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DENIAL OF BLINDNESS FOLLOWING CEREBRAL ANGIOGRAPHY

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In the fifty-eight years since the original report of Anton (1), there has been controversy in the literature as to whether denial of blindness is the result of a specific focal cerebral lesion or of a generalized disturbance of brain function without specific localizing significance.[2] In reviewing the cases of denial of blindness, the majority of reports describe the patients as "confabulating," "disoriented," or "showing a Korsakoff psychosis." Such descriptions lend support to the concept that denial occurs in a milieu of altered cerebral function. Studies of denial of hemiplegia, usually described under the term "anosognosia," bring out identical arguments as to the significance of the phenomenon for localized dysfunction. Indeed, in many reports of denial of blindness, note is made of simultaneous denial of hemiplegia or of other defects.

The literature of denial of illness, as well as clinical and experimental evidence to support their concepts, has been recently summarized by Weinstein and Kahn (23). They conclude that various forms of denial are a unitary phenomenon without cerebral localizing value, and that denial is an adaptation to a defect in the milieu of diffusely altered cerebral function. In this regard, most of the defects denied are of rapid onset, are not limited to one defect, are accompanied by confabulation, amnesia, changes in mood and absence of anxiety. The degree of altered cerebral function which provides the milieu for such adaptation is usually severe. Thus, their reports, as well as those of other authors cited (see footnote 2),

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[2] The reviews of Critchley (6) and Weinstein and Kahn (23) present the two aspects of this problem. For specific reports ascribing the phenomena to focal cerebral disease see Barkman (2), Gerstmann (10), von Hagen and Ives (21, 22), Ives and Nielsen (12), and Pötzl (16). Reports ascribing the phenomena to diffuse cerebral disturbances include Lunn (13), Redlich and Bonvicini (17), Redlich and Dorsey (18), and Sandifer (19).

describe the phenomena of explicit denial of blindness and of hemiplegia as occurring in patients with brain tumors, subarachnoid hemorrhages and vascular disease.

The following case history is presented as exemplifying various aspects of the syndrome of denial. The data support the thesis that the phenomenon is an adaptive response to a defect under the conditions of altered cerebral function, rather than the result of focal cerebral pathology. A patient, under observation for enlargement of the sella turcica presumably the result of pituitary adenomatous growth, was subjected to cerebral Iodopyracet (Diodrast) angiography. Before the procedure he was alert and oriented, but immediately following the second series of injections of Iodopyracet, he developed left hemiplegia, which gradually resolved. In the ensuing hours, blindness developed and was denied by the patient. The syndrome persisted for 48 hours, and then resolved. When the patient was seen in a follow-up visit eight months later there was an amnesia for the period of denial.

Case Report:[3] *E. S.,* a 58-year-old right-handed male, was admitted for diagnostic study to the Montefiore Hospital with a six-months history of headaches and blurring of vision. Four months previously he had an episode of ptosis of the right lid associated with dilatation of the right pupil, which had persisted for a few weeks. Headaches became increasingly severe, and X-ray examination of the skull prior to admission demonstrated an enlarged sella turcica.

He related his own history; appeared neither acutely nor chronically ill; was alert, well oriented, and cooperative, with good memory and calculating ability. He was jovial, made friends readily, and was well liked. He denied previous severe illnesses, or persistent somatic complaints. He was fastidious about his personal belongings and was reluctant to intrude. The general examination was normal except for palpable enlargement of the right lobe of the thyroid gland. Neurological examination was normal except for the cranial nerve examination. His pupils were dilated, the right larger than the left. The reaction to light was sluggish on the right, and the pupils reacted well to near vision. The fundi showed well-outlined papillae with clear margins, definite temporal pallor, and normal vascularization. Visual acuity was 15/20 on the right, and 15/40 on the left. Visual fields to 1/2000 white test object demonstrated a relative bitemporal hemianopsia without macular sparing.

The lumbar puncture and routine blood and urine studies were normal. Skull X-ray revealed enlargement of the sella turcica; atrophy of the anterior and posterior clinoids; and calcifications along

[3] Patient studied through the courtesy of Dr. Nathan Savitsky at the Montefiore Hospital.

the lateral border of the sella. Pneumoencephalography demonstrated encroachment of the cisterna chiasmatis and pontis by a mass originating from the sella. An electroencephalogram showed a slight degree of electrical abnormality on the left side, mainly inferior and posterior. Alpha frequencies and amplitude were symmetric.

