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Part I.—Original Articles.

Amentia and Dementia: a Clinico-Pathological Study.

By JOSEPH SHAW BOLTON, M.D., M.R.C.P., Fellow of University College, London; Senior Assistant Medical Officer, Lancaster County Asylum, Rainhill.

PART III.—DEMENTIA (*continued*).

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Group II.—Progressive and Secondary Dementia.

The present portion of this paper deals with the subject of progressive and secondary dementia. It includes the consideration of those cases of mental disease which, owing to the existence of certain extra-neuronic encephalic morbid states, do not develop a practically stationary condition of mental enfeeblement consequent on the loss of a proportion of the higher cortical neurones, but undergo a more or less rapidly progressive process of neuronic dissolution, which, if the patient survives to such a stage, finally ends in gross dementia.

As has already frequently been stated, the necessary precursor to dementia, in the opinion of the writer, is the symptom-complex which he has already exhaustively considered under the term "Mental Confusion" (*Journal of Mental Science*, July, 1906).

When referring to the causes of mental confusion, he has expressed the view that the necessary precedent to this psychic state is, in at least all severe cases, a *deficient durability of the higher cortical neurones*, which ranks, therefore, as the essential physical basis.

Further, he has dealt with the non-pathological and secondary or exciting causes of this symptom-complex, namely, the *various forms of physical and mental stress*, which, especially at the "critical" periods of life, often excite morbid changes in cortical neurones of deficient durability, although they would be relatively or absolutely without prejudicial influence on normal cortical neurones. This factor merely causes temporary mental disturbances (unassociated with any considerable degree of mental confusion) in such individuals as possess cortical neurones of average durability but of subnormal or abnormal development and of imperfect functional stability.

Lastly, he has referred to the chief exciting or secondary causes of mental confusion which are pathological in nature, and has classed these into two groups. In the first of these groups has been considered the *direct action of toxines*, especially alcoholic excess and the toxæmia which frequently follows childbirth, but also, though more rarely, the different toxæmias and infections.

Whether the cases of mental confusion which arise in consequence of the action of one or more of these causative agents

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recover, or develop a mild or moderate grade of dementia, depends on the resistance of the affected neurones and on the extent and severity of the pathological changes which are produced. In the latter case, the result is an example of the "primarily neuronic dementia" which has been considered in the last division of this paper, and the patient may remain in a stationary condition of mild or moderate mental enfeeblement for many years, or even for life.

In the case of the second group of pathological and secondary causes of mental confusion the results are different, and it is to the consideration of these that the present portion of this paper is devoted. These causes are, in essence, consequences of the *indirect action of toxines*, which results in imperfect nutrition of the cortical neurones and therefore tends to interfere with their vitality and functional stability.

This indirect action of toxines will be considered under two main headings, the contents of which present much similarity as regards pathology but differ considerably in details of procedure and in symptomatological consequences. They are responsible respectively for the types of case described under the terms "Progressive Senile Dementia" and "Dementia Paralytica." In both groups pathological changes exist in the cerebral vessels. These changes in the first group are chiefly of a degenerative or "wearing out" nature, and in the second are partly degenerative and partly of the nature of a reparative reaction. In the first the vascular changes are chiefly the result of natural decay, and in the second they are largely the consequences of an enhanced capacity of reparative reaction which is due to the previous occurrence of a severe and prolonged systemic toxæmia. In the first group general non-neuronic reparative reaction is feeble, and in the second it is variable, and in many cases very marked. In both groups the effect on the neurones is partly caused by imperfect nutrition and partly by secondary toxæmia. In both groups there is a tendency to the formation of a "vicious circle," but in the second this is much the more marked owing to the greater capacity of reparative reaction which exists. In the first group the patients are senile or presenile; in the second they are usually adult, but may be of any age from puberty to advanced senility.

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Group A.—Degeneration of the Cerebral Vessels accompanying Senility or Premature Senility.

As has been shown in the first part of this paper and also in greater detail in a previous paper (*Archives of Neurology*, vol. ii), there is a direct relationship between the presence of degeneration of the cerebral vessels and the development of severe dementia.

The chief facts bearing on this relationship are as follows:

(1) Simple senility (*i.e.*, old age) is not necessarily associated with gross degeneration of the cerebral vessels.

(2) In the insane, gross degeneration of the cerebral vessels may exist without dementia.

(3) Dementia, except in rare cases of slowly progressive presenile involution of the cortical neurones, does not progress beyond a moderate stage, if gross degeneration of the cerebral vessels does not coexist.

(4) In the 200 cases of Series A, and in the 233 cases of Series B, the percentage amount and also the severity of naked-eye degeneration of the cerebral vessels vary directly with the degree of dementia present.

(5) Severe degeneration of the cerebral vessels occurs before the development of gross dementia. In recent senile cases, with the mildest grade of dementia, but with considerable mental confusion, which, had they lived, would on clinical grounds have been expected to develop gross dementia, the percentage of naked-eye degeneration of the cerebral vessels is as great as it is in Groups IV and V (severe and gross dementia). On the other hand, in chronic and recurrent senile cases, with a mild grade only of dementia, naked-eye degeneration of the cerebral vessels is rarely present and is then relatively slight.

Hence the relationship between the presence of degeneration of the cerebral vessels and the development of dementia may be thus summed up: *In a cerebrum which has begun to break down, or where degeneration has progressed to the "moderate" stage,* (Group III, the chronic lunatic with moderate stationary dementia), *the presence or incidence of gross degeneration of the cerebral vessels will cause more or less rapid progress of the neuronic dissolution, with resulting gross dementia.*

In such cases the pathological process in the neurones is

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caused on the one hand by imperfect nutrition and on the other by secondary intoxication from incomplete removal of the waste products of metabolism and dissolution, and relatively little encephalic extra-neuronic reparative reaction occurs owing to the degenerative or "wearing out" nature of the whole process.

The dementia which supervenes progresses rapidly or slowly until death occurs.

It may be added that, whilst "wearing out" of the cerebral arteries ensues at different ages in different individuals, and as a rule only occurs when old age is reached, the premature induction of this by such devitalising agents as prolonged alcoholic excess and by organic affections, particularly of the heart and kidneys, is fairly common. The writer is of the opinion that usually rather more extra-neuronic reparative reaction occurs in the latter cases than in those in which the "wearing out" is due to simple senile decay.

Group B.—Certain Vascular and Neuroglial (and chiefly Secondarily Neuronic) Changes which Follow the Prolonged Action of Toxines, etc.

