CHANGES IN VISUAL FUNCTION WITH PERCEPTUAL ISOLATION

by

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INTRODUCTION: THE EFFECTS OF SENSORY DEPRIVATION ON THE FUNCTIONS OF THE CENTRAL NERVOUS SYSTEM

A disease or injury which impairs or destroys sensation in a peripheral region very often results in some disturbance beyond the mere loss of sensation. For example, a patient who has had an arm or leg amputated may continue to "feel" the absent limb long after it has been removed. Operations for the removal of cataracts are often complicated by visual hallucinations or delirium. A lesion in the visual pathways which immediately impairs only a portion of the field of vision may produce mild hallucinations with altered functioning of the intact visual areas.

These disorders have a common feature, namely, that the central nervous system is being deprived of a former source of the normal and varying patterns of stimulation from the environment. In the past, such disorders have not often been studied together, although separately they have been reviewed at length. A few cross-references between the similar disorders of visual and somesthetic function have recently been made. Brunerie and Coche (1936) and Kujath (1940) made a brief comparison between the phantom limb and the visual hallucinations experienced in blindness or by patients with cataracts. Lunn (1948; cited by Cronholm, 1951) and Bartlet (1951) made similar comparisons and reached the conclusion that absence of normal stimuli from the periphery might be an important factor in the production of hallucinations.

There are probably several reasons why so few references point to the relation between these symptoms. In the first place,

complaints of this kind have come mainly to the attention of medical specialists who have generally looked upon them as secondary complications. Secondly, psychologists who are interested in the physiology of perception have not been guided by theories which would point to the possible significance of sensory deprivation for the function of the central nervous system. Psychiatrists, on the other hand, have usually looked upon such symptoms as unique examples of "dynamic" (emotional) disturbance.

In the following pages evidence will be presented to show that loss of sensation presents a common set of symptoms in vision and somesthesis, and probably in audition as well. Moreover, when these symptoms are studied in the light of recent evidence from neurophysiology, there evolves a theoretical basis for the proposal 1) that the central processes involved in normal perception depend upon continual sensory input of varied pattern, and 2) that absence of variable patterned stimulation may produce hallucinations or other disturbances of perceptual function. An hypothesis to account for these changes will be developed.

A review of these separate perceptual disorders will show how closely they may be related. However, as they comprise a history of discussions of apparently distinct pathological signs, we may begin by reviewing the evidence concerning phantom limbs, going on then to discuss the material concerning visual perception. Finally, neurophysiological evidence will be cited which sheds some light upon the possible neural mechanisms involved in deafferentation or sensory deprivation.

The Phantom Limb

The effects of sensory deprivation on the central nervous system are seen in a variety of somesthetic phenomena occurring with loss of peripheral sensation or lesions in the afferent pathways. The illusory sensations or "phantom limbs" experienced by amputees have been described in detail by Pitres (1897), Bornstein (1949), Cronholm (1951), Hécaen and de Ajuriaguerra (1952) and others. Hécaen and de Ajuriaguerra compiled a list of 1576 amputations and showed that phantom limbs are an almost universal phenomenon, being reported in some form in nearly ninety-three percent of the cases.

According to Ebbecke (1950), all the normal sensations of pain, temperature, and movement may be reported for a limb which is no longer present. With data from three hundred amputees, Henderson and Smyth (1948) classified such sensory phenomena according to three types. These were "mild tinglings" reported in nearly every case, strong "pins and needles" sensations which were most apt to occur with touching of a neuroma in the stump, and thirdly, a wide variety of sensations of touch, motion, or position which the authors thought to be "psychogenic" in origin. Hécaen and de Ajuriaguerra report the apparent shape or position of the missing limb to be quite variable. Often the patient reports that the phantom is in a normal or familiar position, although in some cases it may remain almost permanently flexed. Familiar objects such as rings, bracelets, or clothing may continue to be felt, and so give the patient a compelling sense of the presence of his limb. Shrink-

ing of the apparent size of fingers or toes is common, and may go on to involve the entire phantom limb, leading gradually to its disappearance (Bors, 1951). Leriche (1947) reported that pain was present in some form in as high as seventy-five per cent of phantom limbs, although Kolb (1954) gives a much lower estimate. Time of onset of the phantom sensations is usually immediately following amputation, although in some cases there may be a latent period of several months or even years (Bailey and Moersch, 1941).

The movements attributed to the phantom limb have been described in detail by Lhermitte (1951) and Hécaen and de Ajuriaguerra (1952). Briefly, these consist of voluntary movements in which the patient is able to extend or flex his missing limb, reflexive or synkinetic movements, purely spontaneous movements, and automatic movements which form part of habitual acts. Riese (1932) and others have reported that in some cases the phantom appears only when the patient assumes a particular posture or activates the contralateral limb.

By far the most common cause of phantom sensations is amputation of an arm or leg, although they may be found with loss of a hand or foot alone, or of breast, penis, or nose. Comparable symptoms may occur with localized injuries to the peripheral nerves while the major portion of the limb is unaffected (Riddoch, 1941).

Damage to peripheral nerves or lesions in the afferent pathways may produce alterations or distortions of somesthetic perception somewhat different from the hallucinatory phenomena of the phantom limb. These disturbances, for which Wilson (1927) used the

term dysesthesia, may consist of altered thresholds, changes in the subjective evaluation of pain or stimulus intensity, or changes in the apparent quality of sensation. Teuber, Krieger, and Bender (1949) and Haber (1954) have reported significantly lowered thresholds for light touch, two-point touch discrimination, and point localization in amputation stumps. Lesions of the brachial plexus (Mayer-Gross, 1929; Riddoch, 1941; Lhermitte, 1951) or the lumbosacral plexus (Riddoch, 1941) likewise may produce dysesthesia or phantom sensations.

As might then be expected, spinal cord injury may have the same result (Mayer-Gross, 1929; Böhm, 1936; Lhermitte and Sigwald, 1939; Riddoch, 1941; Becker, 1949; Bors, 1951). Lesions of the brain stem may cause severe disturbances of spatial orientation, with concomitant phantom limb sensations (Lhermitte et al, 1937; Lhermitte 1951; Hécaen and de Ajuriaguerra, 1952). Depending upon the exact site and extent of the lesion, such cases may exhibit far more extensive perceptual disturbances, including pronounced visual hallucinations, which will be discussed later. Head (1920) described in detail the excessive responses to affective stimuli with lesions in the thalamus. These responses, which constitute the so-called "thalamic syndrome", occur in the presence of raised or lowered stimulus thresholds, and appear as exaggerated reactions to normal forms of tactile or thermal stimulation. Cortical lesions may likewise produce phantom sensations (Lhermitte, 1951; Hécaen and de Ajuriaguerra, 1952) including a supernumerary phantom, or "phantom third hand" reported by Critchley (1953) with

parietal lobe injury.

One of the earliest investigations of the phantom limb was made by Weir-Mitchell (cited by Hécaen and de Ajuriaguerra, 1952) following the Civil War in the United States, although knowledge of the phenomenon had existed at least since the time of Paré in 1585 (cited by Pitres, 1897). Weir-Mitchell concluded that the painful phantom sensations which he observed in war veteran amputees were the result of stimulation arising from the stump of the amputated limb. This idea had originated long before, and was held by all of the earlier neurologists. Some have argued much more recently that the phantom sensations, particularly phantom pain, could be attributed wholly to peripheral stimulation (Foerster, 1931b; Leriche, 1947). It will be seen that this view is unsatisfactory, although it is true that painful sensations may disappear with alleviation of a neuroma or inflammation in the scar tissue of the stump (Riddoch, 1941; White, 1944; Henderson and Smyth, 1948; Becker, 1949; Bors, 1951). In some cases electrical, mechanical, or chemical stimulation of the stump ending may enhance the sensations, or elicit them when otherwise absent. (Pitres, 1897; Ebbecke, 1950; Cronholm, 1951). Toxic conditions, hunger, alcohol ingestion, anxiety, excitement, fatigue, drowsiness, or climatic changes may also favor their appearance (Lhermitte and Susic, 1938; Bailey and Moersch, 1941; Hécaen and de Ajuriaguerra, 1952). At the same time, Russell (1949), Russell and Spalding (1950), and Cronholm (1951) have successfully abolished phantom limb pain in some cases with pressure or percussion applied to the stump.

However, it seems that peripheral stimuli coming either from the injured nerve fibres at the site of the lesion, or from irritations in the surrounding tissue, are insufficient to account for the sort of sensation which may appear with phantom limbs. The perception of a ring on an amputated finger, or articles of clothing touching the skin, must of course depend upon learning or past experience. While peripheral excitation may serve as the impetus for such sensations, it is difficult to conceive how anything but pain or bizarre sensations could arise directly from the injured nerves. Furthermore, Livingston (1938) found that pain in conjunction with a neuroma usually has a quality distinct from that which occurs in cases which do not involve peripheral inflammation.

Hughlings Jackson (1889) was among the first to suggest that peripheral stimuli were not necessarily the main cause of phantom limbs. He proposed that the phenomenon was due mainly to spontaneous discharges in the motor areas of the cortex. While the majority of writers since his time have argued for the presence of both central and sensory factors in varying degrees, the overall picture in the literature to the present day shows that little agreement on the mechanisms has been reached.

The evidence presented by Livingston (1938) is typical of the contradictions which have been observed so often. The bizarre qualities of the symptoms, their inexplicable variations in intensity, and the patients' susceptibility to diversion in which they might forget their pain, suggested to him that the pain is of

central origin. It should not, therefore, be amenable to peripheral surgical treatment. Yet he found that injections of procaine hydrochloride near the second to seventh sympathetic ganglia were sometimes successful in alleviating phantom pain. The relief of pain with its associated peripheral vasodilation led him to the conclusion that impulses conveyed by the sympathetic nerves, either afferent or efferent, must contribute to the painful sensations. He has since held to the notion that self-perpetuating circuits are set up by the excessive stimulation of internuncial spinal neurones at the time that the lesion is made. These self-perpetuating circuits may subsequently fire efferent fibres and so produce localized peripheral reactions which give rise to phantom pain (Livingston, 1944).