For further clarification of the pathologic process, carotid angiography was recommended. Under local anesthesia, the right common carotid artery was exposed, and forty cc. of 35% Iodopyracet (Diodrast) was administered. Since the serial angiograms thus made were unsatisfactory, another injection of 15 cc. Iodopyracet was made. Immediately following this injection, the patient developed a complete left hemiparesis, including the face. He was restless, confused and irritable. He appeared drowsy; failed to obey commands and was irrelevant in speech. Vasodilators were administered, and the hemiparesis showed some improvement.

That evening he was restless, directing his gaze most often to the right. When spoken to from his left side, he would turn his head to the right or backward, before finally localizing the voice correctly. He answered questions relevantly. Visual acuity was reduced to light perception, and pupillary reactions were present, though sluggish. He was unable to localize the position of a light nor identify fingers or objects; yet he denied his inability to see, confabulating seemingly appropriate responses. He was oriented for place but only approximately for time and date. Despite a large neck bandage, he denied the recent cutdown. A lumbar puncture was performed. The initial pressure was 140 mm. CSF, final pressure of 60 mm. after the removal of 8 cc. of clear, colorless fluid which had no cells and a protein content of 43 mg%.

Twelve hours later, now oriented in space and time on gross questioning, he was still unable correctly to localize light or perceive objects. There was a residual left hemiparesis. He denied both his weakness and his blindness. When walking about the room he stumbled over objects and bumped into the wall and the bed. He correctly identified the various examiners by their voices, and named a coin, key, pencil and comb by touch. There was no sensory loss on single stimulation; gait was hesitant; and the reflexes were increased on the left with bilateral Babinski responses and absent abdominal reflexes.

Thirty-six hours later the hemiparesis had cleared except for a residual left Babinski response. He perceived light and localized it well in space, but image formation for reading or fine identification was impaired. Despite the partial nature of his vision, he still failed to recognize his impairment, confabulating many responses. An electroencephalogram at this time showed a change from the original record. There was bilateral asymmetry with high per cent time delta activity and a slowed, poorly organized alpha rhythm, mostly on the right.

Forty-six hours later his vision had returned so that he was able

to read. He was oriented, alert, affable and friendly. He maintained that he had been able to read and to see throughout the previous two days. He had an amnesia for the surgery, the hemiparesis and the blindness. During the ensuing weeks, visual acuity returned to normal; with visual fields manifesting minimal bitemporal hemianopic defect. An electroencephalogram one week later showed posterior voltages to be less depressed; per cent time delta activity had decreased; and there was desynchronization of the record on eye opening. There was focal accentuation of the delta abnormality in the right frontal leads. Three weeks later the electroencephalogram showed increased resolution of the abnormalities with increased and bilaterally equal per cent time alpha; decreased per cent time delta and resolution of the electrical asymmetry of the hemispheres.

On examination eight months after this episode and after a course of radiation therapy for pituitary adenoma, this patient was alert, oriented and cooperative; with only occasional complaints of headache. The neurological examination was completely negative with normal visual acuity and a slight (10°) bitemporal hemianopic defect with 1/1000 white test objects. He denied any experience of blindness or weakness but did recall the neck dissection that preceded the angiography. When told of the experience, he jokingly denied the weakness and the blindness by saying that I was mistaking him for another patient.

Discussion: Two aspects of this case report warrant amplification: the significance of the denial phenomenon and the cause of complications following cerebral angiography. The various aspects of denial of illness described by Weinstein and Kahn are well exemplified here. The acute onset of hemiplegia and blindness was followed by a period of restlessness, disorientation, and altered consciousness. Within a few hours, these gross symptoms were replaced by a calm, disinterested, smiling attitude in which the multiple defects of left-sided weakness and blindness were denied. He confabulated, was disoriented for time and date, and later was amnestic for this period. An electroencephalogram demonstrated bilateral diffuse slow wave activity of high voltage. Furthermore, despite the visual loss, the phenomenon of spatial inattention[4] was observed. This complex of symptoms and signs is generally noted in diffuse cerebral disorders. While much effort has gone into localizing these defects, it is difficult to conceive a single focal lesion affecting the visual tracts bilaterally, the right hemisphere in an

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area productive of hemiplegia and the frontal areas assumed to be productive of apathy, denial, and loss of anxiety. While the possibility of a focal lesion as the basis for this syndrome cannot be ruled out, it is more tenable to conclude that diffuse cerebral dysfunction was present. This conclusion is supported by the electroencephalogram, and also by experimental evidence noted below, demonstrating the effect of intra-arterial Iodopyracet as inducing severe vasospasm followed by generalized cerebral edema and increased permeability of the blood-brain barrier.