These appear to be largely of the nature of secondary proliferation after, or of reaction to the injury produced by the poison, toxine or pathogenic micro-organism; and their onset, in the opinion of the writer, is not necessarily coincident in time with its exhibition, but ensues as the result of adverse influences occurring at any subsequent period of life. That this statement is in accord with general pathology can readily be illustrated. Prolonged immunity is common after many of the severe specific infections, which induce profound and more or less permanent protective modifications of general metabolism. Excessive local reparative reaction often occurs after diphtheria, scarlet fever, and syphilis, and results in intractable strictures of orifices. On the other hand, a similar local reparative reaction in the arteries after an attack of syphilis is later on followed by dilatation and the formation of aneurysms. Further, injuries in the subjects of former syphilis frequently result in the occurrence of excessive local reparative reaction, and dense fibrous scars often follow abscesses, vaccination, etc., in the case of such persons. This

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fact is as readily explicable on the ground that the tissues, in consequence of a former attack of syphilis, possess a permanently enhanced capacity for reparative reaction to injury, as on the commonly accepted thesis that the *Spirochæta pallida* still exists in the body, after perhaps as long a period as twenty-five years.

The chief variety of mental disease which falls under the above heading is the dementia paralytica (general paralysis) which is a frequent sequela of systemic syphilis in degenerates and which rapidly or slowly passes on to a fatal issue.

As various authors give the percentage of ascertained previous syphilis in cases of dementia paralytica as anything from 50 or less to 100, and as several writers deny any direct causal relationship between syphilis and dementia paralytica, it would be futile to introduce such diverse and extraneous conclusions into this paper. The writer therefore purposes to confine himself to the repetition of his own previously published statistics on the subject, and especially so as prolonged experience has convinced him of their substantial accuracy.

These statistics deal with 19 private and 83 rate-paid patients, in the case of whom, in the course of a systematic inquiry into their histories, he was able to obtain trustworthy personal details.

In 15 of the 19 private cases there was a history or clear clinical evidence of former syphilis, and the date of infection, where it could be ascertained, was from four to twenty-five years before the onset of mental symptoms. The following details were obtained concerning the remaining four cases:

CASE 3.—Tabetic general paralysis. Was twelve years in the Army, and was then in the police force. Had been married for twelve years without children.

CASE 4.—Tabetic general paralysis. Was an Indian Government official for over thirty years, and at the age of thirty-seven married a half-breed, with whom he lived a jealous and unhappy life.

CASE 8.—Patient stated that he had had several gonorrhœas and orchitis in each testicle on separate occasions.

CASE 15.—Patient stated that he had had several gonorrhœas and gleets, and had also suffered from orchitis.

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Hence, of the 19 cases, syphilis was certain in 15 (79 *per cent.*), and probable in the remaining 4 (21 *per cent.*).

Of the 83 rate-paid cases, syphilis was proved to have existed in 59. The information was obtained from the histories, or from clinical or *post-mortem* evidence, and in some instances from all these sources. Syphilis had also probably existed in another 11 cases, there was no evidence for or against in 11, and it was definitely denied by the relatives in 2 cases. In the latter cases the only evidences against the disease were the direct negatives of the friends and the absence of clinical signs. In other cases where syphilis was proved to have existed an equally definite denial was given by the friends. Of the 59 cases in which it had certainly existed, it was probably "congenital" in 4, and was probably acquired after puberty in the remainder. Where the information was available, the date of syphilisation varied from nine to twenty-five years before the onset of dementia paralytica.

Hence, of the 72 cases which it is possible to employ, syphilis had existed in 59 (82 *per cent.*), and had probably existed in 11 (15 *per cent.*).

The writer therefore considers himself justified in concluding that syphilis is a necessary antecedent to, and is causally related to the development of dementia paralytica.

On the basis of this conclusion it is possible to demonstrate that the course taken by cases of dementia paralytica depends largely on their respective degrees of cerebral degeneracy, and also, as will be shown later in this section, that dementia paralytica is not a special organic disease of the cerebrum, but is a branch of ordinary mental disease.

In the under-developed and poorly-constructed neurones of the imbecile variety of juvenile general paralysis, the process of dissolution is slow, and the neuronic changes, as has been shown by Watson, are proportionately more extensive than are the vascular and neuroglial.

On the other hand, in the better-developed cerebra of the ordinary juvenile general paralytic, who is infected with syphilis at birth or thereabouts, the process of dissolution is more rapid, and vascular and neuroglial proliferation is more pronounced.

Further, in adult cases of general paralysis the course is usually chronic in degenerates, who readily break down under the influence of external "stress," and who, therefore, require

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early segregation, with the consequent relative absence of this factor; and it is commonly more rapid in the less degenerate subjects, who, before breakdown occurs, are frequently subjected to the severest forms of mental and physical "stress," and whose neurones are therefore strained to the utmost before asylum *régime* becomes necessary. In both these types, as the syphilitic infection at the time of its occurrence had acted on already developed neurones, and therefore had not induced still further developmental disabilities in these, vascular and neuroglial proliferation is pronounced.

Finally, in senile cases of general paralysis, in which reparative reaction is naturally more feeble, the course of the process of dissolution is variable, and the general type of the symptomatology and of the morbid anatomy and histology approximates towards that which exists in progressive senile dementia.

Though a former attack of syphilis, as has been stated, is usually the important extraneous factor in the production of progressive (secondary) non-senile dissolution of the higher neurones of the cerebrum, and is responsible for the development of the clinical entity termed "dementia paralytica," other influences, particularly certain of the slowly-acting metallic poisons—*e.g.*, lead—produce a progressive cerebral dissolution of similar character.

Further, of the insane who are the subjects of epilepsy, about 25 *per cent.* suffer from a similar progressive disintegration of the higher neurones of the cerebrum, which, in well-marked cases, presents a clinical symptomatology and a morbid anatomy which in many important details resemble those existing in dementia paralytica.

It may be added that such devitalising factors as prolonged alcoholic excess, etc., play an important secondary part in the development of many of the cases referred to under this heading, by producing morbid changes, not only in the higher neurones of the cortex, but also in the cerebral blood-vessels.

As dementia paralytica consists in essence, as will be seen later, of a dissolution of the (human) centre of higher association, it is necessarily impossible to reproduce this clinico-pathological entity by experiment on the lower animals. Watson has, however, shown that the prolonged exhibition of certain virulent neurone toxines—*e.g.*, abrin and ricin—produces in the cerebrum

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of the guinea-pig or rabbit dissolution of cortical neurones and proliferation of the neuroglia and blood-vessels. There is no doubt that the former is the direct result of neurone intoxication, and that the latter is a reparative reaction to the injury produced. Though such experimental results present no true homology to human dementia paralytica and progressive dementia, they nevertheless indicate that the non-neuronic elements of the encephalon react to neurone destruction, as do the local mesoblastic elements of other parts of the body to destruction of glandular epithelium. From this aspect they are therefore of great importance in that they experimentally support the soundness of the thesis, based on grounds of general pathology and advocated by the writer in the present and previous papers, that the morbid process in dementia paralytica is the pathological ally of that occurring in the different types of progressive dementia, and is, in essence, in no way dissimilar (differences in structure and function being allowed for), from the morbid process which occurs in, for example, certain forms of renal cirrhosis.