Hécaen and de Ajuriaguerra (1952) and others have reported a correlation between the incidence of phantom sensations and the proportional area of cortical representation for the affected limb or extremity. It has been found, for example, that the thumb and radial portion of the hand are more frequently involved than the remaining portion, the hand more frequently than the forearm, and the upper limbs more frequently than the lower. Furthermore, the apparent size or shape of the phantom limb may actually correspond to the porportional areas in the cortex. Thus the thumb may seem unusually large, and the arm surprisingly small.

Riese (1950) published a report based on amputations in twenty-four children which, though far from conclusive, suggests that the phantom phenomenon may be related to learning or level of

development. He found that the symptoms did not develop in any case in which amputation occurred before the age of six years. Similar findings were reported by Lunn (1948; cited by Cronholm, 1951). (Absence of other forms of hallucinations in young children has been reported by Despert, 1948). One may disagree with Riese's conclusion that the age of six or seven must be a crucial stage in the development of the body schema (see below); the data suggest, however, that sensory deprivation may cause overt disturbances of perceptual function only if it occurs after a sufficient level of development.

An important historical contribution to the psychological or "functional" interpretation of the phantom limb was made by Head (1920) when he introduced the notion of the "body schema." This term was proposed for the "standard against which all changes of posture are measured before they enter into consciousness", wherein "every recognizable change [in posture] enters into consciousness already charged with its relation to something that has gone before" (page 605). He reasoned that one builds up a postural model which changes constantly with new perceptual data. It was by means of the schema, he said, that one could project the recognition of posture, movement, or locality beyond the limits of the body. The body schema was thus conceived as a form of memory image, to which the phantom was in part attributed. This concept promptly found its way into the literature, where it has been used widely in connection with phantom limbs ever since (Bornstein, 1949; Hécaen and de Ajuriaguerra, 1952). However, little more

success has been met in explaining why the body schema is disturbed by an injury to the sensory pathways, than in explaining the phantom limb in any other terms. Although it may be a useful functional concept, it has been of little use in explaining physiological mechanisms.

In an attempt to distinguish central from peripheral factors many writers have spoken of psychological and physiological mechanisms in phantom limbs. Beyond a point this is a needless if not an unfortunate distinction. So-called psychogenic interpretations have failed to contribute substantially to the problem, while physiological discussions have unfortunately been preoccupied with the role of peripheral determinants. Until recently the possibilities of central physiological processes have been to a great extent neglected.

In recent years several physiological explanations have been given which might account for phantom limbs in the absence of normal stimulus patterns. Lunn (1948; cited by Cronholm, 1951) felt that peripheral irritations might account for pain and dysesthesia, while more complex tactile and kinesthetic sensations must be attributed to central mechanisms. The cause of central hallucinatory processes he considered to be "the loss of the normal, differentiated, afferent impulses from the periphery" (Cronholm, page 53). This situation Lunn found analogous to the occurrence of visual hallucinations in half-light, and of auditory hallucinations in the deaf. Later, Bartlet (1951) reached the same conclusion after a study of visual hallucinations in cataract patients, and offered a

comparison with phantom limbs in amputees.

Hebb (1949) introduced a theory which would attribute phantom limb pain directly to a decrease in sensory activity. With decreased efficiency in the firing of the afferent system, higher somesthetic nuclei in the ventral thalamus would lack their normal degree of sensory control and become hyperactive in response to any intermittent "triggering" from peripheral stimulation. Under these conditions he suggested that there would be a disruption of organization and hypersynchronous firing in regions such as the ventral nucleus of the thalamus, which would disrupt the temporal organization of firing in the somesthetic cortical areas, and so constitute pain.

Ebbecke (1950) suggested that the particular cortical area subserving an amputated limb may be subject to partial "sleep" in the absence of sensory input which normally keeps it aroused. Given this condition, the isolated cortical area might be reawakened, he said, by impulses from the severed nerve roots, these impulses then being integrated into false or hallucinatory perceptions.

Bors (1951) offered an hypothesis similar to that of Livingston (1944) which would place the responsibility for phantom sensations in the spinal cord. He suggested that "artificial" synapses might be formed in the cord at the time of injury. In this way impulses from the proximal portion of the limb might be routed to the sensory areas formerly connected with the severed portion. In addition, reverberating circuits might be formed which, even with diminished peripheral stimulation, might continue to excite these

pathways. He believed his hypothesis to be borne out by the success of cordotomy in the relief of paraplegic phantom pain.

After an extensive and carefully controlled study of phantom limbs in amputees, Cronholm (1951) presented his theories of central neural hyperexcitability and functional reorganization. While Riddoch (1941) and Henderson and Smyth (1948) and others had briefly suggested a state of hyperexcitability during hallucinatory episodes, Cronholm was one of the first to elaborate such a theory. He proposed that following amputation, neuronepools at the spinal, thalamic, or corticallevel might become hypersensitive to all impulses converging upon them. Stimulation via the severed nerve pathways, or other pathways converging upon the hyperexcitable area, might then be sufficient to elicit phantom sensations. However, not all phantom sensations could be explained adequately by hyperexcitability, particularly those having a definite form and locus. These were best explained, he said, by imagining a change in central neural organization. These two conditions--hyperexcitability and central organizational changes--could presumably account for all forms of phantom limb sensations. These theories will be discussed in greater detail in the concluding portion of this review.

Impairment of Visual Function

In the following pages it will be shown that hallucinations or other visual disturbances may result with disease or injury at any level within the visual system. However, little success has been met in explaining these disorders. This may largely be due to the fact that such disturbances usually have been studied in relation to the

anatomical site of accompanying tissue damage, rather than in terms of the functioning of the visual system as a whole.

An early report on the delirium and hallucinatory state experienced by patients hospitalized for the removal of cataracts was published by Sichel (1863). He reported that in at least seven of his patients delirium developed during the second to fourth night after bandaging of the eyes. In each case the patient was past sixty, and eventually made a full recovery. Schmidt-Rimpler (1879) not only encountered delirium with bandaging of the eyes, but also when he placed a patient in a dark room prior to operation. Posey (1900), Kipp (1903) and others have reported similar effects connected with a variety of eye operations.

The onset of cataract delirium is usually marked by irritability, restlessness, and irrationality or incoherence. The typical case develops during the night soon after the operation, and once developed may continue for periods from a few minutes in length up to several days (Brownell, 1917). Often hallucinations occur during the period of delirium, and may or may not be remembered by the patient after recovery (Kipp, 1903; Brownell, 1917; Boyd and Norris, 1941; Linn et al, 1953). There are relatively few reports in which hallucinations have occurred in senile cataract without some apparent delirium (Flournoy, 1923; Bartlet, 1951), although delirium or minor delusions may develop with no report of hallucinations (Kipp, 1903; Linn et al, 1953).

The visual hallucinations which are reported by cataract patients resemble those with various other optic disorders, and are

similar to the so-called hypnagogic hallucinations which some individuals often experience just prior to falling asleep (Maury, 1848; Leroy, 1933; Lhermitte and Sigwald, 1941). Generally they consist of one or many patterns, figures, faces, or wallpaper type designs, and may involve movement, and appear in all colors, (Flournoy, 1923; Bartlet, 1951). They may occupy all or a portion of the visual field, may appear with the eyes open or closed, and vary in the extent to which they are under the subject's control.

Although hallucinations may accompany cataracts in patients of all ages (Butler, 1920), it is generally recognized that the older the patient is, the greater are the chances of disturbance. Accordingly, these symptoms are most often found in cases of senile cataract. At present so little is known about hallucinations and delirium on the one hand, and the physiological changes during senescence on the other, that one cannot be sure why susceptibility to perceptual or cognitive disturbances should increase with senility. Cellular degeneration and neuronophagia in the central nervous system during advanced age (Zubek and Solberg, 1953) may either directly affect the sensory areas by making them more susceptible to sensory deprivation or, through impairment of subcortical pathways, add to the decrease in sensory input. Either of these interpretations would be consistent with the fact that hallucinations sometimes occur in individuals of advanced age without cataracts, and with no other apparent visual defects except for some impairment of visual acuity (de Morsier, 1938; Bartlet, 1951).

Watson (1872) wrote that any disorder which caused the out-

line of objects to become dim, hazy or fuzzy, such as cataract, chronic choroiditis, or opacity of the cornea, was capable of producing "spectral illusions" or hallucinations. Colman (1894) reported temporary periods of visual hallucination in a patient with choroiditis and floating bodies in the vitreous of the eye. He diagnosed other patients with syphilitic retinitis who presented the same symptoms. Uhthoff (1899) reported visual hallucinations in the blind fields of a patient with bilateral choroiditis and central scotoma. He found no marked difference between these and the hallucinations of another patient following enucleation of the eye. Cases similar to these have been reported by Lhermitte and Sigwald (1941) and Lhermitte (1951).

Schröder (1925) and Niessl v. Mayendorf (1936; cited by Agadjanian, 1946) noted that hallucinations frequently occur with hemianopia or scotoma resulting from lesions in the visual pathways. Hallucinations with lesions of the optic tract have been reported by Uhthoff (1899), Pick (1904), and Weinberger and Grant (1940). In sixteen cases reviewed by Weinberger and Grant, hallucinations ranged from simple forms to complex scenes. Lesions in the midbrain, particularly near the dorsal region of the cerebral peduncles and encroaching upon the reticular formation, produce vivid hallucinations, loss of spatial orientation, vertigo, and somnolence (Van Bogaert, 1927; Lhermitte, 1932, 1951). Visual and auditory hallucinations were reported by Jasper and Van Buren (1953) in a stuporous patient with a tumor occupying the midline nuclei of the thalamus. Lesions in the posterior region of the in-

ternal capsule were reported by de Morsier (1938) to produce similar symptoms, except for the pronounced somnolence. A variety of cortical lesions may also be responsible for hallucinations. These have been reviewed by Van Bogaert (1926), Johnson (1933), de Morsier (1938), Wagener (1948), Lhermitte (1951), Russell and Whitty (1955) and others. Relatively simple or crude hallucinations are more often associated with damage to the visual sensory area (area 17), while more complex forms tend to arise with damage to the so-called visual-psychic areas, and also with lesions in the temporal or parietal lobes.

