finally, when the analysis was sufficiently complete, and the majority of the repressed elements had been recovered, he altogether disappeared from the scene.

The analysis of the case as a whole does not enter into the limited sphere with which my communication is alone concerned. I have, indeed, deliberately refrained from mentioning the content of any of the repressed elements or conflicts, in order not to confuse the real significance of the secondary personality. I may say, however, that the analysis was brought to a successful conclusion, and the patient has been for some time in a normal condition.

(1) Read at the Quarterly Meeting of the Medico-Psychological Association, Long Grove Asylum, February 22nd, 1912.

Aphasia in General Paralysis and the Conditions associated with it.

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CERTAIN features of the speech defects commonly seen in general paralysis bear a resemblance to aphasia, *e.g.*, inability to find required words, and the tendency to omit syllables or words without noticing it, or to transpose syllables or the vowels of successive words. More definite aphasia, however, may occur in general paralysis either as a transitory or lasting symptom. Though it may arise under a variety of conditions it is distinctly uncommon.

This paper is based on the clinical observation and *post-mortem* findings in cases of general paralysis at Long Grove Asylum. Among the subjects of the first 973 *post-mortem* examinations after the opening of Long Grove Asylum there were 273 general paralytics; 226 cases of the disease among 633 *post-mortems* on male patients, and 47 cases among 340 on females.

Aphasia may co-exist with general paralysis as a purely accidental concomitant, *e.g.*, as the result of trauma or of embolism from valvular disease. An instance of the former association of conditions is mentioned in a recent paper by MacFie Campbell (1).

Apart from such coincidences, aphasia may occur in general paralysis; (1) as a purely functional condition without demonstrable *post-mortem* lesion; (2) as a result of special localised intensity of the ordinary morbid process constituting general paralysis; (3) from subdural hæmorrhage; and (4) from focal lesions caused by arterial disease associated with general paralysis.

Aphasia is most often seen in general paralysis as a purely functional condition. It may occur thus as an isolated phenomenon or associated with right hemiplegia. Consciousness may be lost at the onset or preserved throughout. The aphasia may be of motor or sensory type—more commonly the former. Such attacks are fairly common, frequently occur early in the disease, and may even be the first symptoms noted. They have long been recognised, the first reference to them which I have been able to find being contained in a paper of Dr. Clouston, dated 1875, reporting two cases, both of the motor type (2).

The following is a typical instance of this condition:

R. P—, male, æt. 24. Syphilis denied. No physical evidence of the disease, congenital or acquired. History of somewhat severe head injury six months previously, and of an attack seven days before admission, in which speech was entirely lost and right side paralysed.

On admission the patient was dull and disorientated. Speech was drawling and slurred. Slight tremors and ataxy present; tendon jerks exaggerated. Pupils were then equal, regular, and reacted well to light and accommodation. Later both became irregular and sluggish and left larger than right.

Blood-serum exhibited positive Wassermann reaction, and cerebro-spinal fluid positive reaction to Wassermann test and lymphocytosis.

He had a series of aphasic attacks, with some confusion, but no definite loss of consciousness and no motor defect.

In these he could understand some simple orders, but tended to repeat the same action in response to successive commands. Gave his name correctly, but all other utterances, spontaneously or in response to questions, showed marked paraphasia, of which he seemed unaware. Could not repeat words nor read nor write. Could not name objects, but could pick out the one named from a collection of objects.

The condition seemed to be a moderate degree of auditory aphasia.

Mercury and potassium iodide in large doses were tried without effect.

Post-mortem.—Pia was slightly thickened, adherent all over hemispheres. Granularity of ventricles. No marked change in main vessels; no focal lesion.

In such cases the aphasia generally disappears after a period lasting from a few minutes to a few days. In rare cases, however, lasting aphasia may develop in general paralysis, and even careful microscopic examination after death may fail to reveal a corresponding lesion. The same is true of other symptoms, suggesting a focal lesion such as hemiplegia.

In the following case no such lesion was present on naked-eye examination:

H. G—, male, æt. 41. No history of syphilis. Scar in groin. Scar on left leg, suggestive of syphilitic ulceration.

On admission he was fatuous and happy. There was general enfeeblement, with clumsiness and ataxy. Tendon-jerks were much exaggerated. Pupils were equal but irregular, reacted well on convergence, not at all to light.