It may further be added that the results of these experiments indicate the likelihood that the morbid process in acute or advanced cases of dementia paralytica may be much aggravated by such secondary microbic invasions as necessarily occur owing to the decreased resistance of such patients to the attack of organisms, which, under normal conditions, might not be pathogenic.

The cases belonging to the present group of "progressive and secondary dementia" amount to 47 only, thus forming 10·6 *per cent.* of the total of 445 cases of dementia, and 6·5 *per cent.* of the total of 728 cases of amentia and dementia.

They will be divided in accordance with the pathological considerations just adduced, into the following classes:

	M.	F.	T.
Class (*a*).—Progressive senile dementia .	9	15	24
Class (*b*).—Dementia paralytica . . .	14	9	23
Total	23	24	47

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Class (A).

Progressive Senile Dementia.

The cases to be referred to under the term "Progressive Senile Dementia" differ from the contents of the preceding group of "Primarily Neuronic Dementia" in the fact, as has already been indicated, that the dementia is not stationary, but progresses rapidly or slowly until death occurs.

In the case of the preceding group, as the result of morbid changes in the higher neurones of the cerebral cortex in association with (acute) symptoms of mental alienation and such a degree of mental confusion as is the necessary concomitant of these morbid changes, a certain degree of neuronic dissolution results. This finds its symptomatological expression in a grade of dementia which varies in degree from "mild" to "moderate," and it exhibits from the physical aspect certain intra-cranial morbid changes which have been described in the first part of this paper (*Journal of Mental Science*, April, 1905), under Groups II and III, namely "cases with slight morbid changes and where the pia-arachnoid strips rather more readily than natural," and "cases with moderate morbid changes, with subdural excess to the level of the tentorium, and where the pia-arachnoid strips readily." Such cases, as the acute morbid changes, which constitute the physical basis of the "acute" symptoms presented, result in the maiming or death of numbers of the affected higher cortical neurones, pass into, and then for long periods remain in, a stationary condition of mild or moderate dementia.

In the case, however, of the class at present under consideration, that of progressive senile dementia, no such stationary condition of dementia ensues, but dissolution of the higher neurones of the cortex progresses more or less rapidly until the centre of higher association is practically non-existent, extensive dissolution of many of the regions of lower association has resulted, and the patient is consequently in a condition of gross dementia.

This result is due, as has already been shown in summary at the commencement of this section and demonstrated at length in the first part of the present and also in a previous paper, to the existence of gross degeneration of the cerebral vessels. In some

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cases this morbid condition is present at the time of onset of the attack of insanity, and in others gross degeneration of the cerebral vessels gradually develops in stationary cases of moderate dementia. In all such cases, however, whether the cerebrum is beginning to break down or dissolution has already progressed to the "moderate" stage (Group III), the determining cause of a more or less rapidly progressive dissolution of the centre of higher association is the presence or incidence of gross degeneration of the cerebral arteries.

Whilst, as a rule, at any rate in comparison with dementia paralytica, relatively little reparative reaction occurs in the extra-neuronic elements of the encephalon owing to the degenerative or "wearing out" nature of the whole process, in many cases a "vicious circle," similar to that commonly occurring in dementia paralytica, undoubtedly develops—neuronic dissolution being followed by reparative reaction and this by further and secondary neuronic dissolution—and increases the rapidity with which the final result is attained. The progress of the dissolution is also in many cases assisted by temporary and local thromboses, which frequently find symptomatological expression in "seizures" accompanied by temporary paresis and homologous, in the opinion of the writer, with the "seizures" which so commonly occur in dementia paralytica.

Progressive senile dementia thus differs markedly, both in its pathology and in its termination, from the types of dementia which have already been considered. As, however, the essential feature of progressive senile dementia is a senile dissolution of the higher neurones and of many of the lower neurones of the cortex cerebri, the correctness of the inclusion of this type of dementia under the terms "mental disease," or "insanity," may be taken for granted.

It is, however, necessary, as will be seen later, to adopt a different course in the section dealing with dementia paralytica. It will consequently be found that the greater portion of the section referred to deals with evidence which, in the view of the writer, conclusively shows that dementia paralytica is also a branch of insanity or mental disease, and is not a specific organic disease of the cerebrum. In other words, whilst progressive senile dementia requires no justification for its inclusion in the present group of "Progressive and Secondary

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Dementia," such justification is needed, and will be produced, in the case of dementia paralytica.

Certain of the more important features of the morbid anatomy, pathology, and symptomatology of progressive senile dementia will now be referred to. As, however, the morbid anatomy of mental disease has already been considered at length in Part I (*Journal of Mental Science*, April, 1905), and in Part II (*Journal of Mental Science*, April, 1906), and as the symptomatology of mental confusion and its relationship to that of dementia have been fully discussed in Part II (*Journal of Mental Science*, July, 1906), only those details of morbid anatomy and symptomatology in which progressive senile dementia differs from primarily neuronic dementia will be introduced.

Morbid Anatomy and Pathology of Progressive Senile Dementia.

Though naked-eye *degeneration of the cerebral arteries* is not one of the morbid changes which necessarily occurs in primarily neuronic dementia, it is, as has already been stated, a necessary factor to the development of progressive senile dementia. Cerebral vascular degeneration may exist in the absence of dementia. Though evidence of senility or prematurely produced senility of the cerebral arteries, it is not a necessary consequent of old age. On the other hand, the grosser forms of dementia never exist in the absence of macroscopic, or, at the least, of microscopic, signs of severe degeneration of the cerebral arteries, even in cases which have not attained to the senile period of life. Finally, in recent senile cases, with the mildest dementia but considerable mental confusion, which, had they lived, would on clinical grounds have been expected to develop gross dementia, the percentage of naked-eye degeneration of the cerebral vessels is so high as to justify the assumption that, were it possible to invariably make a certain diagnosis, this morbid change would be found to be a constant feature of such cases.

Such, in brief, are the chief facts on which is based the conclusion that a causal relationship exists between degeneration of the cerebral arteries and the development of the grosser forms of dementia; and the writer therefore places degeneration of the cerebral arteries first on the list of the morbid appearances which are found in senile progressive dementia.

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As has been pointed out in the first part of this paper, the intra-cranial morbid appearances which are found in such cases of mental disease as during life exhibited a greater or a lesser amount of dementia, namely, chronic degeneration and fibrosis of the dura mater, excess of intra-cranial fluid, subdural deposits, chronic thickening of the pia-arachnoid, etc., are the macroscopic equivalents of, and vary in degree with, the grade of dementia which is present, and are otherwise independent of the duration of the insanity.

In the several types of "primarily neuronic dementia," such morbid appearances, in agreement with the amount of dementia, are not as a rule present in more than a moderate grade of severity (Group III). On the other hand, in the two classes of "progressive and secondary dementia," namely, "progressive senile dementia" and "dementia paralytica," these morbid appearances in advanced cases attain their maximum intensity (Groups IV and V), in association with the existence of gross dementia and more or less complete dissolution of the cortical neurones of higher association and of many of those of lower association.