The disturbances which accompany cataract operations have been ascribed to a wide variety of causes. While bandaging of the eyes and confinement to bed are generally recognized as key factors (Jean-Sédan, 1939; Pesme, 1939) various writers have attributed the reactions to homesickness, general senility, accompanying arteriosclerosis or kidney disorders, toxic conditions, trauma to the nervous system, grief, worry, or hereditary predisposition to delirium (Parker, 1913; Brownell, 1917). The paranoid delusions, irrationality and disorientation sometimes encountered have, as a matter of course, been interpreted by some psychiatrists in terms of Freudian theory (Linn et al, 1953).

Lunkiewicz (cited by Rau, 1899) and other early investigators attributed cataract delirium to the toxic effects of atropine which was administered to their patients. While overdoses of atropine may produce hallucinations and mild delirium (Sollman, 1934) this clearly could not account for those cases reported by Rau

(1899) and others since, where no such drug was used. Posey (1900) experimented with the dosage of atropine in his patients, and found no relation between its administration and hallucinatory symptoms.

Schmidt-Rimpler (1879) sought to explain the disturbances in terms of inhibitory processes. Making a bold comparison with a contemporary study of reflexes in deafferented frogs, he argued that normal sensory input, particularly visual sensation, activates certain inhibitory centers. With the reduction of sensory input due to cataracts and subsequent bandaging of the eyes, the inhibition is removed, allowing release of hallucinations and unrestrained behavior.

Brunerie and Coche (1936) in one of the infrequent comparisons with the phantom limb, argued that a sensory disorder must furnish bizarre excitations to the cortex. In optic disorders, they suggested that anything from mistiness to total occlusion of vision provides the retina with abnormal sensations, which in turn create bizarre and variable hallucinations. However, Dejean and Ferrié (1939) concluded that the retina and optic tract are incapable in themselves of producing hallucinations, and spoke of the neurologic importance of the fact that removal of occlusive bandages often reduces the hallucinations.

The proposal made by Bartlet (1951) has already been mentioned in connection with phantom limbs (page 10). With phantom limbs and with cataracts alike, he recognized that there is an absence of normal stimuli from the periphery, and argued that the re-

lease of cortical centers from their normal stimulation is more important than any possible irritation arising at the site of a sensory lesion. He cited Russell (1949) and Russell and Spalding (1950) who reported relief of phantom pain by pressure applied to amputation stumps, in which case abnormal but excessive stimulation abolished hallucinations instead of increasing them. To support his ideas, Bartlet also cited the frequent failure of surgical interruption of the sensory pathways to abolish a phantom limb, and the appearance of visual hallucinations in hemianopic blind fields. Hallucinations under drug intoxication, the scotoma and subjective images in migraine attacks, and the visions in psychotic episodes he interpreted as dissociation of part of the brain from sensory control, allowing the cortical centers to create conscious imagery either autonomously, or in response to abnormal afferent stimuli.

Although no theory to explain cataract delirium has been generally accepted, ophthalmologists have nevertheless established routines of treatment or prevention. Zimmerman (cited by Kipp, 1903), Bruns (1916) and Allen (cited by Boyd and Norris, 1941) have all reported success in averting unfavorable reactions in their patients by uncovering the unaffected eye almost immediately following operation, and allowing the patient to walk about or sit in a chair rather than remain in bed. Kipp (1903) found that bedside company and a change in environment could forestall severe reactions in many cases. In general, these procedures are aimed at soothing the patient, reducing the amount of confinement, and producing novelty or diversion.

Weinberger and Grant (1940) attached particular significance to the fact that in sixteen cases with verified lesions of the optic tract they collected reports of a full variety of hallucinations, ranging from simple forms to complex scenes. Since the work of Foerster (1931a) in recording the sensations evoked with electrical stimulation of Brodmann's areas 17, 18, and 19, many writers had assumed that the form taken by visual hallucinations is indicative of disturbance within a given part of the visual cortex--simple patterns of light representing disturbances in the striate area (area 17), and more complex images arising in the so-called visual-psychic areas (areas 18 and 19). Here, however, were cases where the same sub-cortical lesions were capable of producing any degree of complexity in the images. The danger in attempting to localize the source of irritation on the basis of the form of hallucinations has also been pointed out by Duke-Elder (1949).

But Weinberger and Grant had a second criticism against the use of hallucinations in localizing a supposed source of irritation. With hemianopia caused by optic tract lesions, hallucinations occurred sometimes in the blind area and sometimes in the intact visual field, as was also found in a few cases reported by de Morsier (1938) and Lhermitte (1951). Because hemianopic hallucinations usually occur in the blind field (de Schweinitz, 1891; Peterson, 1890, 1891; Burr, 1906; Ormond, 1925; Engerth and Hoff, 1929) it was assumed that when they occupied a specific portion of the visual field in cases without blindness, the source of irritation must still be somewhere within the point-to-point projections

from that field. In citing hallucinations outside of the objectively blind field, Weinberger and Grant argued that lesions limited to one portion of the visual field could cause global disturbances of the entire visual system.

A separate explanation was given by Van Bogaert (1927) and Lhermitte (1932, 1951) for the symptoms which they observed in patients with lesions in the region of the cerebral peduncles and the reticular formation--the condition which they termed l'hallucinose pédonculaire. The vivid visual hallucinations, somnolence, vertigo and loss of spatial orientation they interpreted as a dissociation of the sleep function from the normal bodily "vegetative" functions--a "general hallucinatory state" similar to that described by Goldstein (1908). It was suggested by Zalkind (cited by Agadjanian, 1946) that a more accurate term for this condition would be l'hallucinose mésencéphalique.

De Morsier (1938) felt that a variety of hallucinations, particularly those resulting from thalamic lesions, might be explained on the basis of an involvement of connections between the pulvinar and the visual areas of the cortex.

According to de Morsier (1938) visual hallucinations are usually preceded or accompanied by a variety of defects or distortions of visual perception which are generally not noticed because of the preoccupation of patient and experimenter with hallucinations. Unfortunately he did not present evidence to show whether particular disturbances are usually found with hallucinations of one or another specific cause. Preceding almost all hallucinatory episodes,

he said, there is a reduction of visual acuity in which the patient may notice a grey or white mistiness in a portion of the visual field (amblyopia or positive scotoma). One also finds changes in the apparent size of perceived objects, whereby they may look unusually small (micropsia) or large (macropsia). Objects may appear displaced in space or distorted with respect to the horizontal or vertical. The perception of depth may be affected, flat surfaces may appear warped with concave or convex curvature, and there may be rapid changes in the form or position of objects (metamorphopsia). Corresponding abnormalities frequently appear in the subjects' hallucinations (de Morsier, 1938; Lhermitte, 1951). Lachmund (1904), Van Bogaert (1934) and Morel (1937) have reported these disturbances with cerebral lesions, epilepsy, and alcoholism.

Lesions in the visual pathways may produce threshold changes, metamorphopsia, or similar disturbances within the intact portion of the visual field without hallucinations. Lohmann (1913) reviewed several cases of metamorphopsia and disturbances of adaptation in the intact visual field with hemianopia due to lesions of the optic tract. Similar defects occurring outside of objectively blind areas of the visual field have been discussed by Fuchs (1920), Teuber and Bender (1949), and Bay (1953). In general these may be said to resemble the tactile dysesthesia or threshold changes with lesions of the somesthetic pathways (Wilson, 1927; Teuber, Krieger, and Bender, 1949; Haber, 1954)

It is clear that hallucinations and other disturbances may result with an interruption anywhere in the visual pathways. The

same was found to be true in the case of phantom limbs and tactile disturbances which develop with lesions at all levels in the somesthetic pathways. Similar conditions apparently exist within the auditory system. For example, Colman (1894) reported auditory hallucinations brought on by obstruction of the external auditory meatus by a foreign body. Redlich and Kaufmann (1896) examined eighty-one cases of auditory hallucinations and found defects in the auditory organ in sixty-five per cent of the cases. Goldstein (1908), Klieneberger (1912), Rhein (1913), Berggren (1929), Semrad (1938), de Morsier (1938) and Agadjanian (1946) have reported auditory hallucinations with a variety of causes, including middle ear infections, lesions in the auditory nerve, and lesions within the auditory cortex.

In spite of the foregoing evidence, writers have generally overlooked the possibility which Lunn (1948; cited by Cronholm, 1951) and Bartlet (1951) have seen, that deprivation of stimulus input to the higher centres might be a primary and general cause of hallucinations in any sense modality. Recently, strong evidence in support of this hypothesis was found by Bexton, Heron, and Scott (1954) and Scott (1954), in a study of the intellectual and motivational effects of drastically reduced variation in the sensory environment. In their studies, college students were paid to lie on a bed for several days in an enclosed cubicle, under conditions which severely limited visual, auditory, and somesthetic stimulation. During this period a translucent mask was worn over the eyes, allowing the subject to see only a misty field of light, as

might be encountered in severe bilateral cataracts. The majority of subjects, after spending a day or more in these conditions, reported visual hallucinations, and in some cases there were somesthetic disturbances. These findings will be discussed in detail with the results of the present investigation. They showed conclusively, perhaps for the first time, that in the absence of drugs or direct interference with the sensory pathways, perceptual deficit may produce well-developed hallucinatory activity in the healthy, waking individual.

Physiological Mechanisms

Unfortunately there is little direct physiological evidence concerning the nature of the effects of prolonged stimulus deficit on perceptual function. Some relevant evidence may be found in clinical and experimental data from human and animal brain lesions, which will be discussed here to clarify the symptoms which accompany hallucinations in certain cases, and to point to the fact that there are two separate sources of disturbance to be considered in cases of sensory deprivation.

Isolation of the cortex from a major portion of its sensory input was achieved by Bremer (1935) in his well known studies of the cerveau isolé preparation in the cat. Transection of the brain stem immediately anterior to the pons, effectively severing all but the olfactory and optic pathways to the cortex, immediately produced a behavioral state comparable to deep sleep or barbiturate anesthesia. The electrical activity of the brain in such a preparation was also comparable to that in barbiturate anesthesia, consisting of waves of

10 to 15 cycles per second (compared with a normal frequency of 25 to 30 cycles per second), and marked by periodic waxing and waning of amplitude. Deafferentation of the visual cortex in the cat, with sectioning of the optic radiations (Bremer, 1938) eliminated the normal rhythmic electrical activity of that area. Application of strychnine to the deafferented region essentially restored the original patterns of activity. With these observations Bremer developed his notion of the tonus cortical: a level of reactivity of the cortical areas correlated with the level of sleep or wakefulness. A depression of this reactivity and the onset of sleep were thought to be due to absence of supporting afferent stimulation.

