. be a very bad sign
17. because it means that blood is being lost in the lungs?

The first year medical student (1.1) depicts a ventricle that kind of works in spurts, 'blooping out' bits of blood. The second subject (1.3) explores the possibility of alternative anatomical configurations (possibly like an A-V fistula). Neither subject has sufficient grasp of congestive heart failure to propose a plausible alternative. Although, their intuitions reflect some understanding of ways in which the heart can malfunction.

One second year medical student (2.2) suggested a higher heart rate and a decreased afterload as possible compensatory mechanisms. A third year student developed a lengthy explanation concerning how renal function can increase blood volume. The other second (2.1) and third year students (3.1) and both fourth year students provided explanations in terms of pulmonary edema and/or changes in end-diastolic volume. The physiologist,

who does not have extensive knowledge of clinical medicine, suggested heart rate and increased contractility as possible mechanisms.

The four physicians were very knowledgeable concerning the possible causes of CHF. Both cardiologists qualified the normal cardiac output description by suggesting that this would be observed only at rest and not on exertion. The practitioner also indicated that CHF could be a result of diastolic filling problems and other non systolic problems, in which case you might still see a normal cardiac output.

The most coherent explanation was provided by a resident (R1). His response is represented in a semantic network in Figure 32. The subject provided an exemplar of congestive failure in the form of an infarct (death of heart muscle tissue because of a lack of blood supply). The infarct causes a loss of ventricular muscle mass. The heart attempts to compensate by raising left ventricular end-diastolic pressure and, via the Starling mechanism, increase preload to the extent possible. The raised end-diastolic pressure eventually transmits pressure to the left atrium which, in this condition, raises pulmonary venous pressure to the extent that the pulmonary veins may become 'leaky'. This leads to pulmonary edema and increased pulmonary vasculature pressure. However, if the raised left ventricular end-diastolic pressure is high enough to compensate for the decrease in left ventricular contractility, then a normal cardiac output can be seen in association with congestive heart failure.

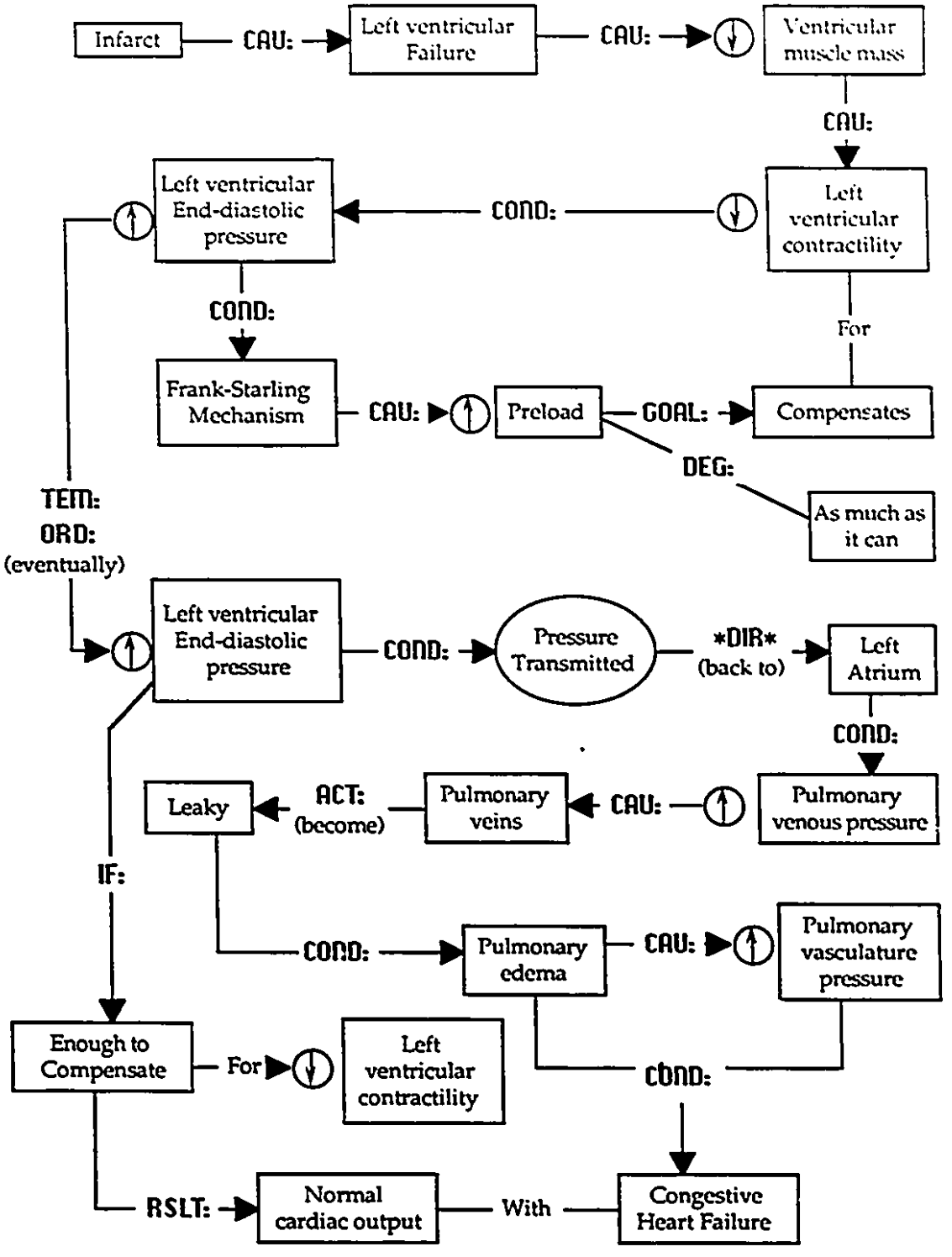


Figure 32. Semantic Network Representation of Response by a Resident (R1) to CHF Problem (Ap8).

The response indicates how a clinical exemplar can be used effectively to elaborate the underlying pathophysiological manifestations of disease. The physicians experienced considerably more difficulty in using clinical exemplars to respond to the abstract physiological questions in earlier sections in the study. This may be due to the fact that the analogical mapping is more distal between the physiological and clinical levels of description than it is between the pathophysiological and clinical.

The second problem (Ap9) focuses on the metabolic disorder of hyperthyroidism and the factors that result in a hyperdynamic cardiac state (high cardiac output). This is a very difficult problem, which has only recently become better understood by medical researchers (Goldman, Olajos, & Morkin, 1984). It is well known that a hyperthyroid state results in an increased heart rate and an increase in contractility. However, the primary determinants are venous factors, specifically, an increased *mean systemic pressure* resulting from an increase in *stressed volume* and a decrease in *venous resistance*. Therefore an increase in cardiac output must also be accompanied by an explanation for the change in the pressure gradient for venous return. It would be unreasonable to expect that subjects were aware of the most recent findings. We were interested in the range of factors considered by subjects. The responses to this problem are summarized in Table 28.

Table 28

Subjects Responses to the Hyperthyroidism Problem (Ap9).