He understood and readily obeyed all orders. He could read and write, and kept in his pocket a piece of paper with his name written on it to show when asked. Almost the only words he ever uttered in response to questions or spontaneously were "Bits and bits."

This condition, apparently one of motor aphasia, lasted till death. He had a number of right-sided fits without loss of consciousness, followed by transitory right hemiplegia. He finally died after a similar attack.

Post mortem.—Brain showed changes of advanced general paralysis. Vessels were in good condition, and no focal lesion of any kind was found.

The second condition in general paralysis which may give rise to aphasia is a localised intensity of the processes in the cortex and pia-mater characteristic of the disease. Such cases have been mainly described by French authors (3), (4), (5). The aphasia in the cases reported has generally been permanent after a sudden onset. In some cases, *e.g.*, one described by Hanot in 1875 (6), there have been prodromal transitory attacks of the same type of aphasia, such resembling the

aphasic seizures noted above. In a case described by Serieux and Mignot, hallucinations of hearing alternated with attacks of auditory aphasia.

Aphasia may also occur in general paralysis as a result of subdural hæmorrhage. This is now a rare complication of the disease and becoming rarer. Trauma is probably a factor in its production. Dr. Mott has pointed out that the decline in its frequency in general paralytics has coincided with that of fractured ribs and hæmatoma auris in the same patients. Also that it is much more frequent in male than female general paralytics, the former being more restless. The latter fact is exemplified by our experience here. No example of the condition has been met with in a female general paralytic.

Subdural hæmorrhage seems to be found with about equal frequency in conjunction with cerebral arterio-sclerosis of the senile type and with general paralysis. In our first 973 *post-mortems* it was associated with senile arteriosclerosis six times, with general paralysis five times, and once it was found in a patient who had suffered from paralysis agitans.

Where, however, it occurs with general paralysis, marked degeneration of the main cerebral vessels may or may not be present. Such was present in two of our five cases, absent in three.

One of the general paralytics in whom subdural hæmorrhage was found *post mortem* exhibited aphasia during life.

The following is a brief abstract of this case :

S. H—, male, æt. 63. Syphilis denied. Pigmented scars on both legs. History of lightning pains.

On admission he was disorientated and apathetic. There was general feebleness and general blunting of sensation. There were general tremors, especially in voluntary movement, and constant athetoid movements of the left hand. Right knee-jerk absent, left very sluggish. Extensor response both sides. Pupils very sluggish to light, reacted well on convergence.

The articulation was typical of general paralysis. His comprehension of words was very defective ; he misunderstood much that was said to him. He occasionally used wrong words and frequently had great difficulty in expressing himself, needing to revive numerous associations before he could find a word.

He had a seizure ten days before death, after which he never regained consciousness.

Post mortem there were well-marked signs of general paralysis, atheroma of main vessels, and pachymeningitis hæmorrhagica over convexity of both hemispheres. No focal lesion of brain.

Aphasia also occurs in general paralytics from focal lesions resulting from disease of the main cerebral vessels.

Two types of such disease may be met with—syphilitic endarteritis and atheroma. MacFie Campbell in the paper mentioned insists on the frequent association of true syphilitic brain disease with general paralysis, but this combination is generally considered rare.

Atheroma of the main cerebral vessels is very frequent in general paralysis but by no means invariable, and it does not seem to be proportional to the duration of the disease, or to the severity of the other changes found in the brain. The frequency of the association probably depends on the fact that both are parasyphilitic in origin though developing independently, the general paralysis being a primary neuron degeneration.

Of the focal lesions resulting from vascular disease in general paralytics, softening from thrombosis is much more common than hæmorrhage. Gross softenings from thrombosis were found in ten general paralytics of our series, hæmorrhage only once.

In spite of the comparative frequency of such softenings in general paralysis aphasia is rare as a result, apparently rarer than when the softening depends on senile arterio-sclerosis.

In our first 973 *post-mortems* softenings resulting from the latter condition were present in 29 cases. In nine of these cases aphasia was observed, and in two others there was a history of its existence before admission. There were also three cases of softening due to embolism, of whom two exhibited aphasia. On the other hand out of ten general paralytics in whom gross softenings were found, only one exhibited lasting aphasia. In two other cases, transitory aphasic attacks occurred probably unconnected with the softenings found *post mortem*.

