A CASE OF ASTASIA-ABASIA AND SPEECH PERSEVERATION, FOLLOWING CARBON MONOXIDE POISONING.

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INTRODUCTION.

EDSALL¹ lists forms of neuritis, palsies, anæsthesias, neuralgias, choreiform movements, intention-tremors, scanning and stuttering speech, incontinence of urine, Landry's syndrome, multiple sclerosis, blindness, and marked mental changes, as sequelæ of carbon monoxide poisoning. McGurn² mentions more than one hundred neurological and other symptomatological conditions, as well as two cases of a syndrome not unlike that of multiple sclerosis. Dana³, in the early days of corpus striatum physiology, associated the lesions observed in the basal ganglia in carbon monoxide poisoning with the symptomatology of the disease, mentioning disturbances of articulation and mentality, but assigning no great motor function to the basal ganglia. More recently Hill and Semarak⁴, Stewart⁵, and Rugh⁶, have collected a number of data on the pathological changes seen in persons dead from gas poisoning. The latter has collected a large bibliography on the subject, consisting chiefly of cases showing one or several of the above mentioned conditions, with many reports on the pathological findings in the basal ganglia. More recently Alpers¹⁰ has reported a case with recovery, showing evidence of complex central and peripheral derangement.

The essential features of the nervous pathology, drawn chiefly from the findings of Hill and Semarak (32 cases) and Rugh (12 cases), are as follows :

1. There is a bilateral ischæmic necrosis of the lenticular nuclei, especially of the globus pallidus. This necrosis may be due to several factors. It may result from degeneration of vessel walls with thrombosis due to destruction of red cells, or as a result of the noxious gases in the blood plasma irritating the vessel wall (carbon monoxide is non-irritant, but illuminating gas may be). A more likely explanation is that in keeping with Haldane's observations. The CO in uniting with the hæmoglobin replaces oxygen ; no gaseous exchange can take place. As a result the endothelium of the vessel is impaired or destroyed, with secondary thrombosis. Still another explanation is that owing to anatomical peculiarities in circulation this particular site is chosen. Unfortunately, much as this explanation pleases, there is at present lack of evidence to prove its truth.

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2. The degree of pathological change varies from slight perivascular lesions of the globus pallidus, to gross softening, depending on the amount of gas inhaled, the duration of life after poisoning, and the presence or absence of sclerotic changes in the vessels. Cases in which the blood-vessels show a high degree of sclerosis will, in the opinion of these observers, be found to exhibit most marked changes in the parenchymatous tissues. This has an important bearing on the disease as it occurs in elderly persons or in those with a history of chronic intoxication.

3. Nerve cells possess a high degree of tolerance to the action of the gas. This is in accord with Haggard's⁷ work on embryonic nerve tissue. When such tissue was exposed to atmospheres of 79 per cent. CO and 21 per cent. O, little or no disturbance in growth was noted. It must be said, however, that when as little as .1 per cent. of illuminating gas was used death of the tissue resulted invariably. In material from a case in which death occurred twenty-seven days after exposure, Stewart noted degeneration of the spinal nerves and the vagus. Whether this was due to a direct action of the gas on nerve tissue or to the processes described above (secondary to vessel changes) it is difficult to determine. The same observer, in addition to the findings described, noted a zone of softening of the deeper layers of the cortical gray matter, punctiform hæmorrhages, and in the spinal cord a washing out of the anterior horn cells, which tended to diminish in the caudal portion of the cord.

Hill and Semarak noted œdema of the brain in about two-thirds of their cases, internal hydrocephalus in about two-thirds, a pink colour in about one-third, petechial hæmorrhages in one half, arteriosclerosis in all, and, in more than half, degrees of vascular degeneration excessive for the age of the individual. There was gross bilateral softening of the lenticular nuclei in slightly less than half, the most marked changes being observed in older persons with vascular degeneration. Of the fifty-six per cent. (18 cases) showing no gross softening, eleven patients were under forty and thirteen died within three days.

In experimentally produced carbon monoxide intoxication¹⁵, brain œdema has actually been seen to develop and evidence of eye changes (papilloedema) has been recorded. The severe headaches of the disease are thought to be caused by this.

Vessel changes are not limited to those supplying nerve tissue (Briggs⁶).

On the neurological side, therefore, this intoxication results in symptoms arising from several levels. Cortical degeneration might well give rise to most of the disturbances of intellect, emotion, inhibition, and possibly to sphincter and pyramidal abnormalities; subcortical degeneration to tremors, disturbances of co-ordination and facial expression, choreiform⁸ or athetoid activities, and possibly nystagmus; cord and spinal nerve degeneration to neuritis and lower motor neurone types of palsy¹⁴. The frequency of symptoms from each of the several levels is, of course, difficult to determine. Depression

is one of the commonest complaints in chronic intoxication, but this may well be the reaction to generalized tissue invasion. In severe acute intoxications depression, amnesia, dementia, and personality changes have been described¹⁰. Nevertheless, the most characteristic syndromes are those which supposedly have their origin in lesions of the basal ganglia.