These morbid appearances are the physiological results of the loss of cerebral substance, caused by the degeneration of the cortical neurones, which is the physical expression of dementia, reacting on the mechanical conditions existing within the cranial cavity. The skull is a closed bony chamber, and were the neuronic dissolution ever so slow in its progress, replacement of the lost cerebral tissue could not well be fully performed by a chronic hypertrophy of the inner wall of the skull-cap and of the cerebral membranes. The progress of neuronic dissolution is, as a rule, however, by no means slow, and in cases of progressive dementia it is relatively rapid, and often very rapid. In consequence of this, the cerebral membranes, especially the pia-arachnoid, make a hopeless attempt at the formation of replacement or scar-tissue, and what space cannot be filled up in this way is replaced by cerebro-spinal fluid.

The writer feels that he cannot too strongly or too frequently insist on the importance of *excess of intra-cranial fluid* in the pathology of dementia. This excess is so commonly neglected in descriptions of intra-cranial morbid changes in favour of gross or fine changes in the dura mater, the pia-arachnoid, or the cerebrum, that it might almost be supposed to be value-

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less as a criterion of the degree of cerebral wasting which is present.

Under normal conditions, as has been shown by Leonard Hill, the intra-cranial fluid is minimal in amount, and this is also the case in all types of uncomplicated amentia or cerebral sub-evolution.

In cases, however, in which but a moderate grade of dementia exists, there is in the majority of cases such an excess of intra-cranial fluid as extends up to, or even above, the level of the tentorium, as well as considerable cerebro-spinal fluid in the pia-arachnoid and the ventricles; and a much greater excess exists in cases of progressive dementia, even when these are only reasonably advanced.

In the case of sub-dural excess alone in senile progressive dementia, for example, in the ninety-two cases contained in Group IV and the seventy-nine cases contained in Group V, which are referred to in the first part of this paper, excess of subdural fluid exists in all. In Group IV it is "slight" in 5·4 *per cent.*, "moderate" (*i.e.*, to the level of the tentorium) in 27·2 *per cent.*, and "great" in 67·4 *per cent.*; and in Group V it is "moderate" in 17·7 *per cent.* and "great" in 82·3 *per cent.*

This excess of intra-cranial fluid, which primarily occurs to replace loss of cerebral substance in the closed bony chamber, interferes with the normal relationship of the pia-arachnoid to the dura mater, and converts a potential space into an actual one full of cerebro-spinal fluid. This fluid, which is often abnormal in composition, necessarily predisposes to the development of a chronic degenerative process in both the dura mater and the pia-arachnoid, as does also the hopeless attempt at the formation of replacement or scar-tissue which is made by these membranes. Hence, any more or less sudden alteration of intra-cranial tension, due, *e.g.*, to a convulsion, a trauma, etc., or even to the change in blood-content from the arterial to the venous side, which occurs at or shortly after death, tends to cause an effusion of blood from the degenerate and often dilated vessels (arteries or veins) of the dura mater, the pia-arachnoid, or both. This effusion, whether recent or partially organised, single or multiple, constitutes the "*sub-dural deposit*," which is so relatively common in cases of well-marked dementia, and particularly so in cases of advanced progressive dementia.

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In the 433 cases referred to in the first part of this paper, for example, in Group I (no dementia), sub-dural deposits existed in 3·1 *per cent.*, and in Group II (slight dementia) they existed in 5·2 *per cent.* All these deposits were of an accidental nature or were recent and sufficiently explicable on general pathological grounds by the cause and mode of death.

In Group III (moderate dementia) these deposits existed in 17·8 *per cent.* of the cases; in Group IV (severe dementia) they existed in 17·4 *per cent.*, and in Group V (gross dementia) they existed in no less than 22·8 *per cent.* of the cases.

Extensive morbid changes in the pia-arachnoid are a constant feature in progressive senile dementia, and the relative severity of these is well illustrated by the following data.

In cases without dementia (Group I) the pia-arachnoid, except in cases of cerebral œdema due to systemic causes, strips naturally. In cases of mild dementia (Group II) this membrane is slightly thickened and strips rather more readily than natural in 74 *per cent.* of the cases, and readily in another 20 *per cent.* In cases of moderate dementia (Group III) it is thickened and at times slightly opaque, and it strips readily in 82 *per cent.* of the cases and very readily in another 16 *per cent.* In cases of severe dementia (Group IV) it is opaque and much thickened and it strips readily in 13 *per cent.* of the cases, very readily in 83 *per cent.* and like a glove in 4 *per cent.* Finally, in cases of gross dementia (Group V), it is very opaque and markedly thickened, and it strips readily in 1 *per cent.*, very readily in 41 *per cent.*, and like a glove in 58 *per cent.*

The final important morbid appearance in senile progressive dementia, namely *cerebral wasting*, will now be considered.

It is usual, even in relatively recent cases in which incomplete removal of the products of neuronic dissolution has occurred, to find the cerebral wasting quite pronounced, and this is still more evident in cases of chronic type. In many of the latter, however, before the hemispheres have been stripped, the wasting is by no means evident, in consequence of the opacity of, and still more, of fibrotic contraction of the pia-arachnoid. The difference in the appearance of a hemisphere before and after stripping is, in fact, in many cases, quite remarkable.

Whilst individual variations in the relative degrees of wasting exist, which may by future study be associated with differences

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in symptomatology, the regions of wasting are on the whole very definite, and by practice can be determined with considerable accuracy.

In uncomplicated cases there is a clear relationship between the grade of dementia and the degree of wasting present, and, therefore, cases of progressive senile dementia, as a rule, present the most clearly-marked examples of the cortical wasting which has developed *pari passu* with dissolution of the higher neurones of the cerebrum.

These regions of wasting are as follows:

(1) The greatest amount occurs in the prefrontal region (the anterior two-thirds or so of the first and second frontal convolutions, including the neighbouring mesial surface, and the anterior third or so of the third frontal convolution).

(2) The wasting is next most marked in the remainder of the first and second frontal convolutions. [In dementia paralytica Broca's convolution should, as a rule, be included here, and (2) and (3) should follow (4)].

(3) It is, perhaps, next most marked in the ascending frontal and Broca'sconvolutions, though this grade should, in many cases at least, follow (4).

(4) It is next most marked in the first temporal convolution and the insula, and in the superior and inferior parietal lobules. In practically all cases it is more marked in the two former than in the two latter.

(5) It is least marked in the remainder of the cerebrum (including the orbital surface of the frontal lobes), particularly the inferio-internal aspect of the temporo-sphenoidal lobe and the posterior pole of the hemisphere.

In the experience of the writer exceptions to this general order are invariably due to vascular or traumatic causes, and should, therefore, be excluded from the ordinary and normal wastings of dementia.