The difference does not seem to depend upon the size or distribution of the lesions. Possibly the explanation is that whereas softenings from senile arterio-sclerosis and from

embolism are frequently multiple, the lesion in general paralysis is often either terminal (followed by coma lasting till death), or else occurs at a stage of the disease when the mental state of the patient precludes satisfactory determination of his power to understand and to produce speech.

The following is a short summary of the case of a general paralytic who exhibited aphasia as the result of cerebral softening.

T. B—, male, æt. 52. No history of syphilis. Scar in right groin.

Condition on admission.—Confused, but not grossly demented. Paresis of right arm and leg, with spasticity and extensor response; sensation could not be satisfactorily tested. There was definite hemianopia. The pupils were equal, regular, and reacted normally at this time.

He exhibited well-marked visual aphasia and object-blindness. He could understand what was said as a rule and could spontaneously utter coherent sentences. When questioned he tended at times to repeat the same reply to successive questions.

He was unable to read and named letters wrongly, and unable to write his name or even letters with his left hand.

He was unable to recognise objects and did not know their purpose. He could not pick out from a collection of objects the one named. When told the name of an object, however, he could state its use.

For a time he improved somewhat; then had a right-sided Jacksonian convulsion followed by increase of his paresis and return to his former state of aphasia.

He had a number of such periods of improvement followed by relapse after a convulsion.

He was given large doses of potassium iodide and mercury without obvious result.

He became very demented, resistive and degraded, and developed Argyll-Robertson pupils.

Post mortem there were found: Typical lesions of advanced general paralysis, moderate atheroma of the main vessels, and an area of softening in the left hemisphere at the junction of the parietal and occipital lobes, extending from the level of the Sylvian fissure to the upper margin of the hemisphere. It involved the cortex and extended in to a depth of one-third of an inch.

The diagnosis of aphasia occurring in general paralysis may be divided into three parts: the determination, firstly, of the fact that the patient is a general paralytic; secondly, of the probable site of the focal lesion (if any) corresponding to the speech defect; and thirdly, of the nature of the pathological process causing this lesion.

The question as to whether the patient is suffering from general paralysis may be very difficult to decide clinically. The alternatives to be excluded in the cases under consideration are usually cerebral syphilis and cerebral arteriosclerosis.

A full discussion of the differential diagnosis of these three conditions is impossible here. The history may be suggestive, especially in such points as duration of symptoms, age of onset, and recency of syphilitic infection. In any of these conditions, the patient may exhibit dementia, epileptiform and apoplectiform attacks, ataxy, clumsiness and tremors, and increase of the tendon-jerks. Diminution or absence of tendon-jerks suggests general paralysis. In all the pupils may be unequal, irregular, and defective in reaction both to light and accommodation. When, however, the Argyll-Robertson phenomenon is clearly present, this is strongly in favour of general paralysis. While articulatory defects may be present in all, the type of speech present in some cases of general paralysis is almost pathognomonic.

Certain features of the mental state also may be very suggestive, especially the patient's self-satisfaction and lack of insight and of distress at his own mental defects.

At the present time the examination of the blood for the Wassermann reaction and of the cerebro-spinal fluid for the same reaction and for lymphocytosis affords valuable evidence, though not quite conclusive as between general paralysis and cerebral syphilis.

Even *post-mortem* the conditions may in some cases be impossible to distinguish without microscopic examination of the cortex, but well-marked granularity of the ventricles (especially the lower angle of the fourth) is strongly in favour of general paralysis.

In passing, I may say that in only a few of the above cases was the cortex examined histologically. In all recent cases the Wassermann reaction was employed where the clinical

diagnosis was doubtful. In all the cases mentioned as exhibiting aphasia or gross lesions accompanying general paralysis the clinical picture was quite characteristic of that disease, and *post mortem* there were present all the usual macroscopic appearances of it with none of those characteristic of cerebral syphilis.

The diagnosis of the site of the lesion from the nature of the speech defect presents nothing special beyond its difficulty in some cases owing to general dementia.

In regard to the diagnosis of the nature of the lesion causing aphasia in particular cases, one must distinguish between those cases in which aphasia is transitory and those in which it is lasting. The cause of the condition in the former is as obscure as that of all general paralytic seizures.