Later observations made a revision of this hypothesis necessary. The sleep state and depressed tonus cortical were seen not as the result of deafferentation per se, but rather as resulting from a diminution of "dynamogenic" stimulation of the cortex from the brain stem reticular formation (Lindsley et al, 1949; Bremer, 1954). While spontaneous electrical activity has been reported in the totally isolated cortex (Kristiansen and Courtois, 1949), wakefulness and facilitation of the cortical integration of specific sensory input are now assumed to depend upon the presence of non-specific stimulation from the brain stem reticular formation (Morison and Dempsey, 1942; Magoun, 1954; Jasper, 1954). Brain stem lesions of the sensory pathways in the cat, sparing the reticular formation, do not produce the somnolence observed in the cerveau isolé preparation (Lindsley et al, 1950).

Clinical studies reported by Jasper and Van Buren (1953) have shown that interruption of the afferent pathways in the brain stem interferes with the normal patterns of electrical activity in the cortex, producing abnormal slow wave discharges. Electroencephalographic changes are relatively minor in cases of lesions of the specific pathways, when compared with lesions affecting the non-specific projections from the brain stem. Patients with lesions of the reticular formation or non-specific pathways usually exhibit drowsiness or pronounced somnolence.

These findings would explain the condition of the patients described by Van Bogaert (1927) and Lhermitte (1932, 1951) with lesions invading the reticular formation in the region of the cerebral peduncles—the condition which these authors termed l'hallucinose pédonculaire (page 20). However, the evidence does not favor their interpretation of a "general hallucinatory state" representing a dissociation of sleep function from the normal bodily processes. Hallucinations and an exaggeration of sleep may both have a common cause in such cases, but there is no reason to assume that hallucinations are produced by a disturbance of the sleep processes.

The chief value of electroencephalographic data and observations from brain lesions, as far as the present topic is concerned, has been to show that two separate functional systems may be involved in cases of deafferentation or sensory deprivation, depending upon the site of interference in the afferent system. Accordingly, it may be necessary in some instances to consider two kinds of

effects, namely, deprivation of the specific sensory projection systems, or deprivation affecting the diffuse reticular or thalamocortical projections. Apart from emphasizing the difference between these effects, there are few electrophysiological data which are helpful in suggesting the more precise mechanisms involved in these disturbances.

Brief mention was made, in the discussion of phantom limbs, of Cronholm's (1951) theories of hyperexcitability and central neural organization. These were the result of a series of experiments to determine the basis of referred phantom sensations elicited by stimuli applied to the patient's body. Cronholm assumed that such sensations must result from a change in the integration of neural impulses. Previous writers (Livingston, 1944; Sunderland and Kelly, 1948) had suggested that abnormal spontaneous activity in the spinal cord might account for phantom pain or the painful sequelae to peripheral nerve injuries. Others (Riddoch, 1941; Henderson and Smyth, 1948) had suggested that phantom sensations were accompanied by increased activity in the sensory cortex. Expanding their suggestions, Cronholm proposed that a central state of hyperexcitability might develop at any level within the central nervous system following amputation. However, he did not adopt any definite hypothesis concerning the pathophysiology of hyperexcitability, nor did his use of the term hyperexcitability carry specific implications concerning neurone excitability thresholds.

Cronholm observed that a stimulus applied to the amputation stump or elsewhere on the body would often be correctly ident-

ified and localized by a patient, while at the same time it evoked unfamiliar and sometimes painful sensations referred to an amputated limb. These referred sensations were often intense, and diffuse in localization. To account for this phenomenon he suggested that spread of excitation from the impulses evoked by the stimulus caused activity in excitable neurone pools within the spinal cord, or at the thalamic or cortical level. He assumed that hyperexcitability would be most pronounced in neurones which were most active normally with stimulation of the intact limb.

Some properties of the referred phantom sensations could not be explained by hyperexcitability alone. This was so in the case of definitive sensations which had a specific locus within the phantom limb. Such cases Cronholm explained in terms of a "central state of functional organization" which had undergone a change following amputation. Permanent changes in organization might lead to continuous or recurrent phantom sensations, with or without facilitating stimulation from the periphery. However, Cronholm employed this concept only where pure hyperexcitability seemed insufficient. There was no indication whether or not organizational change might be a result of hyperexcitability.

Supporting evidence of hyperexcitability or increased sensitivity may be found following injury either to peripheral or central nerve pathways. Exaggerated reactions to unpleasant stimuli were described by Head (1920) in patients with lesions of the thalamus. If the extent of the lesion was not sufficient to destroy sensation in the affected half of the body, application of prick, pressure, heat or

cold, or other noxious stimuli often evoked excessive reactions on the affected side, compared with those on the normal side of the body. Although the sensations elicited were more intense, stimulus thresholds might either be raised or lowered. Similar reactions during the period of regeneration of injured peripheral nerves have been described by Livingston (1944), Weddell, Sinclair and Feindel (1948) and others. Teuber, Krieger and Bender (1949) and Haber (1954) found significantly lowered thresholds for touch, two-point discrimination, and point localization in amputation stumps, compared with thresholds for the contralateral limb. Unfortunately, such evidence by itself does not provide a clear case for the hyperexcitability hypothesis.

Evidence from other sources may be found which not only supports an hypothesis of hyperexcitability, but permits one to go much further in describing the effects of sensory deprivation. This is the evidence pertaining to Cannon's "law of denervation" (Cannon, 1939; Cannon and Rosenblueth, 1949). In its generalized form, this law reads as follows:

> When in a functional chain of neurons one of the elements is severed, the ensuing total or partial denervation of some of the subsequent elements in the chain causes a supersensitivity of all the distal elements, including those not denervated, and effectors if present, to the excitatory or inhibitory action of chemical agents and nerve impulses; the supersensitivity is greater for the links which immediately follow the cut neurons and

decreases progressively for the most distant elements (Cannon and Rosenblueth, 1949, page 186).

Because of its relevance to the present discussion, a brief review of this phenomenon is in order. The formulation of the "law of denervation" was the result of a long series of observations which began in 1855 when Budge (cited by Cannon and Rosenblueth, 1949) first noted that sectioning of pre-and post-ganglionic cervical sympathetic fibres in the rabbit produced a curious "paradox" in the pupillary dilation of the two eyes. Within twenty-four hours the pupil of the right eye, which was deprived of its sympathetic nerve supply, was noticeably dilated. This has since been attributed to a sensitization of the iris muscle to chemical agents present within the muscle tissue.

Later experiments showed that similar effects (contraction, fibrillation) were produced in denervated striated muscles (Cannon and Rosenblueth, 1949; Luco and Eyzaguirre, 1955). Teasdall and Stavraky (1953) have recently shown that section of the posterior nerve roots produces increased excitability of deafferented spinal neurones to cortico-spinal impulses.

Fisher and Stavraky (1944) showed that the law of sensitization could be applied in the case of brain lesions. In patients with lesions of the frontal lobes, or of premotor or motor cortex, injections of mecholyl produced heightened sympathetic reactions, tremors, and muscular movements on the affected side of the body. These results were interpreted as a sensitization of the spinal neurones on the injured side following loss of efferent impulses

from the cortex.

Extension of the "law of denervation" to afferent neurones was made by Drake and Stavraky (1948) who sectioned the dorsal roots serving forelimb and hindlimb muscles in spinal cats. Within eighteen hours after deafferentation, intra-aortic injections of acetylcholine, adrenaline, strychnine, or similar agents produced exaggerated responses on the deafferented side. Contractions were elicited by smaller quantities of the drugs, developed more rapidly, and persisted longer than on the intact side. While these results could be explained by sensitization of the deafferented spinal neurones, the authors felt that the concept of sensitization could be extended to all synaptic terminations within the central nervous system, whether afferent or efferent.

Although the phenomenon has been repeatedly demonstrated, the mechanism of sensitization with denervation is not understood. Possibilities which have been suggested include a decrease in the rate of destruction of stimulating chemical substances, increased permeability of cell membranes to these substances, or changes in the physiochemical properties of cells (Cannon and Rosenblueth, 1949; Goodman and Gilman, 1955). Which, if any, of these interpretations is correct is not crucial to the present discussion. The importance of these observations is to show that denervation--or more specifically, deafferentation--may produce a state of hyperexcitability beyond the site of the lesion.

While there have been no reported attempts to demonstrate sensitization in the cortex, there is no evidence to refute the

assumption that it may occur at this level. At the time when Cannon (1939) developed the "law of denervation" he envisaged it as a possible explanation for Jacksonian epilepsy, in which cortical cells which have been "denervated" by localized tumors might become sensitized, and fire in paroxysmal pattern. The evidence which has been reviewed provides a stronger argument for assuming that sensitization may cause spontaneous hallucinatory activity as a result of lesions in the afferent pathways. The "law of denervation" may thus provide a suitable hypothesis to account for a variety of sensory disorders, including phantom limb pain. It is curious that this principle has not previously been applied to hallucinatory disorders, and that Cronholm, in his hypothesis of hyperexcitability, rejected the notion of Lunn (1948; cited by Cronholm, 1951) that phantom limbs might result from a cessation of afferent impulses.

Organizational changes may account in part for some changes in perceptual function following deafferentation. It will be recalled (page 11) that Hebb (1949) proposed a state of hyper-responsiveness of somesthetic nuclei in the thalamus following the decrease in sensory activity accompanying amputation. This was seen to result in disruption of the temporal organization of firing in the somesthetic cortical areas, which might account for the presence of phantom pain. It was also suggested by Hebb that spatial or temporal organization of firing in the normal processes of thought or consciousness (phase sequences) were dependent upon an interaction of sensory processes with autonomous or spontaneous central activity. One might predict from such a theory that a reduction in sensory input over a period of

time would result in intrinsic organizational changes in the pattern of firing, which might interfere with normal perception, and perhaps constitute hallucinatory activity.