Such exceptions, however, occur not uncommonly in progressive senile dementia, owing to the extensive degeneration of the cortical arteries, which is a constant feature of these cases. The writer here refers not to definite old or recent softenings, but to more or less extensive atrophies of convolutions, which commonly exhibit vermiform or cross-striated markings, and are obviously due to local ischæmias in the distribution of (chiefly) the anterior and middle cerebral arteries. Such

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exceptional regions of wasting are, however, quite readily separable from the normal wasting caused by dissolution or retrogression of the centre of higher association.

They are chiefly found in cases in which acute exacerbations of symptoms, in the form of severe mental confusion with or without convulsions followed by temporary paresis, have occurred; and they are usually absent from cases which have undergone a steady progress to gross dementia. Further, these local wastings are, in the experience of the writer, absent from cases which have for years exhibited stereotyped and repeated motor phenomena, and from the occasional cases of Huntington's chorea which have come under his observation. It is probable that such motor exhibitions are homologous with such normal phenomena of senility as lower jaw and manual movements.

Cases presenting such local atrophies form, in fact, a half-way house between cases of ordinary gross dementia and cases of gross dementia which also exhibit gross lesions of the cerebrum of vascular origin.

The regions of wasting, which have been described above will now be further demonstrated by means of illustrative cases.

On Plate IV are exhibited photographs of two hemispheres from well-marked cases of progressive senile dementia. In both instances the regions of wasting are obvious, but they are especially evident when the figures are compared with the illustrations on Plates I, II, and III. It may be remarked that all the hemispheres illustrated on the plates are of exactly the same relative size.

On Plate I are shown the small and very simply convoluted hemispheres of a case of imbecility with epilepsy. In this case there were no dementia, no cerebral wasting, and no excess of intra-cranial fluid, and the stripped right and left hemispheres weighed respectively 475 and 470 grm.

On Plate II are illustrated the very small and simply convoluted hemispheres of a normal degenerate who died of the secondary intra-cranial effects of middle-ear disease. In this case there were no dementia, no cerebral wasting, and no excess of intra-cranial fluid. The stripped hemispheres weighed but 430 grm. each, a weight which is almost incompatible with continued existence outside an asylum. This patient would probably, in fact, have died at home or in a hospital had he possessed a cerebrum large enough to withstand the stress of

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PLATE I. Fig. 1.

Cerebrum small and very simply convoluted; no morbid appearances.
(*Group I.*)

Photograph of the hemispheres of a case of imbecility with epilepsy. The hemispheres are small and very simply convoluted, but are otherwise of normal appearance.

History.—Male, æt. 47, single, organ blower. He is stated to have got on fairly well at school, and to have suffered from fits since the age of fifteen years. In Claybury Asylum during the last five years of his life. He was simple and childish, and unable to give a connected account of himself. He was unintelligent, and possessed deficient reasoning powers. He was untidy in his appearance, and took little interest in his surroundings.

Post-mortem.—Dura and S.D.: Natural; no excess. Pia: Natural; strips naturally. S.A.: No excess. Vents.: L., normal; IV, a few granulations in the lateral sacs. Vessels: Natural. Encephalon: 1,143 grm. Cerebellum, etc.: 163 grm. R.H.: 485 grm.; stripped 475 grm. L.H.: 490 grm.; stripped 470 grm. Cause of death: Congestion of right lung; cardiac failure.

[*Note.*—The illustrations on this and the following plates are all of exactly the same relative size.]

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PLATE I.

JOURNAL OF MENTAL SCIENCE, JANUARY, 1908.

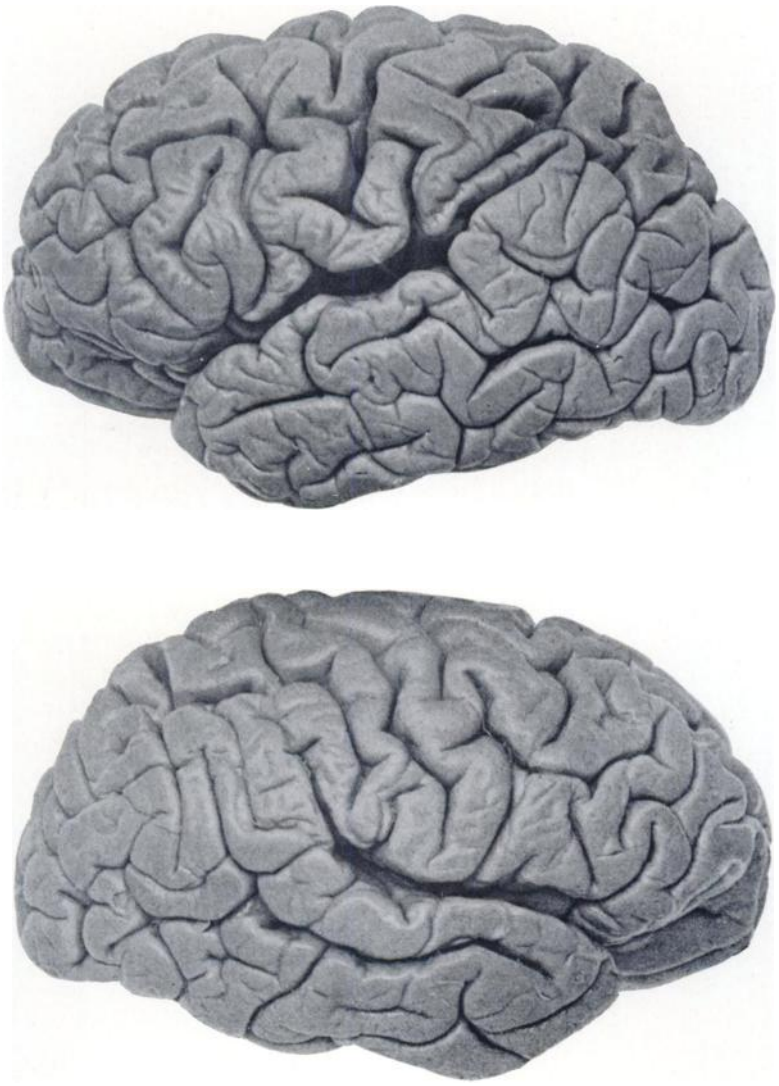

FIG. 1.

To illustrate Dr. J. S. BOLTON's paper.

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PLATE III. Fig. 3.

Cerebrum very large and of an exceedingly complex convolutional pattern; morbid appearances slight. (*Group II.*)

Photograph of the left hemisphere of a case of presenile melancholia with mild dementia. The brain was very large, and the convolutional pattern of the hemisphere illustrated is exceptionally complex. Little or no wasting is visible in the photograph.

History.—Male, æt. 51, merchant. Causes of attack are stated to be heredity of insanity, influenza, and business worries. In Claybury Asylum three and a half months. Patient was a man of considerable intelligence. On admission he had a haggard and anxious expression, and was extremely depressed. He replied to questions slowly and with reluctance. He was very introspective. He was much worried by "voices," but could not tell what they said. He stated that he would like to die, as he had lost large sums of money and was tired of life. He was very unwilling to give any account of himself. He continued depressed, listless, and taciturn, lost flesh, and soon began to refuse his food. Eight weeks after admission he was fed daily by tube for some weeks, and on every occasion he strongly resisted. He then began to feed himself, and stated that he had refused his food owing to the fear of being poisoned. He rapidly lost flesh almost from his admission to the asylum, and died after three and a half months' residence.