The significance of the phenomenon of denial is to be seen in its defensive nature. While undergoing a test procedure, the patient suddenly suffers a catastrophic disability. His initial response of severe anxiety, manifested by restlessness, startle reaction, and irritability, is soon replaced by explicit denial. This primitive, "psychotic" defense is normally present only in childhood. But under the special conditions of cerebral dysfunction, with disturbances in spatial and temporal orientation and perception, denial of reality becomes tenable. It is maintained so long as the disability and the milieu of cerebral dysfunction persist. In this patient, as soon as visual perception was sufficient for reading, confabulation and explicit denial were no longer actively maintained for ongoing events. In the special situation of interviews with the staff during the period of visual and motor loss, the patient manifested no concern and confabulated responses readily. When visual acuity returned and his hemiparesis cleared, he maintained the same friendly, affable, unconcerned attitude. In this regard it is important to note the characterological factor in explicit denial of illness. To the extent that the information is now available, this patient manifested a considerable number of the features described by Weinstein and Kahn (24).

Special note should be made of the phenomenon of spatial inattention. The patient's original visual complaint of blurred vision was accompanied by a minimal bitemporal hemianopia, apparent only on testing with 1/2000 white test objects. During the period of visual loss he was unable to locate a light and confabulated responses. One week later the bitemporal hemianopia was present to 3/2000 white test object, but in addition there was an irregular left homonymous upper temporal field defect to 5/2000 white. Evidence of a left homonymous field defect persisted in examinations for three weeks, after which only residual bitemporal defects were persistently reported. Left spatial inattention was prominent in the first 48 hours of this syndrome only, at the time when visual im-

pairment was maximal, and when hemiparesis was present. When the hemiparesis receded, visual function returned, and orientation was intact, then spatial inattention disappeared. Thus, spatial inattention was an aspect of the total disturbance in function, possibly motivated by the left-sided defects, and was probably not dependent on a specific visual field defect.

The syndrome of blindness and its denial following cerebral angiography is unique. Focal lesions producing transient hemiplegia, hemisensory defects, seizures, aphasia, and various cranial nerve syndromes have been described. In a series of 117 percutaneous carotid angiograms, Fink and Stein (9) noted an 8 per cent morbidity of such transient phenomena. Other series variously report such complications from 3 to 15 per cent of the cases.[5] These figures do not include the few patients in whom the complications as hemiplegia, aphasia or exaggeration of their basic disease are permanent; or who succumb. In these studies of the complications of angiography, emphasis is placed on the relation of the concentration of the contrast medium, the rate and quantity of contrast substance injected and the time within which the injections are repeated. In experimental studies Olsson (14), Broman and Olsson (5) and Bloor et al. (3) demonstrated summation of toxic effects when the contrast substance was rapidly injected into animal arteries; and noted increased vascular permeability, cerebral edema and petechial hemorrhages not limited to the side of the injection. While it is possible that the complications of angiography are the result of thrombus formation at the needle site and focal embolization, it is more likely that diffuse toxic cerebral vascular changes are induced as seen experimentally. The diffuse character of the defects and the transient nature of the phenomena in this patient are readily understood in this context.

Summary and Conclusions: In the course of carotid angiography in a patient with evidence of a pituitary adenoma, an acute transient episode of blindness and hemiplegia developed. Following a short period of restlessness and confusion, the patient became calm, denied his blindness and weakness, confabulated responses to questions, was disoriented, and manifested spatial inattention.

The diffuse nature of the cerebral dysfunction underlying this syndrome is emphasized by noting the distribution of the presumed lesions, the bilateral, diffuse slowing of the electroencephalogram,

[5] For reviews of the complications of cerebral angiography, see Engeset (8), Fink and Stein (9), Green and Arana (11), Perese et al. (15) and Wickbom (25).

and the diffuse nature of the toxic sequellae of intra-carotid Iodopyracet (Diodrast).

The defensive-adaptive significance of the syndrome of denial of blindness and hemiplegia is discussed, with emphasis on the development of this attitude under the special conditions of altered frames of temporal and spatial reference provided by altered cerebral function.