Sensitization would contribute to such organizational changes. As deafferentation produces a state of hyperexcitability in single cells within a sensory or perceptual substrate, these cells might become hyper-responsive to activity in neighboring neurones, setting up new patterns of activity. Sequences of activity in sensitized and normal cells might lead to intrinsic patterns of organization which could constitute any variety of hallucinatory activity. If developed over sufficient time, new connections formed in this manner might become resistant to change with remedial treatment.

It is apparent in the case of cataract hallucinations, and in the experimental studies of Bexton, Heron, and Scott (1954) and Scott (1954) which were mentioned earlier, that deafferentation involving tissue damage is not necessary for the production of hallucinations. A uniform sensory field, devoid of the normal changing patterns of perception, is sufficient to produce hallucinatory disturbances. If one assumes that in sensory monotony a large portion of neurones in the higher perceptual areas remain unstimulated, then the theories of sensitization and intrinsic organization may be extended to cover these conditions.

An hypothesis may be evolved on the basis of the foregoing evidence, which states more explicitly the notions already put forth by Lunn (1948; cited by Cronholm, 1951) and Bartlet (1951) re-

garding the production of hallucinations with absence of normal stimulus patterns (page 10). It may be proposed that normal perception is dependent upon an optimum range of variation in patterned sensory input, and that prolonged absence of variable patterned stimulation may result in hallucinations and disturbances in perceptual function. It is felt that such an hypothesis, based upon the notion of sensitization and intrinsic organizational change, can best account for the wide variety of hallucinatory and perceptual disturbances, and the conditions which influence them, which have been reviewed.

In the following pages an experiment will be described which is designed to show the changes in visual function which occur with extreme uniformity or monotony of the sensory field. Although the experiment is most concerned with limiting the scope of visual perception, somesthetic and auditory perception have also been reduced. The relative effects of the different aspects of restriction will be discussed. Although such terms as functional deafferentation or sensory deprivation might be used in describing this experiment, the term perceptual isolation will generally be employed for the sake of precision.

THE PRESENT INVESTIGATION

A review has been made of clinical and experimental data which provide information concerning the effects of deafferentation or perceptual isolation on behavior and central neural function. Included in the review was the study conducted by Bexton, Heron and Scott (1954). The present study is an extension of theirs, and had the particular purpose of investigating the effects on visual function which result from perceptual isolation. As reported by Bexton, Heron, and Scott (1954) and Scott (1954), a severe reduction in the variation of the sensory environment may produce cognitive and motivational disturbances. It was found that both during and immediately following isolation subjects suffered from confusion and impairment in problem-solving capacity. It was also found that during isolation a large number of subjects reported hallucinatory activity. Certain peculiarities of behavior immediately following isolation, together with a number of comments from some of the subjects themselves, indicated that there might also be some perceptual after-effects. It was the purpose of the present investigation to study the hallucinatory activity in greater detail, and to discover the nature of the disturbances of visual perception, both as described by the subjects, and as measured on a series of tests of visual function.

Procedure

The method used to isolate subjects was essentially the same

as that described by the previous experimenters. Except for slight variations with a few subjects, to be noted in the text, the procedure was as follows. The subject lay on a comfortable bed enclosed in a small cubicle. Above the head of the bed was a shielded sixty watt lamp which was left on continuously. The interior walls and overhead of the cubicle were covered with white cloth. A microphone and speaker were placed close to the subject's head so that he could communicate with those outside.

While the subject was in the cubicle he wore cotton gloves on the hands, and rigid cylindrical cardboard cuffs which extended from the elbows beyond the finger tips to limit tactual perception. These were removed during meals and toilet periods. Throughout the entire session, including meal and toilet periods, a pliable translucent plastic mask was worn over the eyes, admitting diffuse light while excluding pattern vision. Sounds from the outside were reduced by the walls of the cubicle, and further masked by the monotonous hum of the fans and air conditioner. Collodion EEG electrodes were placed on the subject's scalp before the experiment, and remained in place throughout. The electrode leads were attached to a terminal board above the head of the bed. Subjects wore either pyjamas or slacks and shirt. Bedding consisted of a flat foam rubber pillow and a light cotton blanket.

At first subjects were fed on request, but later it became necessary to change the procedure so that they were fed at fairly regular intervals three times a day. The subjects fed themselves while sitting on the edge of the bed at the opened door of the

cubicle. A cigarette was permitted after each meal. As far as possible, trips to the toilet were combined with meal breaks. Brushing of teeth, and rinsing of the hands and exposed parts of the face were allowed, but otherwise no washing was permitted.

Before being accepted for the experiment, prospective subjects were shown the apparatus, and told what the conditions would be. Those who expressed doubts about their ability to tolerate the conditions were not accepted. In addition, they were asked not to volunteer unless they intended to stay for the allotted time, which varied in individual cases from three to six days. It was clearly stated, that there would be no compulsion to stay if, having once entered, they found the conditions intolerable. No subjects were accepted whose vision was seriously defective.

There were 13 subjects in the main experimental group. Two of these differed from the rest, being kept in total darkness until near the completion of the isolation period. All were male college students. The age range was from 20 to 30 years, with a mean of 23.2 years. These subjects were paid at the rate of twenty dollars per 24 hours.

A second experimental group consisted of four subjects, ranging in age from 18 to 22 years, with a mean of 20.5 years. This group wore the mask and EEG electrodes, but did not wear the gloves or cuffs, and were not confined in the cubicle. They were encouraged to talk, listen to records and the radio, walk about, and engage in a variety of activities while wearing the mask. They were paid at the rate of fifteen dollars per 24 hours. The purpose of this group

was to test the effects of restricting only visual perception.

A group of 13 control subjects were given the same perceptual tests as were given to the experimental groups, at approximately the same time intervals. These subjects ranged in age from 20 to 32 years, with a mean age of 24.46 years. The pay was fifteen dollars.

Shortly before the subject began his period of isolation, he was given a battery of tests of visual perception. The time taken in giving these tests was approximately 2½ hours. Usually these were given early during the day on which the experiment began, or else during the evening of the preceding day. A wide variety of visual tests were chosen, some because of their recognized value in the clinical testing of visual disorders. These were again administered after the subject left the cubicle, beginning approximately 20 minutes after emergence.

During the day before isolation the subject was also given a short series of tests of tactile discrimination, two-point threshold, and spatial orientation. These were part of a separate study of the effects of perceptual isolation on somesthetic function, conducted by Mahatoo (1955). The same tests were repeated 48 and 72 hours after entering the cubicle.

On entering the cubicle, wearing the mask, the subject was asked specifically to describe his visual field, and was told to report any noticeable change as time went on. Particularly, he was told to report any kind of "visual images" which might appear. When hallucinations were reported, careful notes were taken, and a number of recordings were made of subjects' reports.

When the subject came out, finally, from the cubicle, he was seated on the edge of the bed facing the room. He was told that when the mask was removed he was to look carefully at his surroundings, and report how things looked. He was then systematically asked to describe the appearance of a series of "inspection objects". Here again, notes were taken, and in some cases voice recordings were made. When these observations were completed the visual tests were begun.

EEG records were taken with all subjects at regular intervals throughout their stay, and during periods of hallucination. In addition, 7 subjects were tested daily for metabolic rate, body temperature, pulse and blood pressure. These tests were given only during meal periods.

Perceptual Tests

The following tests of visual perception, administered before and after isolation, were presented in the order shown.

1) Critical flicker frequency. A one-half-inch-diameter disc, viewed at a distance of 12 inches, was illuminated by a small neon bulb which was connected to an oscillator. The score was the mean fusion frequency for 5 ascending trials, starting at 18 cycles per second.

2) Figural after-effect, as described by Köhler and Wallach (1944). The subject was seated 6 feet from the inspection figure which consisted of two black rectangles 1½ by 3 inches. These were to the left and above, and to the right and below a fixation point. Following two minutes inspection, a test figure consisting of a

1 1/8 inch square on either side of the fixation point was substituted. The vertical upward displacement of the right hand square necessary to make the two squares appear level was recorded.

3) Size constancy. A row of 17 grey-green discs against a white background was placed 12 feet from the observer. The discs were graded from 1 to 3 inches in diameter. Six test discs were placed, one at a time, at a distance of 3 feet from the observer, who matched them with instructions to "pick the far disc which looks the same as the near one".

4) Visual acuity. The subject was seated 10 feet from a white card containing a row of 14 vertical black lines, 1/64 of an inch in width, 3 inches in length, and separated by 1 inch. A gap appeared in the top, middle, or bottom portion of each line, the width of the gap decreasing from 3/32 of an inch in the first line, to 1/64 of an inch in the last. Three such cards were presented. Score was the number of lines in which the position of the gap was correctly identified.

5) Phi movement. The subject was seated in a darkened room 10 feet from a screen containing two 1/16 inch diameter illuminated holes, separated by a horizontal distance of 5 inches. A variable speed sectored disc was rotated behind the screen. Maximum and minimum rates of alternation of the lights producing phi movement were recorded with a photoelectric cell and kymograph. Three descending runs were made.

6) Brightness contrast, adapted from the method used by Thurstone (1944). Two illuminated red circles, 7/8 inch in diameter

were presented 3 feet from the subject. The right hand circle was embedded in a brighter red circle 4 3/4 inches in diameter, while the left was seen against a black background. The experimenter reduced the illumination of the isolated circle on the left until the subject reported that the illumination of the two small circles appeared equal. The score was the mean variac setting for 8 trials.

7) Autokinetic effect. The subject was dark adapted for 3 minutes, then seated 10 feet from a pin-point source of light. Time of onset of apparent movement was recorded. A 6-inch-square field of light surrounding the pin-point source of light was controlled by a variac. Two settings were made to determine the amount of light necessary to stop the autokinetic movement.

8) Color adaptation. The subject looked with one eye through a 2-inch plain polaroid filter mounted in the front of a box. Inside the box, 6 inches from the front, was a 2-inch yellow polaroid filter, surrounded by a white circular field 4 inches in diameter. After fixating a point in the centre of the yellow disc for 90 seconds, the subject closed his eye for 10 seconds and then again fixated on the dot while the disc was rotated to reduce the saturation of yellow. A reading was taken in the number of degrees of rotation necessary to make the yellow disc apparently match the surrounding white area, or else to appear blue in comparison with the surrounds.