Responses	P	1.1	1.2	1.3	2.1	2.2	3.1	3.2	4.1	4.2	R1	R2	Ph	CP	AC	Totals
Increased contractility			1	1	1	1	1	1	1	1	1	1	1		1	12
Increased heart rate				1			1	1		1	1	1	1	1		8
Stroke volume	1	1		1				1								4
Decreased afterload									1							1
Arterial Vasodilation						1			1							2
Decreased arterial resistance									1							1
Decreased peripheral resistance														1		1
Arterial vasoconstriction										1						1
Increase stressed Vascular Volume											1					1
Increased venous Pressure					1											1
Decreased Venous Resistance															1	1
Metabolic/ oxygen demands	1	1								1						3
Sympathetics						1	1			1		1	1		1	6
Totals	2	2	1	3	2	3	3	3	4	5	3	3	3	2	3	42

The hyperthyroidism problem proved to be rather difficult. No subject correctly accounted for the situation. Thirteen subjects attributed the hyperdynamic cardiac state to an increase in contractility and/or an increase in heart rate. Six subjects, including a second year student (2.2), a third year student (3.1), a fourth year student (4.2), a resident, the physiologist, and an academic cardiologist, correctly suggested that sympathetic factors were involved.

Only three of the subjects made any reference to any of the venous return factors. A second year student (2.1) explained the problem in terms of

its effect on venous pressure, that is, increasing the pressure gradient for venous return, and the academic cardiologist focused on a diminished venous return. A resident (R1) was the only subject to explain the effects of hyperthyroidism in terms of an increase in stressed vascular volume—the most significant factor, and its effect on the pressure gradient.

This problem reveals a cardiocentric bias in that subjects clearly favored explanations framed in terms of cardiac factors, with little consideration given to venous return factors. Hyperthyroidism is an endocrinological problem, and cardiologists would not routinely encounter such a problem in clinical practice. The responses given to this problem suggested that even expert subjects experience difficulty applying familiar concepts in unfamiliar contexts.

Clinical Problems

This section focuses on three brief clinical problems that were designed to test subjects' abilities to apply concepts pertaining to cardiac output and venous return to diagnostic situations. The problems are as follows:

Ap10 A patient has a fever, rigor, shaking chills, BP=80/60, skin is warm and clammy. A catheter is put into the pulmonary artery and Cardiac output is 9 L/min., left atrial pressure is 8 mm Hg and right atrial pressure is 2 mm Hg. Explain the possible underlying pathophysiology based on the evidence you have.

Ap11 A 26 year old male suffered a motorcycle accident three months ago, and fractured his femur. One week ago he had his cast removed. He subsequently complained of shortness of breath, light-headedness, and pleuritic chest pain. On examination his blood pressure was 80/60 and his jugular venous (central venous) pressure was 16 cm (normal is <8). Explain why the blood pressure is low.[38]

[38]Problems Ap11 and Ap12 also asked subjects to propose a therapeutic approach to each problem. The analysis of this part of the problem is not included here.

Ap12 A patient suffers a rupture of his mitral valve, She presents with cold, clammy extremities, altered mentation, decreased urine output, BP=80/60, jugular venous pressure (central venous pressure) = 12 cm. (0-8). Explain why the blood pressure is low.

The first problem presents a seemingly anomalous situation, in which a patient presents with low blood pressures and high cardiac output. The subjects were asked to explain the possible underlying pathophysiology. This problem represents a classic example of septic shock. Circulatory shock is a failure of the circulation to adequately oxygenate tissue. Septic shock is usually the result of a severe infection and is a direct result of endotoxins (a toxin present in bacterial cells) in the blood. The typical patient presentation of septic shock, as is the case with this problem, involves a high cardiac output, elevated pressures, and a low blood pressure. This is also known as the warm phase in which the patient presents with warm and clammy skin and a hyperdynamic state. Although the exact mechanisms are not known, the pathophysiology is characterized by a low peripheral resistance, and a decrease in stressed vascular volume. This may be followed by recruitment of unstressed volume to stressed volume that might explain the increase in venous return despite an elevated right atrial pressure. There is a decreased arterial resistance which can account for the low blood pressure and may result in inadequate perfusion. The subjects' response to the septic shock problem is presented in Table 29.

Table 29

Subjects' Responses to the Septic Shock Problem (Ap10).

Responses	P	1.1	1.2	1.3	2.1	2.2	3.1	3.2	4.1	4.2	R1	R2	Ph	CP	AC	Totals
Patient has an infection					1		1	1								3
Patient is in shock		1														1
Septic Shock							1	1	1	1	1	1		1	1	8
Warm phase									1							1
Hyperdynamic state							1		1	1		1		1		5
Low vascular/ venous resistance	1						1				1			1	1	5
Increased heart rate.	1					1										2
Increased contractility											1					1
Reduced afterload										1						1
Increased stressed volume											1					1
Increased venous pooling										1						1
Peripheral vasodilation								1		1		1				3
Presence of endotoxin											1					1
Systemic heart failure			1													1
Possible endocarditis					1											1
Greater demand for oxygen	1															1
Internal bleeding		1		1												2
Some sort of clot		1														1
Peripheral circulatory failure															1	1
I don't know*			1	1	1	1							1			5
Totals	3	3	1	1	2	1	4	3	3	5	5	3	0	3	3	40

* Response is not included in the totals

The responses indicate that subjects with clinical experience were immediately able to recognize septic shock. Every subject from third year medical students to the cardiologists, with the exception of the physiologist, identified the source of the problem. Five subjects, including the premedical

student, a third year student (3.1), a resident (R2), and both cardiologists, suggested a low vascular and/or venous resistance. Most subjects did not elaborate much on the pathophysiological mechanisms.

The physiologist experienced particular difficulties in explaining the clinical problems. This is not surprising since he is not involved in the practice of clinical medicine. The premedical student and the first and second year medical students could not identify the source of the problem. However, they were able to produce some interesting hypotheses. Here are two excerpts that illustrate this point.

Excerpt from the premedical student

11. Okay he has a fever
12. and his temperature has increased.
13. how does temperature affect the cardiovascular system.
14. How does a cold affect the cardiovascular system?
15. The body is trying to fight this illness he's got.
16. So it's working harder.
17. It demands more oxygen.
18. As a result the oxygen demand must be greater
19. and the heart output has to be greater
20. and everything else follows.

Excerpt from a first year medical student (1.1)

11. Well this person doesn't appear to be circulating blood very effectively.
12. there appears to be some sort of,
13. something is blocking it up,
14. increasing the pressure of the atriums.

.....

35. The skin is warm and clammy,
36. doesn't that happen during shock.
37. Um, that doesn't explain (long pause).
38. Okay see I don't get this.
39. If you do have a high cardiac output and low blood pressure,
40. why are,
41. what's maintaining the pressure in the other two areas.

The premedical student, in responding to the applied problems frequently referred to the need for the heart to work harder to supply the tissues of the body with oxygen. In this case, he was partly correct since the tissues are not being properly perfused and the patient is in a high output state. The first year medical student was able to pick up specific cues and made some correct inferences from the available evidence. However, she was unable to tie the strands together and account for the problem.