CASE HISTORY.

The case here presented is probably one of this class. It is unusual, in as much as few of this type, with the given etiology, have been described in the literature; and it adds another observation to the interesting problem of voluntary versus involuntary motility.

An American salesman, age 46, entered the hospital complaining of difficulty in walking and speech.

Family History.—His father hanged himself during an attack of depression, and his mother died in an insane asylum after a prolonged mental illness in old age.

Previous History.—His birth and early childhood were normal. He had none of the common childhood diseases. He played well with other children and attained a fair standing in his school work. He was sociable and friendly, although somewhat inclined to stay at home and not associate as freely with those of his own age as did other members of his family. He indulged moderately in autoerotic practices until he was about seventeen, after which he sought heterosexual expression. Industrially, he was quite successful. He occupied himself as salesman the greater part of the time, and was employed for sixteen years by the firm with which he was last affiliated. At seventeen he began drinking heavily, and for the last ten years of his alcoholism he consumed at least a quart of whisky per week. At thirty-nine he had delirium tremens, and at forty-one he ceased to use alcoholic beverages.

At the age of twenty-three he developed a depression which lasted one year. This was unaccompanied by morbid somatic sensations. At no time has the patient been aware of delusions, illusions, or hallucinations (except during the attack of delirium tremens) or compulsive phenomena. He has always readily become depressed, although his depressions were usually transitory in character. He readily loses his temper, and is sensitive to the opinion of his fellows. He has never noted prolonged or short periods of excitement. It was during a fit of depression that he attempted suicide (see below).

He married for the first time when he was twenty-eight. He was divorced by his first wife at thirty-nine because of her complaints of brutal treatment, and because of his meanness in things financial. Immediately after, he married a widow with three small children and lived happily with her until three years ago. At that time domestic trouble ensued. He attributes his domestic strife to his progressive loss of sexual power. After two months of strained relations of this type, the patient developed a 'nervous breakdown,' and became greatly depressed. He claims that he saw his wife put "pills" in his coffee, and feared that she was seeking his life. He thereupon left her and shortly after attempted suicide by gas.

Present Illness.—After his exposure to gas (three years ago), he remained unconscious for 48 hours. For a month and a half he could not 'walk or move.' Sometime shortly after this he was able to sit up and move himself about in a wheel chair. Six months after his poisoning he began to walk as at present. He devised

a cane with a hook on the lower end of it, which enabled him to drag his lagging foot forward. His speech defect could not be accurately dated, but apparently came on about the same time as his difficulty in walking. Since that time he has been much hospitalized, repeatedly diagnosed as hysteria, given much suggestive and manipulative treatment, but always presenting essentially the same picture as at present. His sister, who has nursed him, has noted, however, a gradually progressive mental change. He has become more and more untidy about his clothes, often goes about with his trousers unbuttoned, occasionally soils himself when in bed, chiefly with urine, but on several occasions, even in public places, with faeces. He eats with little regard for table manners, drops food about himself, and soils his linen regardless of reprimand. This is very different from his behaviour before the poisoning.

Physical Examination.—The patient is big and somewhat obese, and appears older than his actual age. His facies is sad or apprehensive, and immobile except during occasional outbursts of weeping. Usually there is little facial expression.

The muscles supplied by the cranial nerves show no defect, with the exception of the Parkinsonian facies. The tongue is protruded in the midline promptly and presents no atrophy or tremor. There is occasional very slight drooling of saliva. The voice is thin, high-pitched, and lacking in volume. It is also entirely lacking in intonation, and is dull and frequently explosive. Certain consonants are especially difficult to articulate. During conversation words are repeated over and over again. There is a gush of words when an answer is given to a question. For example: "How do you feel?" "I feel fine, fine, fine." It was noted that he repeatedly had difficulty in the expression of those words that had an emotional association. The rate of speech is extremely rapid. When asked to repeat slowly certain test words after the examiner, there is no perseveration, and the quality of the voice seems better, but when asked to repeat the same words without the model sounds to copy from, the perseveration reappears. Frequently when a word persistently repeats itself, he will spell the word out, as, "What colour is this chair?" "White, white, white, white . . . W H I T E," all at a very rapid pace, the spelling as rapidly as the remainder of the flow.

Motor System.—The patient stands erect. There is no muscular weakness. There is a cog-wheel rigidity at the elbow, when the forearms are passively moved. The gait is clumsy and spastic. In starting to walk, the left leg is pushed in front and the right remains behind. Thus he may remain for several moments, after which the hindmost leg may be brought slightly forward, but not in front of the foremost leg. The latter leg may then be raised slightly and pushed forward. The steps are very short and the patient alternates between shuffling and sticking. There is always especial difficulty in getting started, after which motion is continued as described. He uses a cane, and when this is taken from him he waves his arms about and leans on or towards anything that will afford support. When this is not possible, he spreads his legs and extends his arms literally in an attempt to balance himself. When the patient sits down on a chair, there is no gradual recline or 'easing' into the chair, but a descent en masse, so that he strikes with a thud and rolls well back. At times, especially in the morning, the patient walks apparently normally. The steps are still short, but the natural progression is followed. Even when his walking is at its worst, usually late in the evening or night, he is still able to step over obstacles placed in his path. There is a slight tremor of the hands. The handwriting is more or less characteristic of that commonly seen in paralysis agitans (see fig. 1). On one occasion, while addressing a letter, the patient was seen to write the name of the city twice. He plays the piano with

a peculiar rapidity, and with faulty fingering and expression, and insists that he perseverates on certain phrases, but the playing was so poor that the observer could not be certain of the fact. His playing, according to friends, is said to have materially altered, but this remains uncertain.