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Denial of Blindness Following Cerebral Angiography

In the fifty-eight years since the original report of Anton (1898), there has been controversy in the literature as to whether denial of blindness is the result of a specific focal cerebral lesion or of a generalized disturbance of brain function without specific localizing significance (1). In reviewing the cases of denial of blindness, the majority of reports describe the patients as "confabulating," "disoriented," or "showing a Korsakoff psychosis." Such descriptions lend support to the concept that denial occurs in a milieu of altered cerebral function. Studies of denial of hemiplegia, usually described under the term "anosognosia", bring out identical arguments as to the significance of the phenomenon for localized dysfunction. Indeed, in many reports of denial of blindness, note is made of simultaneous denial of hemiplegia or of other defects.

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rapid onset, are not limited to one defect, are accompanied by confabulation, amnesia, changes in mood and absence of anxiety. The degree of altered cerebral function which provides the milieu for such adaptation is usually severe. Thus, their reports, as well of other authors cited (1), describe the phenomena of explicit denial of blindness and of hemiplegia as occurring in patients with brain tumors, subarachnoid hemorrhages and vascular disease.

The following case history is presented as exemplifying various aspects of the syndrome of denial. The data supports the thesis that the phenomenon is an adaptive response to a defect under the conditions of altered cerebral function, rather than the result of focal cerebral pathology. A patient, under observation for enlargment of the sella turcica presumably the result of pituitary adenomatous growth, was subjected to cerebral Iodopyracet (Diodrast) angiography. Before the procedure he was alert and oriented, but immediately following the second series of injections of Iodopyracet, he developed left hemiplegia, which gradually resolved. In the ensuing hours, blindness developed and was denied by the patient. The syndrome persisted for 48 hours, and then resolved. When the patient was seen in a follow-up visit eight months later there was an amnesia for the period of denial.

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For further clarification of the pathologic process, carotid angiography was recommended. Under local anesthesia, the right common carotid artery was exposed, and forty cc. of 35% Iodopyracet (Diodrast) was administered. Since the serial angiograms thus made were unsatisfactory, another injection of 15 cc. Iodopyracet was made. Immediately following this injection, the patient developed a complete left hemiparesis, including the face. He was restless, confused and irritable. He appeared drowsy; failed to obey commands and was irrelevant in speech. Vasodilators were administered, and the hemiparesis showed some improvement.

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Discussion:

Two aspects of this case report warrant amplification: the significance of the denial phenomenon and the cause of complications following cerebral angiography. The various aspects of denial of illness described by Weinstein and Kahn are well exemplified here. The acute onset of hemiplegia and blindness was followed by a period of restlessness, disorientation, and altered consciousness. Within a few hours, these gross symptoms were replaced by a calm, disinterested, smiling attitude in which the multiple defects of left sided weakness and blindness were denied. He confabulated, was disoriented for time and date, and later was amnestic for this period. An electroencephalogram demonstrated bilateral diffuse slow wave activity of high voltage. Furthermore, despite the visual loss, the phenomenon of spatial inattention (2) was observed. This complex of symptoms and signs is generally noted in diffuse cerebral disorders. While much effort has gone into localizing these defects, it is difficult to conceive a single focal lesion affecting the visual tracts bilaterally, the right hemisphere in an area productive of hemiplegia and the frontal areas assumed to be productive of apathy, denial, and loss of anxiety. While the possibility of a focal lesion as the basis for this syndrome cannot be ruled out, it is more tenable to conclude that diffuse cerebral dysfunction was present. This conclusion is supported by the electroen-

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Summary and Conclusions:

In the course of carotid angiography in a patient with evidence of a pituitary adenoma, an acute transient episode of blindness and hemiplegia developed. Following a short period of restlessness and confusion, the patient became calm, denied his blindness and weakness, confabulated responses to questions, was disoriented, and manifested spatial inattention.

The diffuse nature of the cerebral dysfunction underlying this syndrome is emphasized by noting the distribution of the presumed lesions, the bilateral, diffuse slowing of the electroencephalogram, and the diffuse nature of the toxic sequellae of intra-carotid Iodopyracet (Diodrast).

The defensive - adaptive significance of the syndrome of denial of blindness and hemiplegia is discussed, with emphasis on the development of this attitude under the special conditions of altered frames of temporal and spatial reference provided by altered cerebral function.

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