The second problem (Ap11) describes a patient who had a motorcycle accident that required him to wear a cast for three months. The problem represents a prototypical case of a pulmonary embolism. The embolism is a result of a clot that formed while the patient was wearing the cast. The *shortness of breath*, *light-headedness*, and *pleuritic chest pain* are all symptoms indicative of pulmonary complications. The *elevated jugular venous pressure* is indicative of a significant increase in afterload on the right ventricle that is causing right-sided heart failure. Blood pressure is a product of cardiac output and arterial resistance. Therefore, a drop in cardiac output can result in decreased blood pressure. Subjects' responses to this problem are presented in Table 30.

Table 30

Subjects' Responses to the Embolism Problem (Ap11).

Responses	P	1.1	1.2	1.3	2.1	2.2	3.1	3.2	4.1	4.2	R1	R2	Ph	CP	AC	Totals
Cast caused a clot to form in leg		1									1		1			3
Embolism					1	1	1									3
Pulmonary embolism						1	1		1	1	1	1		1	1	8
Increased afterload on the right side							1		1	1	1			1	1	6
Right-sided heart failure					1		1	1				1			1	5
Decreased cardiac output	1	1						1	1	1	1	1				7
Decreased preload							1	1	1	1		1		1		6
Atrophy of muscles													1			1
Increased vascular resistance											1					1
Arteries narrowed	1															1
Increased pressure in right heart					1				.							1
Pulmonary edema				1				1								2
Hemmorhage			1	1												2
Blood accumulating in lungs			1													1
Blood pooling in the veins		1				1	1									3
Totals	**2**	**3**	**2**	**2**	**3**	**3**	**6**	**4**	**4**	**4**	**5**	**4**	**2**	**3**	**3**	**50**

To solve the problem, one must first have the insight that there is an embolism, and that the symptoms indicate that it is lodged in a pulmonary vessel. Eight out of fifteen subjects, including a second year student (2.2), a third year student (3.1), both fourth year students, both residents, and both cardiologists, recognized that there was a pulmonary embolism. These subjects recognized the evident pulmonary symptoms, such as shortness of

breath. Each of these subjects was able to develop a causal explanation of why the blood pressure was low. Six of the eight subjects suggested that the elevated jugular venous pressure was indicative of a high afterload on the right ventricle and that this was diminishing cardiac function. Three of these subjects also added that there was evidence of right heart failure.

The clearest exposition of the causal sequence of events was provided by a third year student (3.1). Here is an excerpt from her response:

Excerpt from a third year medical student (3.1)

I think he has a pulmonary embolus from his leg,
a DVT, deep vein thrombosis in his leg
because that's what we seem to be suggesting in this scenario.
He's been lying in his bed for three months
and now he's got shortness of breath and chest pain.
So lets say that he's got a pulmonary embolus.
His blood pressure is low.
.....
What happens is you have gloop of blood
going from your left ventricle to your right atrium
and then through your lungs
and if you block it up in your lungs right here,
all the cars move down the highway
and they all pile up over here.
So your right ventricle is now pumping against,
into a huge afterload.
So that's why it's failing,
that's why it's allowing blood to back up,
as in the JVP (jugular venous pressure),
and it's not put out very much past that stupid embolus.
So your left ventricle isn't seeing much of,
much of a preload.
So it's not putting out much.
So it's not keeping your blood pressure up.

The subject's answer began with an explanation, accounting for how the patient came to have a pulmonary embolism. She then explained the sequence of hemodynamic effects beginning with a backup of pressure from the left ventricle leading to elevated pressures and a huge afterload. The

increased afterload is causing a further backup of blood and limiting venous return and the preload to the left ventricle. This is why the blood pressure is low.

The first year students and the premedical student, having had very little experience with clinical problems, could not recognize the source of the problem. They attributed the difficulties to a narrowed artery near the femur or an atrophied muscle, or a hemorrhage. One medical student (1.1) suggested the possibility of a clot, and another first year student recognized that there were pulmonary problems. The physiologist had considerable difficulty explaining the clinical situations. A third year student (3.2) recognized the pulmonary symptoms and the right-sided heart failure, but could not explain the etiology of the problem. A second year student (2.1) recognized that there was an embolism "lodged in his heart or somewhere near it" but did not recognize the pulmonary symptoms. The two second year students considered other possibilities, such as an atrophied muscle causing an increase in the compliance of a vein before they realized that this could not account for the pulmonary problems.

The third problem (Ap12) describes a patient who has just suffered a rupture of the mitral valve and presents with symptoms of circulatory shock. The mitral valve is the valve between the left atrium and the left ventricle. This problem, like the previous one, requires subjects to explain why the blood pressure is low. When the mitral valve ruptures, with each contraction, there is a backward regurgitation of blood, as well as some forward flow. Consequently, the forward cardiac output decreases. If the forward cardiac output decreases for any given systemic vascular resistance, there would be a fall in arterial blood pressure. The subjects' responses to this problem are presented in Table 31.

Table 31

Subjects' Responses to the Mitral Valve Rupture Problem (Ap12).

Responses	P	1.1	1.2	1.3	2.1	2.2	3.1	3.2	4.1	4.2	R1	R2	Ph	CP	AC	Totals
Backward regurgitation of flow with contraction	1	1	1	1	1	1	1	1	1	1	1	1	1	1	1	15
Loss of pressure gradient		1	1	1	1		1	1	1	1	1	1	1	1		12
Diminished contractile force	1	1	1		1		1	1		1	1					8
Decreased forward cardiac output /stroke volume	1	1	1		1	1	1			1	1	1	1		1	11
Not getting enough blood to extremities			1		1			1								3
Raised left atrial pressure												1				1
Blood has accumulated in the lung							1	1			1					3
Elevated pulmonary venous pressures							1									1
Right heart is failing							1									1
Pulmonary edema								1				1				2
Decreased systemic circulation									1	1						2
Shock syndrome														1		1
Increased peripheral resistance							1								1	2
Totals	3	4	5	2	5	2	8	6	3	5	5	5	3	3	3	62

This problem presented no difficulties for the subjects. Each of the fifteen subjects recognized that there would be a back flow of blood on each cardiac contraction. The subjects were able to explain the loss of blood pressure as resulting from either: a diminished contractile force; the loss of a pressure gradient for forward flow out of the left ventricle; or a decreased

forward ejection of blood from the left ventricle. The premedical student and the first year medical students had to first recall the exact location of the mitral valve and were then able to provide correct explanations. This problem further suggests that when subjects can exploit their knowledge of structure function relationships, they can apply physiological concepts to account for disturbances in structure or function

Summary

This section presented the results of subjects' responses to the applied problems. In general, subjects' responded better to these problems than they did to either the basic physiology questions or the integration questions. In particular, subjects responded accurately to problems that focused on specific structure-function relationships. There are several studies that cite the inherent difficulty students have in understanding these relationships (e.g., Feltovich, et al., 1989; Patel, Kaufman, & Magder, 1991). Nevertheless, it appears that structure-function correspondences are easier to comprehend and reason about than are the abstract physiological processes related to the regulation of cardiac output. Each of the subjects had a well-developed conceptual model of the anatomy of the circulatory system and could exploit this knowledge to respond to these problems.