Post-mortem.—Dura and S.D.: Natural; no excess. Pia: Slight fronto-parietal milkiness and thickening. S.A.: Slight excess. Vents.: L., natural; IV, granulations in the lateral sacs. Vessels: Natural. Encephalon: 1,645 grm. Cerebellum, etc.: 205 grm. R.H.: 720 grm. L.H.: 705 grm.; stripped 680 grm. Cause of death: Cardiac failure, hypostatic pneumonia. The body was extremely emaciated.

PLATE II.

JOURNAL OF MENTAL SCIENCE, JANUARY, 1908.

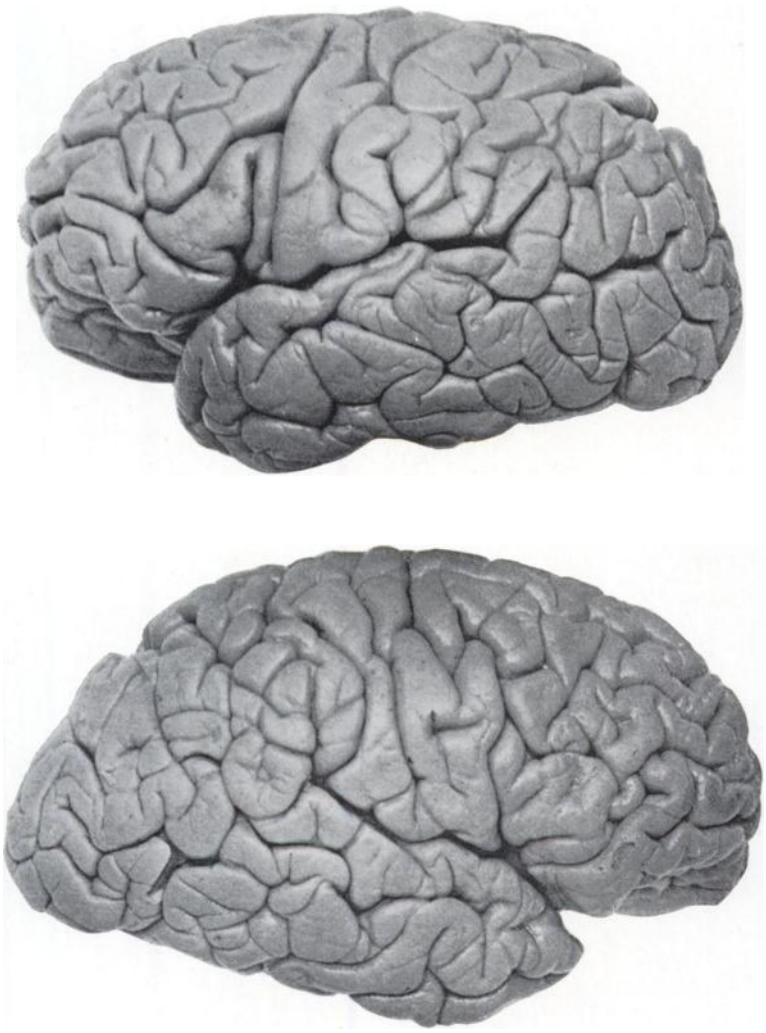

FIG. 2.

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PLATE II. Fig. 2.

Cerebrum simply convoluted and very small; no morbid appearances beyond œdema due to the local disease. (*Group I.*)

Photograph of the hemispheres of a case of extra-dural and cerebellar abscess. The hemispheres are very small; the right is fairly and the left simply convoluted. The brain is otherwise normal in appearance.

History.—Male, æt. 39. Father suffered from paralysis. Married nineteen years, eight children alive. Had lead poisoning six years ago. He exhibited symptoms for a month before, and died thirteen days after his admission to Claybury Asylum. He exhibited physical symptoms which suggested general paralysis. He was confused, helpless, and defective in his habits. He had a convulsion five days after his admission.

Post-mortem.—Dura and S.D.: Natural; no excess. Pia: Much œdema; strips like a glove everywhere. S.A.: No excess. Vents.: L., slightly dilated; IV, granulations in lateral sacs. Vessels: Natural. Encephalon: 855 grm. Cerebellum, etc.: 125 grm. R.H.: 430 grm.; stripped 408 grm. L.H.: 430 grm.; stripped 410 grm. Cause of death: (*a*) Pneumonia; (*b*) abscess of cerebellum, local meningitis, extra-dural abscess, necrosis of petrous bone, middle-ear disease.

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PLATE III. Fig. 3.

Cerebrum very large and of an exceedingly complex convolutional pattern; morbid appearances slight. (*Group II.*)

Photograph of the left hemisphere of a case of presenile melancholia with mild dementia. The brain was very large, and the convolutional pattern of the hemisphere illustrated is exceptionally complex. Little or no wasting is visible in the photograph.

History.—Male, æt. 51, merchant. Causes of attack are stated to be heredity of insanity, influenza, and business worries. In Claybury Asylum three and a half months. Patient was a man of considerable intelligence. On admission he had a haggard and anxious expression, and was extremely depressed. He replied to questions slowly and with reluctance. He was very introspective. He was much worried by "voices," but could not tell what they said. He stated that he would like to die, as he had lost large sums of money and was tired of life. He was very unwilling to give any account of himself. He continued depressed, listless, and taciturn, lost flesh, and soon began to refuse his food. Eight weeks after admission he was fed daily by tube for some weeks, and on every occasion he strongly resisted. He then began to feed himself, and stated that he had refused his food owing to the fear of being poisoned. He rapidly lost flesh almost from his admission to the asylum, and died after three and a half months' residence.

Post-mortem.—Dura and S.D.: Natural; no excess. Pia: Slight fronto-parietal milkiness and thickening. S.A.: Slight excess. Vents.: L., natural; IV, granulations in the lateral sacs. Vessels: Natural. Encephalon: 1,645 grm. Cerebellum, etc.: 205 grm. R.H.: 720 grm. L.H.: 705 grm.; stripped 680 grm. Cause of death: Cardiac failure, hypostatic pneumonia. The body was extremely emaciated.

PLATE III.

JOURNAL OF MENTAL SCIENCE, JANUARY, 1908.