The retinae show a moderate degree of arteriosclerosis, with some vessel tortuosity, but no hæmorrhages. The radials are rigid and slightly tortuous.

No abnormalities are found in the reflexes except a reduction of both abdominals and cremasterics. Tests for co-ordination are well performed. When lying in bed, the patient can touch accurately with his feet an object held in mid-air, and perform movements requiring first the one leg and then the other. Immediately after such a performance, when tested in walking, the characteristic gait was observed. No changes in sensation, either deep or superficial, are noted, and no disorder referable to the sympathetic system. The right leg is smaller in diameter than the left.

Mental State.—The patient is listless and inactive. He sits about reading newspapers or dozing in his chair. The above-mentioned untidiness in habits was noted. On one occasion the patient defæcated on the floor of the ward. He explained his action by saying that he could not get to the lavatory promptly enough. Outbursts of weeping without adequate environmental stimuli were noted on several occasions. Mild depression is the usual affect, and a peculiar childishness as regards the creature comforts (gluttonous appetite, behaviour at table, greediness, etc.) has been observed. The patient can whistle, but a true smile has never been noticed. His judgment is poor, although he expresses a constant desire to get well and return to work.

The routine laboratory studies were negative.

COMMENT.

The exact neurological classification of this syndrome is unimportant. There were periods of personality derangement, as revealed in the record (manic-depressive episodes). The history of excessive alcoholism over a long period, together with the appearance of senility, make it more than likely that the patient has a reasonable degree of cerebral arteriosclerosis. This would make the action of the gas more drastic, if the previously mentioned observation be correct.

Foix¹¹ calls this syndrome pseudobulbar palsy. Others prefer to limit the term, not permitting inclusion of a case such as the above. In view of the poor family and past history, some might doubt its organic nature. Oppenheim¹³ mentions that he has seen a case of astasia-abasia following carbon monoxide poisoning. The suggestive facies, the cog-wheel rigidity of the musculature, the manner of sitting down, and the handwriting, leave in my mind no doubt as to the organic nature of at least part of the syndrome. Speech difficulties have been noted before³. The personality change and sphincter disturbance may well be ascribed to cerebral degeneration. The astasia-abasia, however, remains a difficult symptom to explain.

Assuming a segmental arrangement in the basal ganglia, as has been frequently suggested, it is conceivable the difficulty in walking may be a manifestation of a pathological change of co-ordinating mechanisms at the leg

12/19/23.

Dr. Wolff,

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Dear Sir:

I am about to tell you that I am going to get well & better than with the assistance of you gradually. That my hope will be realized very soon for a speedy recovery.

There seems to be a general feeling of optimism of the future under your care and I trust that the results you are showing you will be realized and that you will enjoy good health yourself and also that your family are all well, and with my very kindest regards and your wishes I am.

Yours sincerely,
Frank Klebsaum
New York City, N.Y.

Fig. 1

level. Further possibilities are that the will to walk is very weak, or the resistance to be overcome in executing the act extremely great.

Kennedy, Davis and Hyslop¹² recently described vegetative changes occurring as postencephalitic symptoms, suggesting that this disease may be a factor in supplying the missing link between so-called functional and organic diseases. That volition plays a part in the symptoms of lesions of the basal ganglia is probable. Patients with severe Parkinsonian paralysis have been observed to have many variations in their clinical pictures, depending on such factors as the time of day and the emotional stress appended to an act of the will.

Foix¹¹, in his description of the autopsy findings in individuals with clinical pictures similar to the above, apparently agrees with most authors in stating that the characteristic syndrome must be the result of a bilateral lesion affecting association tracts on both sides, or of a unilateral lesion so placed as to intercept tracts on either side. Several types have been described, but the essential features are degeneration of the basal ganglia, some supraganglial and peduncular degeneration, and possibly changes in pons and medulla.

This case throws no light on the anatomical site responsible for disturbances in motion and behaviour. Most destructive processes in the brain due to gas are too diffuse to permit of fine discriminations and assignment of physiological activity to a definite area. I believe, however, that the case is of interest in suggesting that conditions commonly associated with psychogenic disturbances or other evidences of personality derangement may be expressions of an actual lesion in the brain or brainstem. At present we know little about what may be in common between the types of change that can be produced by suggestion on the one hand, and by severe arterial damage on the other.

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