To respond to specific problems, an understanding of clinical medicine was a definite asset. There was a more prominent effect of expertise in the accuracy of identifying and explaining the sources of these problems. In particular, the four physicians in the study, the two residents and the two cardiologists provided the most complete, accurate, and coherent responses. It was noteworthy, that one of the residents (R1), who experienced considerable difficulty responding to the basic physiology questions

consistently provided among the best responses to the problems in this section. In responding to certain clinical problems, such as the septic shock problem, third and fourth year students did noticeably better than the more junior students. The first and second year students had not yet begun their studies in clinical medicine and lacked specific knowledge to solve these problems. As would have been expected, the physiologist had difficulty responding to the more clinically-oriented problems.

The premedical student and first year medical students did not have the prerequisite knowledge to accurately respond to several of the problems in this section. As was the case in previous sections, these subjects tended to resort to a teleological mode of reasoning. For example, several of their explanations focused on the metabolic needs of muscle tissue and the body or the heart responding to fulfill these needs.

In certain cases, misconceptions that were evident in earlier sections, clearly affected subjects' responding. The premedical student who had exhibited a misconception concerning the nature of pressure-flow and pressure-volume relationships, suggested that a buildup of pressure created by a tumor in the liver, would cause cardiac output to increase. A second year student, whose earlier responses suggested a misconception related to resistance and compliance, selectively ignored certain cues in a problem and suggested that a negative pressure in the veins of the leg would reduce the storage of volume and increase venous return.

The problem pertaining to the effects of hyperthyroidism on cardiac output proved to be rather difficult for all subjects. Subjects focused almost exclusively on cardiac function determinants of blood, when in fact the problem is largely due to venous return factors.

CHAPTER 9

CONCLUSIONS AND IMPLICATIONS

This chapter presents a general discussion and summary of the results within the context of the issues raised in previous chapters. This is followed by a discussion of the limitations of this research. The last two sections evaluate the possibilities for further research and examine the instructional implications of this study.

General Discussion of the Results

The first hypothesis suggested that subjects with greater degrees of expertise and students more advanced in their training, should exhibit greater degrees of generativity and robustness in applying domain-specific concepts and provide superior explanations and more accurate predictions. In other words, the expectation was that we would observe a progression in mental models or in conceptual understanding with expertise. In general, there was an increase in understanding with expertise, as exemplified by the accuracy of prediction and explanation across question types. However, there were substantial individual differences, and there were conceptual errors and errors in analysis at each level of expertise.

The first two questions were open-ended questions that spanned the scope of the two superordinate concepts, cardiac output and venous return. The responses to these questions foreshadowed many of the findings that followed. The more expert subjects tended to provide the better responses, listing more factors and providing superior explanations. The majority of subjects were able to identify and discuss the primary determinants of cardiac output. Subjects did not produce responses as complete and as

coherent as the responses to the question concerning factors that affect blood returning to the heart. The less advanced students were particularly unsuccessful in developing an adequate account of factors that affect venous return. Certain subjects expressed a cardiocentric bias, indicating that the heart controls the flow of blood, as exemplified by the expressions, *the venous system is a passive reservoir for storing blood* and *whatever the heart pumps out it has to get back.*

Teleological Reasoning

The novice subjects, including the premedical student and the first year students, lacked a process vocabulary for expressing causal events in the system. As predicted, they frequently engaged in teleological reasoning to respond to questions and problems throughout the study. This manifested itself in two modes of expression. In the first case, subjects would suggest a response, as when the heart is reified as an active agent, trying to push blood out of its chambers. The second mode of expression is reflected by the suggestion that the system is trying to achieve a purpose or respond to a demand, such as the need to deliver oxygen to exercising tissues.

These results are consistent with studies by Richardson (1990), who found that students in physiology courses favoured teleological responses over mechanistic ones. Arnaudin and Mintzes (1985) found that students referred to function in the circulatory system using vitalistic expressions. Carey's research with young children suggests that they possess a "naive-psychology" theory of biology. These findings lend some credence to the speculative proposal that there is analog in the biological sciences to the phenomenological primitives, identified by diSessa (1983; in press) in understanding the mechanisms of physical causality. An understanding of

biological function may be rooted in one's earliest experiences of fulfilling biological needs. For example, the act of eating in response to or in anticipation of hunger, may form the basis of the minimal abstractions from experience, that constitute a naive theory of biology.

A teleological mode of thinking is firmly established in productive and functional modes of thought. Subjects in the experiment would often correctly reason about an outcome to a problem using teleological reasoning. However, this mode of reasoning would just as often lead to erroneous explanations. Subjects would tend to resort to this kind of reasoning, either when they could not construct a mechanistic account, or when they lacked a more precise terminology (e.g., the amount of blood pushed out by the heart on a beat instead of stroke volume). This is likely a common source of a reductive bias that results in simplified mental models of a complex system (Feltovich, et al, in press). As is the case given in physics, through instruction and formal learning, p-prims get supplanted in many contexts by more complex explicit knowledge structures which include physiological laws (diSessa, in press). However, this pattern of thinking may continue to exert substantial influence, and more expert subjects may resort to this kind of reasoning when faced with difficult problems. There was some evidence to support this contention.

Understanding Pressure-Flow and Pressure-Volume Relationships

There were several basic questions related to pressure gradients. In general, subjects demonstrated correct conceptual models of the pressures in the different segments of the circulatory system, although only a few subjects identified mean systemic pressure as the forward pressure for venous return. There were also two questions examining subjects' understanding of

pressure-flow relationships under various changing conditions. One described a situation, which characterized a Starling resistor (also known as a vascular waterfall), in which a vessel collapses and opens according to the pressure difference between the surrounding and inflow pressures. More than half the subjects did not understand this concept.

The premedical student exhibited a fundamental misconception related to pressure-gradients. He clearly believed that any increase in pressure, whether it is an opposing pressure or a driving pressure, would result in an increase in volume and an increase in flow. Therefore, any of the variables that are suggestive of an increase in tension, resistance, or pressure (e.g., contractility, afterload) were viewed as propagating an increase in volume or in flow. This line of reasoning invariably led to errors in prediction and explanation.

There was a tendency towards increased conceptual understanding with expertise in responding to the questions related to cardiac output, although there were very salient individual differences with respect to particular concepts. The first year students and the premedical students often did not have a clear understanding of the meaning of each concept, although, one first year student (1.1) responded quite accurately relative to her peers.

Understanding Cardiac Function

There were several problematic concepts. The Frank-Starling Law of the heart is one of the fundamental principles of cardiovascular physiology. In general, subjects understood this law reasonably well. However, seven subjects, including every subject up to second year medical school and a fourth year student (4.2), did not recognize or did not remember this

mechanism up to the point that they were provided with a prompt. This was most surprising, since it is one of the most often discussed cardiovascular principles in lectures and in textbooks. In addition, only 2 subjects, including the premedical student and the cardiologist practitioner, explicitly mentioned the physiological limits of cardiac function.

A fourth year student (4.1) experienced considerable difficulty in responding to questions related to afterload. He appeared to understand the concept of afterload in that he could articulate its meaning. However, he made a serious error of analysis. The subject relied extensively on the pressure-volume loop diagram, which was presented to subjects as a memory aid, to calibrate his judgments of the effects of the determinants of cardiac output. He repeatedly failed to retrieve correct correspondences between the graph features and the functional relationship. The representation generated from the graph suggested that afterload has no effect on stroke volume. This single inference resulted in many errors in explanation and prediction.