9) Shape constancy. A white equilateral triangle, 3 inches on a side, was mounted on a tilting grey card, and viewed at a distance of 3 feet. Five settings were made, in which the subject was asked to select from a series of 15 triangles of increasing altitude,

the one which looked to be nearest the shape of the tilted triangle.

10) Brightness constancy. Strips of Hering grey paper were mounted on a card at a viewing distance of 7 feet. The subject was shown a large piece of Hering grey paper, and told its exact position in the series. This was then placed in shadow, and equated with one of the darker shades in the series. Judgments were made with five shades of grey.

11) Rate of figure-ground reversal. A Necker cube, with sides 1 inch long, was viewed from a distance of 5 feet. The number of reversals were counted in half minute intervals for three minutes.

12) After-movement. A 12-inch-diameter spiral was rotated in expanding direction at two revolutions per second for 30 seconds, and viewed at a distance of 10 feet. The duration of contracting after-movement was timed. Three trials were given.

13) Tachistoscopic perception. Nonsense forms were presented on a screen 5 feet from the observer. Exposure time was set at 1/100 of a second. Eight line drawings were presented randomly above, below, to the left, and to the right of a fixation point. These measured approximately 3 inches in height and width on the screen. The subject was to choose the correct form from a set of four placed before him after each presentation. Different forms were presented in pre-and post-cubicle testing.

14) Paper and pencil tests. These consisted of a 10 minute pencil maze test, and three short multiple-choice tests of perceptual speed of two minutes, one minute, and two minutes duration respectively. Separate forms were given before and after isolation.

When the mask was removed, the subject was asked to describe the appearance of a number of objects in the room, some of which will be described in the results. Special apparatus used at this time was as follows.

A grey card 24 by 40 inches with two parallel black lines one half inch wide, 35 inches long, and 3 inches apart was placed in front of a white screen 3 feet from the subject. There was a fixation point in the center of the card. The subject viewed the lines in vertical and horizontal position.

An apparatus similar to that described by Ogle (1950) for measuring the apparent fronto-parallel plane was used before and after isolation. The subject looked through a reduction screen at a white background, in front of which were 9 vertical strings of nylon thread. These consisted of a fixed center string, and 8 adjustable strings suspended from tracks which radiated at angles of 4, 8, 12 and 16 degrees to either side from a point in the center of the interocular base line. The center string could be fixed at a viewing distance of 70, 80, or 90 centimeters. While the subject fixated the center string, the adjustable strings were set one at a time until they appeared to the subject to be arranged in a straight line perpendicular to the line of sight (the apparent fronto-parallel plane). Deviation in millimeters from the true fronto-parallel plane were marked on a chart. Six subjects were tested on this apparatus.

Results

Hallucinations

Nine of the 13 subjects in the main experimental group report-

ed visual hallucinations during isolation. These were of the type reported previously by Bexton, Heron, and Scott (1954). Typically, a subject first reported that he was seeing something with his eyes closed, which appeared to be in front of his eyes rather than a part of his visual imagination. For all subjects, the appearance of such visual activity while fully awake was a new experience.

The form or content of the hallucinations ranged from simple patterns to complex scenes. Arbitrarily, four levels of complexity may be distinguished. At the simplest level, the hallucinations consist merely of patches of light, streaks, or thin lines. Somewhat more complex are the patterns which resemble floral "wallpaper" designs, or textured surfaces. Next are objects such as animals, ships, or printed words. Finally, some subjects report complex hallucinations such as landscapes, groups of people, or scenes containing meaningful sequences of activity.

Often the subjects report that what they are seeing occupies all of their normal field of vision, although at times the activity will be limited to the center of the field or perhaps to one side. Some subjects report that by moving their eyes they can see more of the scene in front of them, as though looking at a real picture. Particularly during the stage in which "wallpaper" patterns are seen, a number of very small patterns may be reduplicated in rows all across the visual field. The hallucinations may appear to be in either two or three dimensions. Usually there is movement, either of the entire field, or of objects within the field. Occasionally the entire field will develop a persistent direction of movement. Sudden

turning of the head may produce swirling or stretching movements within the visual field.

Those subjects who experienced more complex hallucinations usually first reported the simpler forms. Complex hallucinations were experienced less often than the simpler variety, which may be due in part to the fact that not all subjects remained in isolation long enough to reach a more developed stage. There are marked individual differences in the time of onset of the first hallucinatory activity, and in the time course of development of hallucinations from one level of complexity to the next. The times at which different stages of hallucinatory activity were reported by nine subjects in the main experimental group are shown in Table 1.

Of the 29 subjects studied by Bexton, Heron, and Scott (1954) and Scott (1954) under conditions almost identical to those of the present experiment, four were reported to have been confident that they experienced no hallucinations, and seven others were either unable to establish whether they were asleep or awake while having visual experiences, or were uncertain of the time at which their hallucinations occurred. The time of onset of hallucinatory activity in the remaining 18 subjects, together with the times at which "complex" activity was first reported, are shown in Table 2.

Once having started, the hallucinatory activity usually continued during a large part of the subject's waking hours for the remaining period in isolation. Some subjects reported that the visual activity became more intense as time progressed, while others found that it waxed and waned.

TABLE 1

Time in hours of first report of different types of hallucinatory activity in 9 subjects

Subject	Total hours in isolation	Lights, streaks, lines, etc.	Simple patterns	Objects	Scenes
P.J.	72		26		
I.H.	95	0.5	7	24	72
K.M.	88	1.5	50		
B.H.	50	46			
F.B.	115		54	54	100
A.R.	97	42	42		
R.O.	79	55	55		
C.D.	67		12	41	
B.P.	120			38	38

Average time of onset of first reported activity: 30.6 hours

TABLE 2

Time in hours of reports of hallucinations in 18 subjects in previous experiments

Subjects	Total hours in isolation	Onset of first activity	Beginning of complex activity
1	135	72.5	80.5
2	127	41	41
3	102	54	
4	96	73	
5	93	34	
6	91	85	
7	80	10.5	30
8	79	9	35
9	76	34	
10	74	14	69.5
11	69	19	20
12	66	20	50
13	63	16.5	41
14	53	17	28.5
15	50	43	43
16	38	25	
17	32	11	
18	30	15	

Average time of first reported activity: 33.0 hours

An attempt was made to establish the amount of control which may be exercised by the subject over what he saw. Subjects who had developed hallucinations of the more complex type were asked to try to see an object suggested by the experimenter. Rarely did they succeed. Typically the subject would report an object similar to the one which had been requested, suggesting that one might influence the conceptual category of the hallucinations, but not the specific content. Thus one subject failed to see a bottle of beer suggested by the experimenter, but reported an empty bottle upside-down.

The hallucinations were first seen with the eyes closed, and only later, and in a few subjects, were they seen with the eyes open. Occasionally with the eyes open the subject might see a faint outline of patterns which became clearer if the eyes were closed.

As mentioned above, two of the 13 subjects in the main experimental group were kept in total darkness until near completion of their period of isolation. This was done to test whether hallucinations would appear without the continuous presence of diffuse light. One of these subjects (K. M.) is included among the nine who experienced visual activity while in isolation. Both he and the other subject who was kept in darkness reported hallucinations almost immediately when the opaque mask was replaced by the translucent mask toward the end of the experimental session. The influence of light was further investigated by putting other subjects in complete darkness while they were hallucinating. All five subjects with whom this was done (all persistent hallucinators) reported an immediate increase in vividness which lasted for the first few minutes of dark-

ness. In three cases the visual activity disappeared within two hours after the beginning of darkness, and in the remaining cases it was greatly diminished.

All subjects in the present experiment who experienced hallucinations reported that they increased during periods of activity or physical discomfort. In some subjects, hallucinations made their first appearance under such conditions.

Finally, hallucinations were reported by two of the four subjects in the second experimental group, who wore the translucent mask, but were not otherwise isolated. With one of these, the hallucinatory activity made its first appearance while he was being taken for a walk out of doors.

Perceptual Disturbances

All subjects reported perceptual disturbances when the mask was removed. These included spontaneous movements of the visual field, movements induced by changes in the observer's position, surface and linear distortions, and color and contrast effects. A few subjects reported momentary diplopia and difficulty in focusing. All subjects reported some dizziness or nausea, which was pronounced in a few cases. The disturbances were most extreme immediately following removal of the mask, and were largely dissipated within fifteen or twenty minutes, although in a few cases there were effects which lasted for several hours.

Spontaneous movement. Twelve of the 13 subjects in the main experimental group reported some form of spontaneous movement in the visual field--movements which were independent of any motions on the

part of the observer. Most common were reports of shimmering or undulation of the field, which some compared to the effect of looking through "heat waves". There were also reports of systematic movements in portions of the field, such as drifting, rotation, or expansion. After the first few minutes the field grew steadier, and some subjects reported that the area of central vision became stable, while things in the periphery were still in motion. For some time after removing the mask, some noticed that an object fixated for more than a few seconds might alter its shape, or expand and contract. All of the above effects were reported either with one or both eyes.

Induced movements. Eight of the 13 subjects in the main experimental group reported apparent changes in the position of objects with gross body movements or turning of the head or eyes. This may in part be related to a disturbance of size constancy. The most common experience of this sort, reported by 7 subjects, was for objects to seem to approach or withdraw from the observer as he moved quickly forward or back. Some of these subjects also reported that the object seemed to expand or contract as they approached or withdrew from it.

Three subjects reported changes in position induced by turning of the head or eyes from side to side, wherein objects toward which the eyes were turning seemed to fall back, while those away from which the eyes were turning seemed to move closer to the observer.

Surface and linear distortions. One of the most compelling

distortions was that in which a flat surface, viewed at a distance of three or four feet, appeared to bend either in concave or convex direction about an axis lying in the median optical plane. This was most noticeable with the floor boards viewed by the subject while seated on the edge of the cubicle bed. Here the boards seemed either to sweep up into a ridge or down into a hollow, the center of which lay in the midline of the subject's visual field. In some cases the effect reversed itself from concave to convex curvature in the course of five to ten minutes. The effect was also obtainable with a plain grey surface laid over the floor, and with an upright surface such as a wall. The distortion was seen with one eye, but not as markedly as with two.