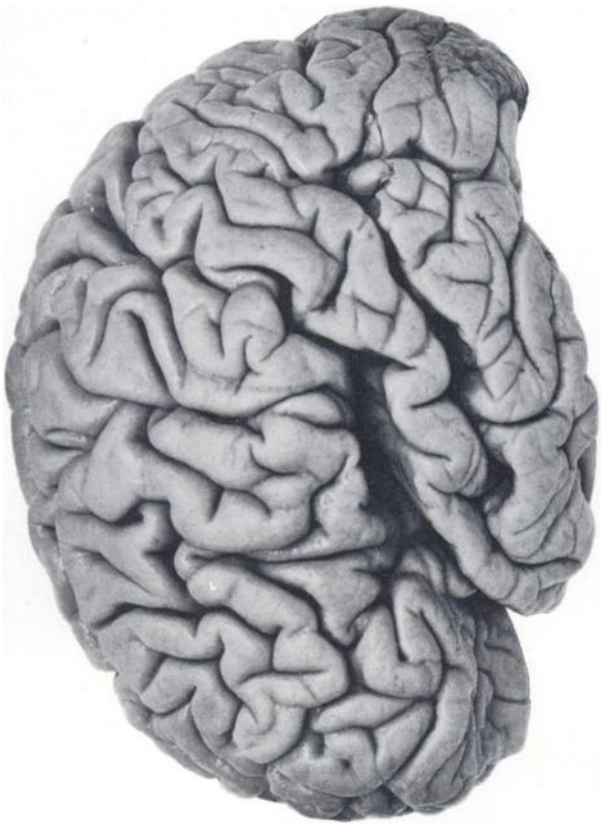

FIG. 3.

To illustrate Dr. J. S. BOLTON's paper.

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PLATE IV.

JOURNAL OF MENTAL SCIENCE, JANUARY, 1908.

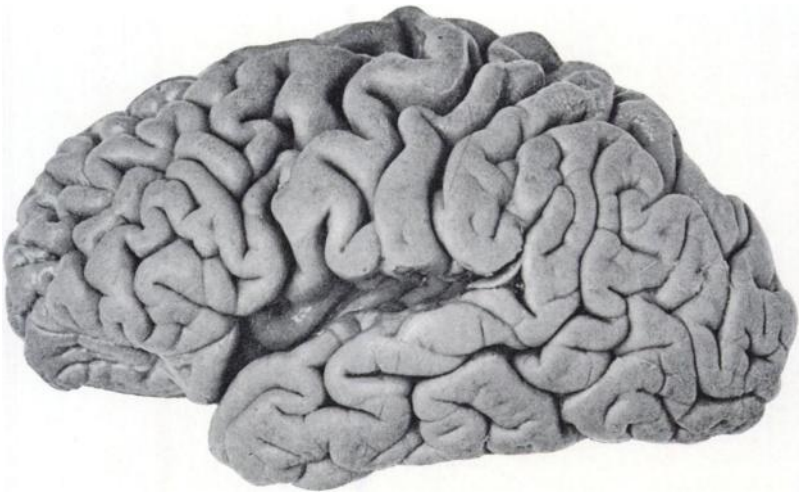

FIG. 4.

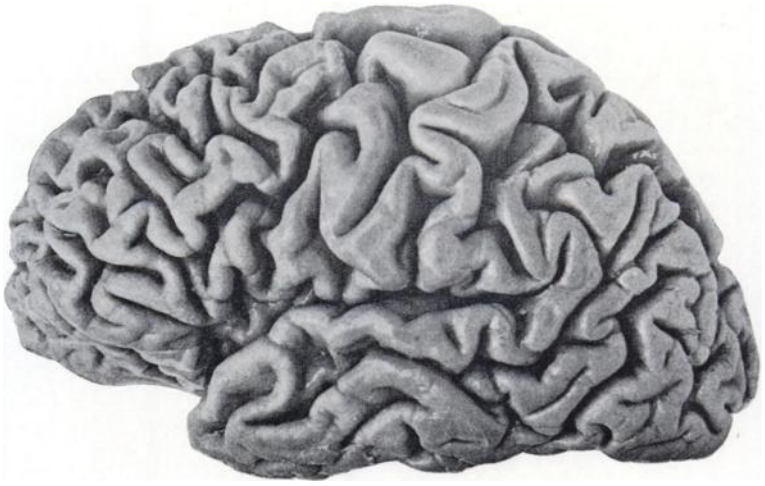

FIG. 5.

To illustrate Dr. J. S. BOLTON's paper.

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PLATE IV. FIG. 4.

Cerebrum exhibits the morbid appearances associated with severe dementia (Group IV). Hemispheres were probably, in their original state, of average size and convolutional pattern.

Photograph of the left hemisphere of a case of marked dementia (Group IV). The figure shows wasting, which is very marked in the prefrontal region, marked in the rest of the frontal region, the superior parietal lobule, the anterior part of the inferior parietal lobule and the first temporal gyrus, and less marked elsewhere.

History.—Female, æt. 89, widow. No family or personal history. Died in Claybury Asylum after a residence of five and a half years. On admission was talkative and reacted well to questions. She had delusions of persecution by electricity. Her memory was very good for her age. Two years later she was noisy and troublesome, had delusions on the subject of marriage, worked in the needle-room, and had developed dementia. Later on she constantly heard voices, and she was at times very noisy, and she became very demented before her death.

Post-mortem.—Dura: Adherent in the left frontal region. S.D.: Moderate excess. Deposit: Large recent film, as thick as brown paper, in the left middle and posterior fossæ above the tentorium; scattered blood-flakes elsewhere. Pia: Fronto-parietal opacity and considerable thickening; strips very readily. S.A.: Considerable excess. Vents.: L., moderately dilated; IV, lateral sacs slightly granular. Vessels: Markedly atheromatous. Encephalon: 1,150 grm. Cerebellum, etc.: 135 grm. R.H.: 492 grm.; stripped 455 grm. L.H.: 485 grm.; stripped 446 grm. Cause of death: Broncho-pneumonia, senile decay, marked renal cirrhosis, vascular degeneration, and cardiac hypertrophy.

FIG. 5.

Cerebrum exhibits the morbid appearances associated with gross dementia (Group V). Hemispheres were probably, in their original state, of average size and convolutional pattern.

Photograph of the left hemisphere of a case of gross dementia (Group V). The figure shows wasting which is extreme in the prefrontal region, very marked in the fronto-parietal region, and less marked elsewhere.

History.—Female, æt. 75, milliner. No family or personal history. Died in Claybury Asylum after a residence of five years. On admission was confused and had no idea of time or place. Was restless, fearful and somewhat resistive, and was of defective habits. Two years later she did not know her name or age, was unable to look after herself, and was wet and dirty. At the time of her death she was at times restless and noisy, and was quite helpless and grossly demented.

Post-mortem.—Dura: Some congestion in occipital region. S.D.: Enormous excess. Pia: Considerable fronto-parietal opacity and thickening, nearly natural elsewhere; strips like a glove in the frontal region and the first temporal gyrus, readily over the parietal lobules, and nearly naturally on the orbital surface, the lower temporo-sphenoidal region, and the occipital pole. S.A.: Great excess, largely under arachnoid. Vents.: L., much dilated; IV., granulations in lateral sacs. Vessels: Considerable atheroma throughout. Encephalon: 1,045 grm. Cerebellum, etc.: 145 grm. R.H.: 410 grm.; stripped 370 grm. L.H.: 418 grm.; stripped 383 grm. Cause of death: Gangrene of the right lung, recurrent carcinoma of breast, vascular degeneration.