There were a few questions pertaining to afterload and contractility that demanded that the subject conduct an analysis over more than a single beat of the heart, which necessitated propagating effects of increased pressures through the circulatory system over time. This presented considerable difficulty for most subjects. It appeared that most of the subjects who made errors on these questions, did not attempt to simulate the consequences or run a mental model. Rather, they attempted to retrieve the causal relationships from memory.

One first year student made an interesting error in analysis for determining the effects of heart rate. In evaluating the effects of various heart rates on cardiac output, he plugged in variables into the formula for cardiac output, which is stroke volume times heart rate (CO = SV x HR). He

interpreted this equation as describing a positive linear function, suggesting that there is a one-to-one relationship between any change in heart rate and cardiac output and that no other variables are factored into the equation. The student did not consider that stroke volume could be compromised at very high heart rates. This is what Perkins and Simmons (1988) refer to as *equation cranking*, where one uses a formula in a ritualistic manner because it seems to fit, and it returns a value.

Understanding Venous Return

The primary determinants of venous return were not well understood by many of the subjects, including a few of the physicians. In particular, mean systemic pressure and stressed volume could not be defined by most subjects. Five of the students suggested it was a kind of average pressure in the system. Two of the physicians appeared to interpret the term as being synonymous with mean arterial pressure. The term *mean* refers to an average and students are aware of many averaged measures. In a clinical context, mean arterial pressure or mean systemic arterial pressure are the more common terms. The term, mean systemic pressure, can be a source of terminological confusion. This is analogous to the *common connotation bias* discussed by Feltovich and colleagues (Feltovich, et al, 1989), although the common usage is in clinical settings, rather than in everyday language.

Every subject understood the concept of compliance. However, six subjects, including both fourth year students and both residents, confounded the effects of venous resistance with compliance. The notion is that since an increase in venous resistance is associated with a decrease in compliance, then the net effect of resistance would be to increase venous return. This reasoning is counterintuitive and is almost certainly a result of formal

learning, rather than acquired from daily experience. It is similar to a particular pattern of misunderstanding, which Perkins and Simmons (1988) refer to as "Gordian". The Gordian pattern occurs when advanced students or experts elaborate a theory that has serious undetected errors. The error causes individuals to miss or ignore anomalies and results in imprecise or distorted conclusions. Several physiology textbooks fail to make this distinction explicit. For example, Berne and Levy (1990), in their widely used textbook *Physiology*, describe a conceptual model of the venous system that can be interpreted as supporting this misconception.

Themes of Integration

There were several sources of errors evident in subjects' responses to the integration questions. There were a number of students, most notably, a fourth year student, who suggested that arterial resistance and venous resistance interact, when in fact, there is a dissociation between the two variables. Changes in one of these variables does not have any direct effect on the other. This resulted in a number of erroneous explanations. The second source of misunderstanding pertained to the role of right atrial pressure as a coupling mechanism for venous return. Few subjects appreciated the multi-faceted function of right atrial pressure. In fact, no subjects indicated that cardiac function affects the gradient for venous return by altering right atrial pressure.

A third source of misunderstanding in the integration questions was related to the physiological limit of the heart to distend in response to increases in volume. In general, subjects followed one of three models. The first model correctly predicts that cardiac output reaches a plateau at which point there will be no further increases in cardiac output. This model was

consistent with the responses of the three experts and three students. The second model, as expressed in the responses of five subjects, including a resident (R1), three first year students, and the premedical student, indicates that cardiac output would keep increasing linearly, and that there was no upper boundary. The third model, as suggested by the responses of four subjects, including a resident (R2), a fourth year student (4.2), a third year student (3.2) and second year student (2.1), predicted that cardiac output would increase until a point at which it will go into failure. Two subjects, a fourth year student (4.2) and a resident (R2) expressed this belief in response to several questions. This misconception is consistent with the 'mechanical overstretching' misconception documented by Feltovich and colleagues (1989). It is probable that this misconception, the belief that the heart begins to fail at higher preloads, is also intertwined with a lack of understanding of the coupling of venous return and cardiac output.

Diagramatic Representations

The integration of venous return is usually taught in conjunction with a diagrammatic representation of the intersection between the venous return and cardiac function curves. Only the expert subjects used the curves in their analyses of the problems. It is most surprising that none of the second or third year students used the curve as a basis for explaining the role of right atrial pressure because these subjects had most recently received instruction in this subject.

Graphs, and more generally diagrams, offer unique representational advantages (Larkin & Simon, 1987), but they also present particular representational difficulties for the student trying to understand a scientific concept or a mathematical relationship (Leinhardt, Zaslavsky, & Stein 1990).

An appropriate diagram can present perceptually enhanced data structures that result in a minimization of search, and an explicit representation of information, that would otherwise require extensive computation from a sentential representation (Larkin & Simon, 1987). Diagrams are extensively used in diverse domains of science as necessary adjuncts for problem-solving, including the domain of cardiovascular physiology, and are presented to students as exercise problems in textbooks. However, there is rarely any information given on how to draw or interpret diagrams (Chi, Bassok, Lewis, Reimann & Glaser, 1989). Novice problem-solvers experience considerable difficulties in working with diagrams. They frequently extract less information and less accurate information (Lesgold, et al, 1988), and have trouble using their diagrams to support problem-solving inferences (Katz & Anzai, 1990; Anzai & Patel, 1992).

Coherence and Consistency of Mental Models

Functional dependency diagrams were used to characterized subjects' understanding of sets of (partial) functional relationships or components of functional relationships that hold between the variables that were considered in the study. This method of analysis permitted the placement of subjects' individual misconceptions or errors in analysis in the context of their mental model of the circulatory system. The analysis suggested that a salient error, such as the belief that afterload does not affect cardiac output, reverberates throughout the model and can produce a consistent and reliable pattern of errors. This is most apparent when a model is otherwise coherent and consistent. Perhaps, if a subject's model was fragmentary, an isolated error would have minimal consequences. The mental models developed by subjects were generally internally consistent. Misconceptions, at more advanced

levels of training, become firmly entrenched in one's network of knowledge and are supported by elaborate justifications.

It was hypothesized that misconceptions would arise in problems of greater complexity, when memorized knowledge would be insufficient and the subject would have to engage in reasoning that would challenge the robustness of one's understanding. This hypothesis proved to be largely incorrect. Misconceptions emerged in the most basic and elementary physiology questions. In some cases, these misconceptions carried over into more complex problems, and in other instances, they appeared to be overridden by other kinds of knowledge. This suggests that errors in fundamental core concepts can have potential consequences for clinical reasoning.

Applied Problems and Structure-Function Relationships

It was proposed that students should respond better to the basic physiology section than to the more applied problems. Physicians would be more likely to respond more accurately to the applied clinical problems. In general, all subjects responded with greater accuracy and with more coherent explanations to the applied problems. The physicians and advanced students provided superior responses to the problems that required clinical knowledge. They more readily identified the conditions of application for the pertinent concepts. The physiologist experienced considerable difficulty in responding to the problems that necessitated the use of clinical knowledge. This subject has tuned his knowledge to address analytic problems in physiological research rather than in clinical medicine.