A second form of surface distortion, reported by four subjects, appeared as a convex bulge near the area of fixation on an upright surface viewed at a distance of three to six feet. This differed from the distortion described above in that it was not formed by an axis dividing the visual field, but occupied a circular area in the center of the field.

When confronted with the grey test card containing a pair of parallel black lines, 11 subjects in the main experimental group reported curvature of the lines. In 8 cases the vertical lines appeared to bow outward away from the point of fixation. In 3 cases the opposite effect was reported, wherein the lines curved inward toward the area of fixation, in the shape of an hour-glass. In all cases the effects were obtained with one or both eyes.

Three subjects reported that the direction of bowing was re-

versed when the pair of lines were rotated through 90°, with the reversal beginning at approximately 45°. Thus, if the lines were perceived as barrel shaped when in a vertical position the curvature became less noticeable as the lines approached a 45° slant, and became hour-glass-shaped as the lines approached the horizontal.

Six of the 13 subjects reported that a horizontal line seen slightly above eye level at a distance of two or three feet, seemed to curve downward at both ends when fixated in the middle. This effect was enhanced if the line or stick was rotated slowly about the fixation point and then stopped in a horizontal position. When seen below eye level the ends of the stick appeared to curve upward. Again, the effects were obtained with one or both eyes, and were more pronounced with both eyes.

Color and contrast. Eight of the 13 subjects reported that colors looked unusually bright or luminous, and that light and dark contrasts were exaggerated. These effects were most pronounced during the first few minutes following removal of the mask, although in some cases they lasted for several hours.

Blurred vision and diplopia. Eight subjects in the main group reported temporary difficulty in focussing. This was most pronounced in the first moments after the mask was removed, and persisted for some time with fine detail. There were only two subjects who reported more than momentary diplopia.

The perceptual disturbances described above are summarized in Table 3, which shows the incidence of reports among the subjects

TABLE 3

Incidence of reports of perceptual disturbances following isolation

Subjects	Total hours in isolation	Shimmering undulation	Drifting, rotation, expansion	Movement in periphery	Movement after fixation	Induced movement (approach)	Induced movement (lateral motion)	Surface bent about midline	Bulge in center of field	Curved ends of horizontal line	Parallel lines deflected	Colors intense	Contrast exaggerated	Blurring	Diplopia
Cubicle															
P.J.	72	X		X				X			X	X		X	
I.H.	95	X	X	X				X	X		X	X	X	X	X
R.G.	108			X	X	X	X	X		X	X	X	X		
K.M.	88	X						X			X				
B.H.	46	X									X			X	
F.B.	115	X		X	X	X	X		X	X	X	X		X	
J.S.	92	X		X		X			X	X					
A.R.	97	X									X				
R.O.	79	X	X		X	X					X			X	
T.N.	94					X		X	X	X	X		X	X	
J.T.	96	X		X	X		X	X		X	X		X		
C.D.	67	X				X		X					X	X	
B.P.	120	X	X			X		X		X	X		X	X	X
Ambulatory															
D.U.	78	X		X				X			X		X	X	
M.Q.	80	X		X				X		X	X				
R.L.	110														
R.C.	92	X	X		X	X	X	X		X	X	X		X	
Experimenters															
T.S.	144	X	X	X	X	X	X		X	X	X	X	X		
B.D.	150	X	X	X	X	X	X	X		X	X	X	X	X	
W.H.	146	X	X	X	X	X	X	X		X	X	X	X		X

in the main experimental group, together with reports provided by three of the experimenters who underwent isolation before the beginning of the experiment. Reports given by the four ambulatory subjects are also shown. It may be concluded from the results with this group that many of the disturbances are due specifically to visual isolation, and not upon the more general isolation imposed by the cubicle.

In addition to the effects which have been described, there were a number of incidental reports which were too infrequent to be included under separate headings. Among these were sensations of strangeness or "unreality" concerning the visual environment, particular distortion of human faces, exaggerated tallness of experimenters standing in the room, exaggerated depth or perspective, accentuated positive after-images (described by two subjects who continued to "see" after closing the eyes), unusually strong negative after-images, and momentary "yellow vision" in which the whole room appeared to be under a yellow light.

The concave or convex distortion of the floor boards suggested that there might be a measurable disturbance of stereoscopic vision. An apparatus designed to measure the apparent fronto-parallel plane (Ogle, 1950) was completed in time to test 6 subjects--3 from the main experimental group and 3 from the special ambulatory group. Five of the subjects reported curvature of flat surfaces. In four of these the curvature was in a convex direction, and in the remaining case it was concave. In each case the apparent fronto-parallel plane showed extreme deviations from the original settings in the direction opposite to the perceived curvature. The sixth subject, who reported no curv-

ature of flat surfaces, showed no marked deviation in the apparent fronto-parallel plane. The measurements are thus consistent with the described surface distortions in each case. Graphs showing the settings for the apparent fronto-parallel plane before and after isolation in the five subjects who reported surface distortions are shown in Figure 1.

Perceptual Tests

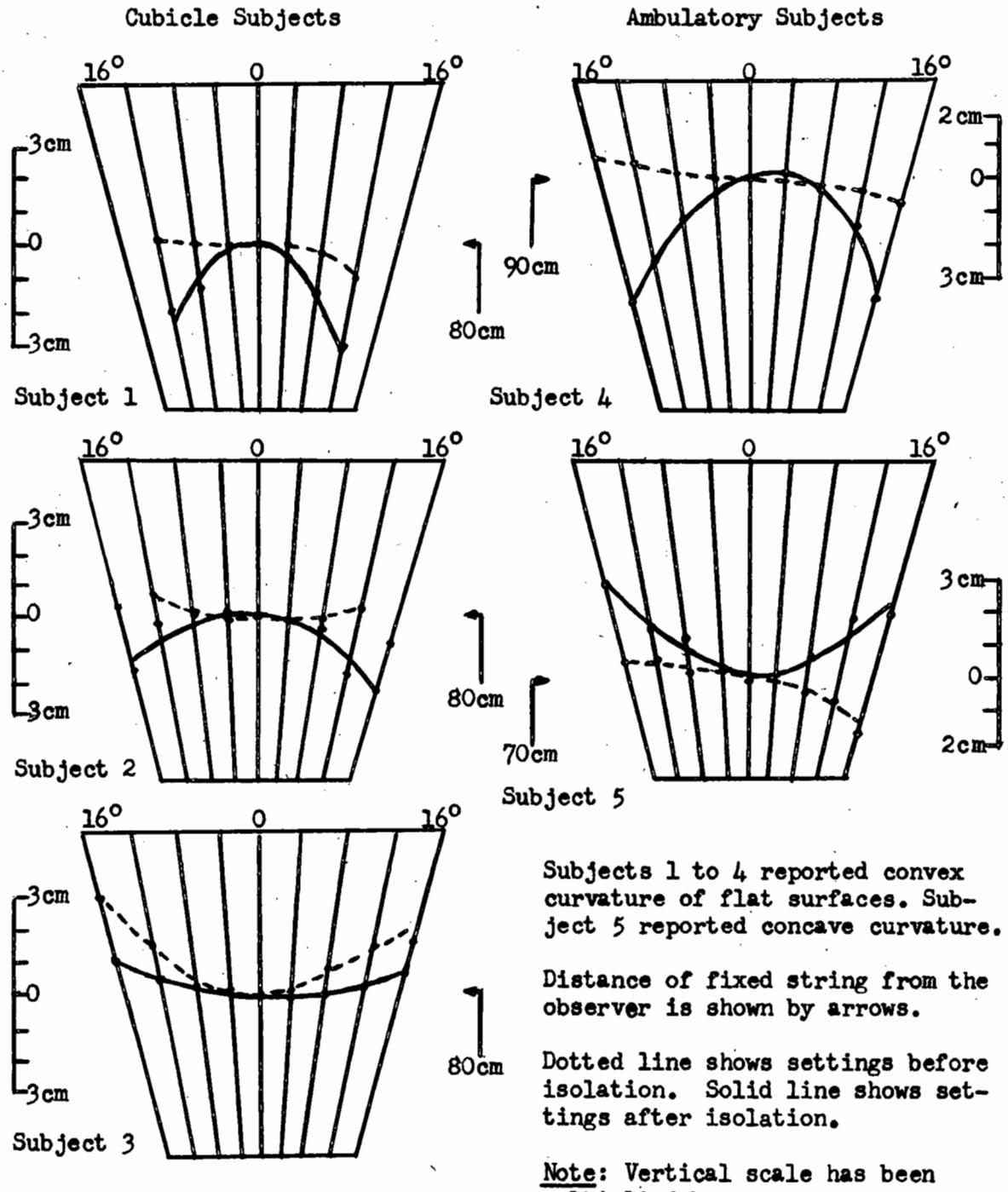
Means of the differences in perceptual test scores for the experimental subjects before and after isolation were compared with those for the control subjects between first and second testing. These are shown in Table 4, together with probability values (based on t tests) of the differences between the means.

On four tests the changes in scores for the main experimental group were found to differ from the changes in the control group at the .05 level of confidence or better. These were the tests of figural after-effects, on which the experimental subjects showed significant increases in displacement; size constancy, on which the experimentals responded more in accordance with the true size of the retinal angle; autokinetic effect, which was more persistent in the experimentals, and color adaptation, which was more pronounced in the experimentals. In no case was the experimental group significantly different from the control group at the time of first testing.

Tests of significance were also made in which the scores of the four ambulatory subjects were combined with those of the other experimentals. Probability values of .05 or less which were obtained by this treatment are also included in Table 4. The same tests again

FIGURE 1

Graphs showing changes in apparent fronto-parallel plane



Subjects 1 to 4 reported convex curvature of flat surfaces. Subject 5 reported concave curvature.

Distance of fixed string from the observer is shown by arrows.

Dotted line shows settings before isolation. Solid line shows settings after isolation.

Note: Vertical scale has been multiplied by 5.