Of one of the conditions under which lasting aphasia is found I have no personal experience, namely, those cases where it results from a focal intensity of the diffuse process. In the reported cases, however, the onset has been sudden, and, judging from the published account, it would have been difficult or impossible to distinguish them clinically from cases with a focal lesion of vascular origin.

In cases with subdural hæmorrhage symptoms of cerebral compression sometimes exist at the onset, such as vomiting, slow pulse and respiration, choked disc on one or both sides, and contraction followed by dilatation of the pupils—the change in the latter being earlier on the side of the lesion. The resulting paralysis is frequently bilateral, and choreiform or athetoid movements in the affected limbs are relatively frequent.

In conclusion I should like to report a case presenting some points of special interest where the diagnosis is doubtful, the patient being still alive.

B. R—, female, æt. 40. No definite history of syphilis, but a history of venereal disease in husband. Several pigmented scars over lower part of back and abdomen. Has had several miscarriages and no living children.

Condition on admission.—Excitable, loquacious, self-satisfied and flippant. No motor or sensory defect except fine tremor of face and hands. Pupils unequal, irregular, inactive to light, reacted well to accommodation. Articulation normal.

Condition remained unchanged until, six months after admission, lumbar puncture was performed.

Owing to extreme restlessness a preliminary hypodermic injection of hyoscine hydrobrom. gr. $\frac{1}{100}$ and morphine hydrochlor. gr. $\frac{1}{4}$ was given. Ten c.c. of clear cerebro-spinal fluid were withdrawn and gave a positive Wassermann reaction.

She regained her normal state in a couple of hours and appeared to remain so for two days. On the third day she developed right hemiplegia, at first flaccid, later spastic. She was conscious, but unable to speak.

On examination her condition was as follows: Pupils equal and fixed; jaws tightly clenched; risus sardonicus; bilateral paresis of tongue. Occasional stertor and difficulty in swallowing; paralysis and marked spasticity of right limbs. All tendon-jerks much exaggerated, especially on right side—patellar clonus on that side. Plantar reflex, extensor on right, flexor on left; deep and universal analgesia.

She was fully conscious. She could understand what was said and obeyed simple orders. She could read, and could write with her left hand spontaneously and to dictation. No object-blindness. She could phonate, but could not articulate any word, though when asked if she knew what she wanted to say she nodded, and could indicate the number of letters in the word by holding up fingers.

There has been considerable improvement in the motor symptoms during the past two months, but the defect in speech has remained unchanged. She can phonate, bnt has uttered no articulate word since the attack. This defect seems quite out of proportion to the bulbar symptoms.

In this case the diagnosis of general paralysis is fairly certain. The type of aphasia is that called by the exponents of the classical views "subcortical motor aphasia," and by Marie and his followers "anarthria."

The main questions of interest are the site and nature of the lesion underlying the defect and its relation to the lumbar puncture.

The association of the two may, of course, be a pure coincidence. In no case have symptoms resembling those seen in this patient been described in the literature as developing after lumbar puncture.

According to a recent paper by Lusk (7) on the accidents following lumbar puncture and spinal anæsthesia, all those reported are attributable either to the paralytic effect of the

drug, or to damage to the spinal cord or cauda equina caused by the needle.

Dr. G. M. Robertson has advanced the theory that subdural hæmorrhage is started by a fall of intra-cranial pressure usually caused by spasm of the cerebral vessels. It is conceivable that the fall of the tension determined by the withdrawal of fluid might act in this way.

On the other hand, lumbar puncture has been used for the diagnosis of subdural hæmorrhage and has been said to relieve the symptoms temporarily.

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"*Forced Feeding*," *with Special Reference to a Case continuously fed by the Nasal Tube for over Nine Years.*

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THE subject of the few remarks I have to offer is one with which those who attend the insane soon become familiar. No other class of physicians can speak of it with more authority. It is not a wide field, and at the first blush would not appear to lend itself to much divergence of view. Yet from the opinions one occasionally hears expressed, I think it is expedient that our attitude towards the questions associated with artificial feeding should now and again be reviewed. When to feed artificially, its relative dangers, its moral effect, or even the best method to adopt, are points upon which observers differ.

We naturally base our opinions on the cases with which we have dealt or have actually seen; while the extent and variety

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