The majority of problems presented situations in which there was an obvious disturbance in structure or in function. Each of the subjects had a

well-developed representational model of the anatomy of the circulatory system and could exploit this knowledge to respond accurately to these problems. For example, most subjects could readily identify the hemodynamic effects of a tumor in the liver or explain how the equilibrium is restored between the output of the two ventricles in acute left ventricular failure.

In our previous research, investigating students' understanding of the cardiopulmonary system, we documented the difficulty that students had in coordinating structure and function to explain a problem produced by an embolism (Patel, Kaufman & Magder, 1991). Feltovich has documented similar difficulties in students' representations of the subcellular structures in the cardiac muscle (Feltovich, et al, 1989). These two studies both investigated subject domains that required a vertical integration of multi-level structures from subcellular to systems level to patient problems. The subject matter of the research reported in this study was largely in terms of systems-level physiology. It is probable that students' possess superior representations at this level of abstraction relative to their representations at more fine-grained levels of analysis. It is also possible that vertical integration between levels is more demanding. It also seems likely, that structure-function correspondences are easier to comprehend and reason about than are the abstract physiological processes related to the regulation of cardiac output. This would lend some support to the argument put forth by Chi and colleagues (Chi, 1992; Chi Chiu, & de Leeuw, 1990), suggesting that structure-function relationships are easier to learn than are abstract constraint-based relationships.

Specific problems presented situations that asked subjects to identify the source or sources which can cause a particular change in cardiac output.

In particular, a problem related to extreme exercise and another asking about the hemodynamic effects of hyperthyroidism, presented subjects with an opportunity to apply concepts pertinent to cardiac output and venous return. The results indicated a cardiocentric bias, in that subjects predominantly focused on concepts related to cardiac output. The effect was most pronounced in the hyperthyroidism problem. Subjects were generally able to provide a better than adequate explanation of the effects of extreme exercise. However, no subjects were able to develop an adequate explanation of the hyperthyroidism problem, and only three of the subjects made any reference to any of the venous return factors. This would suggest that even expert subjects experience difficulty applying familiar concepts, such as those related to hemodynamics, in unfamiliar contexts (i.e., an endocrinological problem).

Spontaneous Analogies

There has been increasing recognition of the central role of analogy in cognition (Vosniadou & Ortony, 1989). Analogies are ubiquitous in problem-solving (Holyoak, 1985), in explanation (Gentner, 1989), and in instruction (Spiro, Feltovich, Coulson, & Anderson, 1989). Analogies can be a most effective means for using prior knowledge to understand and integrate new concepts. However, the improper use of analogies can result in the development of significant misconceptions (Spiro et al, 1989).

Most subjects in the study used analogies at one time or another to explain complex phenomena. Analogies served different purposes for different groups and for different subjects. Sometimes the analogies resulted in accurate explanations and predictions and at other times they resulted in errors in judgment.

The most novice subjects, including first year students and the premedical students employed analogies as a means of making sense of concepts that were relatively new to them. As predicted, the best analogical matches were made between analogs that shared both surface and structural similarities. This finding is consistent with results of Holyoak and Koh (1987) and Bassok and Holyoak (1989). One of the best examples of this was the analogy produced by a first year medical student (1.1) in response to a question related to the effects of an increase in venous capacitance and cardiac output. After first incorrectly predicting that this would produce an increase in cardiac output, she reminded herself of the physics definition of capacitors, as a device for storing electric charge. If there is an increase in the storage of potential energy or blood volume is conserved in one location, then less will be transmitted. The analogy led the subject to reconsider and correct her initial response. The analogy was successful because the source domain preserved sufficient literal and structural information.

The two other first year students had studied physics and one student (1.2) in particular, generated many analogies from the physical sciences. He was consistently unsuccessful in retrieving and mapping analogies from a physical science source domain. In an effort to explain the length-tension relationship as it relates to the Frank-Starling mechanism, he selected a source from the wrong phenomenological category. He understood the physical principle governing length-tension relationships. However, the source analog was "objects that bend", such as fiber or metal rather than "objects that stretch", such as a balloon or a garden hose and his analysis failed to yield a plausible explanation. The same student consistently drew analogies from the physical domain of kinematics, rather than statics, in which pressure-volume relationships are subsumed under or dynamics which

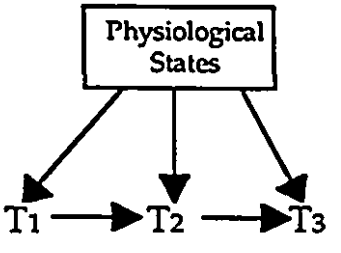
characterize pressure-flow relationships. In these analogies neither the surface similarities nor the structural similarities mapped from the source domain to the target. This situation is perhaps, not uncharacteristic of someone learning about a complex new topic for the very first time in which here or she has only a crude representation of the target domain. Therefore, the selection of a source domain can be exceedingly difficult.

A third year student (3.1), with an undergraduate degree in engineering, very effectively used analogies from the domains of statics and dynamics to explain complex problems. On several occasions, she was confronted with a description in which she understood but could not clearly articulate an explanation. In these circumstances, the subject developed an analogy to a commonplace object that exemplified the principle under consideration. For example, she used the analogy of a water hose to explain how the equilibrium would be restored when venous return temporarily exceeded cardiac output. The hose is an appropriate analog to blood vessels because it is a vessel that carries fluid from a high pressure source to a low pressure area and it is distensible. Rigid tubes are not distensible, but are commonly used to illustrate principles of pressure-flow relationships in the circulatory system (Spiro, et al, 1989). On another occasion, she explained the concept of vascular resistance, and specifically the viscosity of fluids by comparing the process of sucking molasses from a straw as opposed to sucking milk from a straw. In both cases, the analogies were generated from a formal principle, which involves recognizing a situation in which a principle or equation may apply, and retrieving an analogous example of that principle (Clement, 1988).

The physicians in the study, including both residents and cardiologists, and the fourth year students generated clinical analogies to explain various

phenomena. Exemplars were generated via association because they instantiated a particular causal relationship or a particular principle. The success of the analogy depended on the goodness of fit for the mapping. For example, the cardiologist practitioner, in response to a question that asked how a decrease in aortic pressure would affect stroke volume, accessed an analogy related to a common therapeutic practice. Vasodilators are used to reduce aortic pressure in a patient with a low cardiac output. In this case, the causal relationships were a good fit.

In some instances, clinical analogies were inappropriately used because the target domain was misrepresented. In other instances, clinical analogies were produced that failed to map onto the physiological situation because variable slots present in the target domain were missing from the source domain (Spiro, et al, 1989). For example, a resident (R2) attempted to determine the effect of an increase in afterload on end-systolic volume, on a single beat of the heart, by thinking in terms of a common clinical condition, aortic stenosis. In his analysis, aortic stenosis produces an increase in afterload which leads to a hypercontractile state and thus reduces end-systolic volume. The graphic presented below helps illustrates the nature of the problem.