TABLE 4

Mean differences of perceptual test scores in experimental and control groups, and significance values for differences between groups

Test	Mean differences 1st to 2nd testing (Exp.)	(Control)	Significance values of .01 or less*
Cff	- 0.27	- 0.01	
Figural after effect	0.38	- 0.25	less than .02 (.02)
Size constancy	6.31	- 1.23	less than .02 (.02)
Shape constancy	- 4.60	0.15	less than .10
Brightness constancy	- 3.86	0.08	
Visual acuity	1.62	- 0.46	less than .10
Phi (maximum RPS)	0.41	0.04	
" (minimum RPS)	0.04	- 0.04	
" (range)	0.36	0.07	
Brightness contrast	- 2.56	2.03	
Autokinetic effect	2.31	- 1.65	less than .01 (.001)
Color adaptation	-11.13	4.11	less than .02 (.01)
Rate of reversal (1st min.)	1.54	1.85	
(3rd min.)	- 0.54	3.23	
(total)	3.76	9.46	
After-movement	2.24	- 1.07	less than .10 (.05)
Tachistoscope	1.75	0.54	
Pencil mazes	2.09	3.46	
Perceptual speed 1	- 1.09	- 0.23	
Perceptual speed 2	- 0.27	0.54	
Perceptual speed 3	- 0.36	0.54	

* Figures in brackets indicate significance values of .05 or less obtained by addition of ambulatory group to main experimental group

show significant differences. In addition, the test of after-movement becomes significant at the .05 confidence level. The sensation of after movement was of longer duration in the experimental subjects.

Notable trends were also found with the tests of shape constancy and visual acuity. The trend on the test of shape constancy conforms with the results of the size constancy test, in which the subjects respond more in accordance with the true retinal image.

The experimental subjects show some improvement in visual acuity following isolation. The fact that this test did not reach a convincing level of significance is not surprising in view of the possible disturbances of oculo-motor adjustment. The trend toward improved acuity is consistent with the results obtained by Mahatoo (1955) who found significantly lowered two-point touch thresholds in the same subjects.

Discussion

It has been shown that visual function may be severely affected by a prolonged period of isolation from patterned visual stimulation. The hallucinations and perceptual distortions brought about in the present experiment correspond closely to those which were reviewed earlier in cases of damage to the visual system. It would thus seem that such disturbances may result whenever patterned perception is held below an optimum level, regardless of whether the conditions interfering with perception are in the form of tissue damage or environmental restriction. When a comparison is made with the symptoms which were cited in the clinical evidence, the disturbances produced in a number of the experimental subjects may be con-

sidered severe. In this connection it is worth noting how rapidly the experimental subjects recovered once normal visual stimulation was restored. It is not possible to say whether this would have been true with much longer periods of isolation.

The hallucinatory activity and perceptual distortions reported by the experimental subjects bear a striking resemblance to the disturbances produced by mescaline, d-lysergic acid, and similar drugs. Klüver (1942) has described a number of hallucinatory and perceptual "constants" which appear almost universally under mescaline intoxication. These include geometric forms and designs, reduplications of hallucinatory images, and distortions of size or shape in the perception of real objects. The same effects appear with sensory deprivation or perceptual isolation. However, one cannot safely go further in considering the nature of these disturbances until more is known about the specific actions of hallucinogenic drugs within the central nervous system.

In the earlier discussion of possible physiological mechanisms in sensory deprivation, it was mentioned that two kinds of effects might sometimes have to be taken into account, namely, deprivation of the specific sensory projection systems, or deprivation affecting the diffuse reticular or thalamo-cortical projections. This question is pertinent to the present discussion. Disturbances of visual function in the experimental subjects might have been produced by failure of the reticular system to maintain the level of cortical arousal, or <u>tonus cortical</u>, which is required for normal visual function. This is especially true of those subjects who were

isolated in the cubicle, for in their case there was limitation not only of visual perception, but of somesthetic and auditory perception as well, and hence they were deprived of a broad range of non-specific ("arousal") stimulation.

It is also possible that the visual disturbances were due to absence of specific visual input. The results obtained with the four ambulatory subjects are crucial to this question. At the same time, the electroencephalographic data obtained from the experimental subjects should also be taken into account.

Since three of the four ambulatory subjects experienced the same disturbances (hallucinations and perceptual distortions) as did the cubicle subjects, the interpretation in terms of specific visual isolation is strongly favored. This view is also supported by the fact that in a number of subjects the extent of hallucinatory activity increased during periods of restlessness or activity, when the level of non-specific "arousal" function would have been increased. Thus it would seem that non-specific stimulation of the visual cortex might facilitate the production of hallucinations in the absence of patterned vision.

The two interpretations are not, of course, mutually exclusive. It is entirely possible that there are both specific and non-specific effects. The electroencephalographic data are consistent with this supposition. While the effects on the EEG were more marked in the cubicle subjects, the results with both experimental groups show the same trends. In general, there was a progressive slowing in the alpha rhythm, together with a decrease in amplitude and an in-

crease in slow-wave activity (2 to 3 cycles per second). These effects were possibly due to a decrease in tonus cortical as indicated by the fact that a sudden "startle" stimulus tended to bring about a momentary reversal of trends in the EEG. There were no consistent changes in daily metabolic rate, body temperature, pulse, or blood pressure which would account for the EEG changes. Records taken during periods of visual hallucinations consistently showed "flattening" or increased low voltage activity. This may have been a blocking due to the subject's paying attention to his hallucinations. The electroencephalographic data by themselves give no indication of the relative effects of specific and non-specific function with respect to the visual disturbances.

The nature of the hallucinations is generally consistent with the notion of a sensitization, based upon the "law of denervation", which was put forth earlier. This law stated that when a functional chain of neurones is interrupted, the subsequent elements in the chain become supersensitive to the action of chemical agents and nerve impulses (page 28). It was suggested that interruption of the afferent pathways might cause sensitization which would result in spontaneous hallucinatory activity.

For the most part, hallucinations only developed after the subject had remained in isolation for a number of hours--the average time of onset being somewhat over thirty hours. In all cases persistent hallucinatory activity required some time to develop. Individual differences in initial threshold for hallucinatory activity might account for those cases which developed sporadic

hallucinations within a very short time after entering isolation.

The progressive development of the hallucinations from simple to more complex forms is also consistent with the notion of sensitization. It was stated in the "law of denervation"(page 28) that sensitivity decreases progressively away from the site of interruption in a chain of neurones. Similarly, the time taken for sensitivity to develop has been shown to increase with the distance from the lesion (Luco and Eyzaguirre, 1955). Accordingly, it would be expected that simpler hallucinations, correlated with activity in the primary visual areas, would develop in a shorter time than more complex forms, which must be correlated with activity at a higher level.

In the review of clinical evidence a reference was made to de Morsier's (1938) generalization that visual hallucinations are almost always accompanied by disturbances of visual perception. These were said to include distortions of form and apparent movement in the visual field, which are comparable to the disturbances reported by subjects in the present experiment. To what extent hallucinations are related to perceptual disturbances in the experimental subjects is not clear. Some subjects who reported disturbances following isolation had not developed hallucinations, although it is possible that they might have gone on to develop hallucinatory activity had they remained a little longer in isolation. Unfortunately there is no evidence of the time taken for the perceptual disturbances to develop, since these were never observed until after removal of the mask at the end of the experiment.

With respect to the perceptual tests it should be noted first that the subjects had considerably recovered from the effects of isolation before testing began. Three of the tests which showed significant changes were measures of stimulus after-effects. These were the tests of figural after-effects, color adaptation (which is also assumed to be a measure of negative after-image intensity), and after-movement. These effects are poorly understood in themselves, and no attempt will be made here to explain their nature. Assuming the test of color adaptation to be correlated with the intensity of negative after-images, it is worth noting that after-images sometimes are strengthened in cases of brain injury (Bender and Teuber, 1946, 1949).

The most significant test results were obtained with the autokinetic effect, which again is poorly understood. The increased persistence of the movement in the experimental subjects would appear to be consistent with the spontaneous movement and instability of the visual field experienced when the translucent mask was first removed. Woodworth and Schlosberg (1954) relate the autokinetic effect to loss of spatial orientation. Disturbances of orientation in the cubicle subjects, (as measured by somesthetic function,) were found by Mahatoo (1955).

The disturbances of size constancy are almost certainly related to the induced movements in the visual field which occurred with head or eye movements of the observer. It has been shown by Gibson (1954) that stability of objects in the field of vision must be a form of constancy phenomenon. The nature of size constancy or

its disturbance is not, however, easily explained. Kohler (1951, 1955) has shown through the use of distorting lenses that perceptual constancies apparently develop in accordance with the aspects of visual perception which are most repeatedly perceived, and that learned constancies may break down if the patterning of perception is altered for a sufficiently long period of time. These data, together with the results of the present experiment, suggest that some of the basic learned constructs of visual perception must be continually reinforced by normal patterns of stimulation if normal perceptual organization is to be maintained.

In conclusion, the results of the present experiment indicate that normal visual function depends upon an optimum range in patterned sensory input. Prolonged absence of variable patterned stimulation results in hallucinations and perceptual disturbances. At present, the perceptual disturbances themselves seem best accounted for in terms of intrinsic organizational changes within the visual system, while hallucinations may be explained by sensitization of neurones according to the "law of denervation". Changes in sensitivity might be an important factor in determining organizational changes. It is felt that these findings may account for a variety of clinical disturbances of visual perception.

SUMMARY

1) An experiment has been described in which human subjects were isolated from patterned visual stimulation for periods lasting up to several days.

2) In a number of cases there were pronounced visual hallucinations during isolation. These disappeared immediately when patterned vision was restored.

3) Immediately following isolation there were a variety of disturbances of visual perception. These included movements in the visual field and a number of surface and linear distortions. These effects largely disappeared within fifteen or twenty minutes after patterned vision was restored.

4) Significant changes were found on tests of figural after-effects, size constancy, autokinetic effect, color adaptation and after-movement.

5) In all respects the results obtained with ambulatory subjects were comparable to those obtained with subjects isolated in the cubicle. It thus appears that the results are largely due to specific visual isolation, rather than to deprivation of the reticular activation system.

6) It is concluded that normal visual function is dependent upon an optimum range of patterned visual stimulation. It is felt that absence of appropriate stimulation may account for a variety of clinical disorders of visual perception.

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