In reasoning about physiological states, one needs a more fine-grained time-line. The question asks about the effects of afterload on a single beat of the

heart. If T1 represents the end-systolic volume on the prior beat of the heart and T2 represents the end-systolic volume following an increase in afterload, then it is this physiological state in which we are interested. Afterload reduces stroke volume and there is a greater end-systolic volume. On the subsequent beat(s) of the heart, compensatory mechanisms come into effect and the net effect maybe a return to baseline or even a decrease in end-systolic volume. The problem with this analogy is that clinical inferences have a *coarser* time dimension, that encompasses the effect of the initial stimulus plus compensatory mechanisms and other systemic responses. It therefore becomes difficult to disambiguate the primary effect from secondary and tertiary effects.

Analogies were used effectively in responding to the applied problems. Subjects used clinical exemplars to illustrate the mechanisms underlying a particular medical condition. For example, a resident (R1) explained how a patient could have a normal cardiac output with congestive failure, by referring to how an infarct affecting the left ventricle could cause congestive heart failure and yet produce a pathophysiological state commensurate with a normal cardiac output. In this case, the target and source domains are in closer proximity and exemplars can be used successfully.

Progressions of Mental Models

As discussed previously, there was a kind of progression of mental models as a function of expertise. As would be expected, the physiologist could respond with considerable facility to the basic physiology questions and had great difficulty explaining the situated problems. There were clear differences in conceptual understanding among the experts. The two cardiologists responded very differently to the various questions. In the more

complex questions, the practitioner tended to focus on a single possible explanation, while the academic cardiologist was able to generate several possible alternatives and identify delimiting factors that could produce different results. The practitioner found the basic physiology questions to be quite laborious, and though he was able to correctly predict most of the correct outcomes, he had difficulty deducing particular relationships. The academic cardiologist clearly possessed a more differentiated conceptual model of the cardiovascular and circulatory system. This may be a function of his research activities, where he would have had to reason about the system in a more analytic and direct way than would the practitioner.

With the possible exception of the physiologist and the academic cardiologist, there were specific flaws in the mental models of each of the subjects. Although, these two subjects made errors, and the physiologist had difficulty with the clinical problems, there were no obvious conceptual flaws. In any rich and complex domain involving intricate causal systems, even experts are likely to have deficiencies in their knowledge at a certain level of detail. The use of simplified and incomplete mental models is ubiquitous in human cognition (Norman, 1983).

I would like to briefly reconsider two models from the domain of medical AI and evaluate their implications for characterizing mental models in the context of medical cognition (Chandrasakeran, et al, 1989). The first model is reflected in the retrieval of stored causal knowledge, which can be accessed and used to resolve ambiguities in the presentation of a clinical problem. The second model includes an explicit representation of structure, function, and behaviour. A physician could use this mental model to account for a patient's condition by running the model and envisioning the consequences in terms of different behavioural outcomes. These models, in

some sense, resemble two of the sequence of models proposed by Forbus and Gentner (1986) for learning physical domains. The first model, the *causal corpus*, consists of sets of causal connections between variables and is analogous to the stored-compiled knowledge model. The second model is referred to as the *naive physics* stage in which the disparate local connections of the causal corpus are replaced with qualitative models organized around the notion of process, which resembles the structure-function model.

It would be interesting to speculate on the goodness of fit between the two models of causal understanding and the observed data in this study. We hypothesized that students entering medical school would be expected to have preconceptions or naive theories about the structure and function of the heart. This hypothesis was generally not confirmed. Every subject appeared to have a better than adequate representation of the anatomy of the circulatory system, which they could exploit to construct mechanistic explanations. It appeared that subjects had access to both stored compiled causal knowledge and a qualitative model embodying structure-function relationships.

The use of stored causal knowledge is computationally more tractable. When causal relationships could be easily retrieved from memory, they would be used. In certain cases, subjects had learned the cause and effect relationship but did not appear to be able to place this knowledge in a broader systemic context. This was, at times, characteristic of second and third year students who had knowledge of a particular causal relationship, which they retrieved to correctly predict an outcome, but could not generate any explanation to account for the systems' behaviour. The use of analogies also provided a means, particularly for the more advanced subjects, to retrieve a piece of causal knowledge from memory without engaging in any

complex analysis. However, when the situation dictated the need to construct a model of an anomalous situation, such as the normal cardiac output during congestive heart failure or the extreme disequilibrium evident in the acute left ventricular failure problem, subjects were able to do so.

There appeared to be costs and benefits to the use of the these two models. The use of compiled stored causal knowledge can be accessed easily and can be used effectively when the causal relationships are well understood. However, this approach is associated with reductive biases and has limited utility in novel or anomalous situations (Feltovich et al, 1989). The qualitative model approach, which incorporates an explicit representation of structure-function relationships, is very powerful model for explaining anomalous situations. However, the analysis entailed by running such a model is very demanding. In addition, any flaw in the model, such as a belief that venous resistance can have a direct effect on arterial resistance, is most likely to produce a recurrent patter of errors.

This description is consistent with a characterization of expertise offered by Smith, diSessa and Roschelle (in press), that suggests that expert knowledge exists in an emergent form in novices, and novice-like models tend to be reused even in subjects at more advanced levels of training. The progression of mental models may be one of refining, tuning, and elaborating one's existing models. The development of expertise may also involve recognizing anomalies in existing models and developing 'patches' that make explaining domain problems more tractable (Patel & Groen, 1992).

Limitations to the Study

There are a number of limitations to this research. The most apparent one is related to the size and restrictions of the sample which limit the

generality of the conclusions. The students were all selected from the same school and had received the same training by the same instructor in cardiovascular physiology. In addition, we deliberately excluded subjects with degrees in physiology, because we believed that they would not be representative of most medical students, in that they would have received more training in this subject matter. In a given year, physiology students comprise between 15-20% of students entering medical school at McGill university. Ideally, we would have had a matched sample of physiology students, but this was not viable. In addition, 30% of the medical students enter McGill medical school from college, without an undergraduate degree. They were also excluded from the study. This would necessarily limit the scope and generality of the conclusions.

There were also limitation in terms of the content of the study. In particular, the applied problems may have not been of sufficient complexity to test subject's understanding of the pertinent concepts. The choices made between the depth and breadth of the material may also limit the scope of the conclusions. For instances, certain topics such as the regulatory effects of baroreceptors and the skeletal muscle pump have a significant influence in determining the flow of blood and were not included in the study. On other hand, important topics, such as the Frank-Starling mechanism could have been explored in greater detail.

In any complex domain, there are controversies related to certain fundamentals. For example, the relative contribution of venous return to cardiac output is still the subject of some debate (Compare Berne & Levy, 1990 and Green, 1987). The kinds of analysis we engage in, to some extent, forces us to take sides in ongoing debates, and necessarily prejudice our conclusions.

Future Research

This research suggests several possible extensions. It is advantageous to extend the generality of the study. One possible extension of this research would be to develop a computerized microworld, which simulates cardiac output and its regulation. This would provide us with a basis for experimentally testing the predictions that subjects would make, as changes are introduced into the environment (e.g., an increase in afterload). The results could be used to test and extend the validity of the findings, since we could obtain dense behavioural measures in a well controlled setting. The findings could be useful in the development of a computerized learning environment. A promising approach for such learning environments is suggested by the notion of progressions of qualitative models (White & Frederiksen, 1987). In this type of microworld, students are presented with alternative models that represent the system from different but coordinated perspectives, such as at the macroscopic and microscopic level.

This study did not directly address the process of learning. There are several learning paradigms, such as learning via self-explanation (Chi et al, 1989) or learning by doing (Anzai & Yokohama, 1984; Anzai & Patel, 1992) that could provide a vehicle for investigating how students learn about concepts related to cardiovascular physiology. This would also suggest ways in which the transfer of knowledge and conceptual understanding can be promoted.

This research hinted at the difficulty students have in using diagrammatic forms of representation in understanding concepts in this domain. Cardiovascular physiology and other medical textbooks are filled with graphs, diagrams, and various schematic representations. How subjects

use these visual representations and what aspects of these diagrams present difficulties for students are issues that need to be addressed.

The theoretical and methodological framework developed for this study could be extended to other biomedical domains and to other scientific disciplines. This would contribute to a general theory of conceptual understanding.

Finally, our belief is that this research has practical implications for the practice of medicine. It would be worthwhile to explore the extent to which physicians' and students' mental models of circulatory physiology affect the decisions and choices they make in diagnostic and therapeutic contexts. For example, decisions made in fluid management and in the delivery of inatropic agents (i.e., contractility drugs) may be affected by an individual's understanding of cardiac output and venous return.

Implications for Instruction

As discussed in the first chapter, there are at least three fundamental and interrelated issues pertaining to the role of basic science knowledge in medical problem-solving. The first issue is what should students and physicians understand about significant biomedical concepts. The second concern is what correspondences or points of intersection are there between these concepts and clinical knowledge. The third related issue is what is the functional utility of this biomedical knowledge in clinical practice. These issues necessitate a theory of competence (Gelman & Greeno, 1989). The first issue, which pertains to the research presented in this document, is one of conceptual competence. Conceptual competence refers to the implicit understanding of general principles of the domain. The subsequent two issues relate to utilizational competence. As discussed previously,

utilizational competence is the understanding of relations between features of a task setting and the requirements of performance. Utilizational competence provides the interpretive knowledge to assess a situation and indicates the relationship between the actions of a procedure and the domain principles (Greeno, Riley, & Heller, 1984).

According to Glaser and Bassok (1989), a theory of competence is the first essential component to a theory of instruction. The other two requirements are an analysis of the initial state of the learner's knowledge and an explication of the transition mechanisms that results in a student's development from the initial state to the desired state of learning. The first two prerequisites are domain-specific. The transition or learning mechanisms would emerge from a general learning theory.

This research is related to the issue of competence, specifically conceptual competence. It has been suggested that clinical knowledge and basic science knowledge constitute two separate domains connected at various discrete points (Patel, Evans, & Groen, 1989b) and that basic science or biomedical knowledge can be arranged in a hierarchical schema of the scientific sources (Blois; 1990; Schaffner; 1986). The ultimate goal then, in developing a model of competence, is to generate a clinically-relevant epistemology of basic science concepts within the context of this hierarchical framework.

It is generally assumed by medical educators, at least implicitly, that the functionality of basic science knowledge will become apparent to medical students once they commence clinical practice. However, the scope of application of basic science principles is not as evident in the practice of medicine, as in the applied physical domains (e.g., engineering). There are also complex correspondences between the different levels of abstraction. It

is possible that students and practitioners cannot experience the same kinds of epistemic challenges to counter their naive intuitions. Consequently, even striking anomalies resulting from fundamental misconceptions and biases can frequently go undetected, and may carry over into clinical practice.

The reform of the medical curriculum to address these issues, is not a trivial task. However, we can begin with the assumption that there are chains in the link that need strengthening. For example, a certain prerequisite knowledge in physics is necessary to adequately understand the domain of hemodynamics. We cannot assume that students have this prerequisite knowledge, nor that they can map this knowledge on to the target domain.

In this research, we have been able to identify and characterize a series of misconceptions, biases, and errors in analysis which represent impediments to conceptual understanding. These can be added to the ever accumulating base of biomedical concepts that present unique and unusual challenges for medical students (Patel, Kaufman, & Magder, 1991, Brumby, 1984; Feltovich et al, 1989; Dawson-Saunders, Feltovich, Coulson, & Steward, 1990). These difficulties can be singled out for special attention.

Basic science instruction focuses on an extensive range of concepts in a rather short period of time (about 18 months). Feltovich and colleagues (Feltovich, Spiro, & Coulson, in press; Spiro, Feltovich, Coulson, & Anderson, 1989) have developed a framework for advanced knowledge acquisition, with particular reference to the domain of medicine. The crux of their argument is that advanced instruction has goals that make unique demands on the design of effective instruction and testing. In introductory learning, the primary goal is to expose subjects to large bodies of knowledge with minimal emphasis on conceptual proficiency. Frequently, the same methods of instruction and

testing are employed in situations of advanced knowledge acquisition. The concomitant effect of this instructional strategy is that students acquire complex concepts in a rote learning or context-dependent manner that precludes true conceptual understanding. This lack of conceptual understanding is manifested in misconceptions, biases, and acquired schemata that are severely limited in their scope of application (Feltovich, et al, in press).

We concur with Feltovich and colleagues that there is a need to prioritize and select particular clusters of concepts, which are of significant generality, and attempt to place more effort into the in-depth teaching of these concepts. Medical schools need to present concepts in diverse contexts and make the relationships between the specific and general aspects, explicit. This also entails striking the right balance between presenting information in applied contexts (e.g., as illustrated by a clinical problem), yet allowing the student to derive the appropriate abstractions and generalizations to further develop their models of conceptual understanding. This would enhance the opportunities for promoting forward-reaching and backward-reaching transfer (Salomon & Perkins, 1989).

In addition, there is a need to address the epistemic frame, which involves the general norms having to do with the grounding of concepts and constraints in a domain (Perkins & Simmons, 1988). Medical students need to appreciate that, rather than something to be endured, biomedical science can provide coherence to explanations and that this can facilitate medical problem-solving (Patel & Groen, 1992).

Invariably, cognitive science and educational researchers call for sweeping curricular reform, with suggestions that are big on generalities, but short on the specifics of implementation. On the basis of the current state of

educational research concerning learning in medicine, we cannot provide a blueprint for medical schools to adapt a comprehensive revision of the format for basic science curricula consistent with cognitive principles of conceptual understanding. Nevertheless, some of the implications discussed can provide guidelines for a kind of curricular change that does not require a major structural overhaul. In addition, medical schools need to make better use of available resources (Patel, Arocha, & Kaufman, in preparation). For example, medical texts are filled with diagrammatic representations that provide a potentially rich source of information that can significantly enhance conceptual understanding. Yet, the evidence suggests that they are greatly underutilized by students.

The development of models of conceptual competence are an essential ingredient for promoting the development of a clinically-relevant basic science curriculum. Although this research is still at an early stage, we believe that we have gained some insight into the elaboration of a model of conceptual understanding for the domain of cardiovascular and circulatory physiology. This work has also contributed to a broader theory of conceptual understanding which has implications for instructional practices in complex domains.

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