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the disease. Under the circumstances, however, he presented such mental symptoms as relatively obscured those of his physical disease and suggested that he was suffering from early and acute general paralysis. This case, in fact, is an illustration of a truth which the writer considers to be beyond the range of controversy, namely that whilst great individual variations in the weight of the cerebrum are compatible with permanent sanity, a minimum weight (probably about 500 grm. per hemisphere) nevertheless exists, below which this is difficult or impossible to preserve.

It will be noted that whilst the hemispheres of this case are simply convoluted, those of the case illustrated on Plate I are still more so. In agreement with this detail the latter patient was an imbecile organ-grinder who suffered from epilepsy, and who resided in an asylum for the five years preceding his death.

On Plate III is shown the left hemisphere of a merchant of considerable business ability who suffered from presenile melancholia with a mild grade of dementia. In association with this there existed some excess of intra-cranial fluid. The unstripped right and left hemispheres weighed respectively 720 and 705 grm., and the left hemisphere in its stripped condition weighed no less than 680 grm. The slight degree of cortical wasting which exists is not obvious in the illustration. The convolutional pattern is exceptionally complex and the hemisphere contrasts markedly with those illustrated on Plates I and II, and especially so when it is borne in mind that all are of exactly the same relative size.

On Plate IV are illustrated the left hemispheres of two cases of progressive senile dementia. These illustrations present very different appearances from those exhibited by the other figures, which, for purposes of comparison, are shown in the preceding plates.

Fig. 4 is a photograph of the left hemisphere of a case of marked dementia (Group IV). The patient, a female, died at the age of eighty-nine years, after a residence of five and a half years, during which dementia gradually developed. She died before the final stage of cerebral dissolution had been reached. The cerebral arteries were markedly atheromatous. There was considerable excess of intra-cranial fluid and also a large recent subdural deposit. The stripped right and left hemispheres

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weighed respectively 455 and 446 grm., and they had probably originally been of average size and of nearly average convolutional pattern. The wasting is very marked in the prefrontal region, marked in the rest of the frontal region, the superior parietal lobule, the anterior part of the inferior parietal lobule and the first temporal gyrus, and less marked elsewhere.

Fig. 5 presents a still greater contrast to the hemispheres illustrated on Plates I, II, and III. It represents the left hemisphere of a case of advanced progressive senile dementia (Group V). The patient, also a female, died at the age of seventy-five years, after a residence of five years, in the final stage of cerebral dissolution. The cerebral vessels were atheromatous. There was enormous excess of intra-cranial fluid. The stripped right and left hemispheres weighed respectively 370 and 383 grm. The cerebellum, however, weighed 10 grm. more than did that of the last case, and therefore, presumably, the cerebral hemispheres had also originally been of greater weight than those of the last case. It is hence probable that the hemispheres of the present case had been originally of at least average size and convolutional pattern. The wasting is extreme in the prefrontal region, very marked in the fronto-parietal region and in the first temporal gyrus, and quite evident, though less marked, elsewhere.

Judging from his general experience as well as from these two individual brains, the writer considers the former to be the more degenerate and the latter to be the less durable cerebrum. The progress of the cerebral dissolution in the first case was relatively slow, in spite of the great age of the patient, and of the presence of gross degeneration of the cerebral vessels. This indicates the existence of a certain degree of neuronic durability. The cerebral hemispheres had also, in their original condition, probably been the smaller and rather the more simply convoluted of the two. On the other hand, in the latter case, cerebral dissolution had, when the patient died, progressed to about the maximum degree which is compatible with life.

Symptomatology of Progressive Senile Dementia.

It is unnecessary to introduce here a detailed description of the symptomatology of progressive senile dementia, as the subjects of mental confusion and dementia have already been

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considered at length in a previous section (*Journ. Ment. Sci.*, July, 1906). On reference to this section it will be seen that both the symptomatological differences between simple and presumably recoverable mental confusion and the mental confusion of progressive senile dementia, and also the more complex phenomena of lower association which are frequently presented by the latter type of mental disease, have been fully referred to.

The present purpose of the writer will therefore be served by the repetition of a case which illustrates with exceptional clearness the chief characteristics of the mental confusion of progressive senile dementia. The interest of this case is increased by the fact that the exciting cause is stated to be intemperance, for this factor has not in any way obscured the details of symptomatology to which it is necessary to draw the attention of the reader.

CASE 21.—*Admitted September 22nd*, 1904 (Hellingley Asylum). Exciting cause, intemperance. Duration prior to admission said to be fourteen days.

Female, married, nurse, æt. 75. Admitted four days ago.

A wrinkled old woman who says that her name is "Sarah C—x, a large family we are." This is her married name and her maiden name was H—s. She then states that she married again and that her present name is W—m. (Isn't your name Mrs. B—d?) "I am, sir, because I was a widow and married Mr. R. B—d." She recognises the nurse as "Mrs. W—m's daughter. Mrs. P—r it was once I know. Weren't your grandmother's name P—r?" She then tells me that the nurse is "Mrs. P—r's grand-daughter, isn't it? I know the old lady and I know your mother." She states that she has seen me before at Bishopstoke. She does not know whether my name is P—r or not. "I know Mr. P—r and Mrs. P—r and thought you were Mr. P—r." She calls a patient named M. B—d "Mrs. T—r," and another named S. P—x "Mrs. P—r," and a nurse "Mrs. P—r's daughter." She thinks to-day is Sunday (Monday), and that the date is the 25th or 26th (26th). She replies that the month is "not February is it?" (September), and that the year is "I don't know whether it is 101 or 102" (1904). (Age?) "I'm getting on for forty. It's a nice little age, isn't it? I suppose you're beginning to shave it, aren't

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you?" (Out to-day?) "Yes, I've been out to see the cricket match to-day." She states that she saw her husband at Bishopstoke this morning. She brought her husband's breakfast home with her—bread, butter, and oysters. I tell her that I don't know a *soul* in Bishopstoke, and she remarks, "A *soldier* there, are you?" She replies that she has children at home. The youngest is five or six, and she has twenty-five living, and thinks it likely that she will have another to make twenty-six. When asked where she is she replies that it is "about one mile from Bishopstoke Station here." When again asked the same question she remarks, "Very nice place, I like it very well. I should think it was a bonny place myself." I then ask her if she is a country woman, and she replies, "Southampton woman." She answers questions quickly and apparently rationally, but as a whole does not volunteer much information about herself. She laughs and looks about slyly from face to face as if she thinks that she is amusing. She has evidently lived a rather dissolute life, as she says, "I went to Bishopstoke this morning. I enjoyed myself I can tell you. I always do when I go on the spree. I was along with your nephew last time I saw you, and with his father this morning." She is very erotic. When I touch her chin to get her to open her mouth she tells me I am a rascal, and that "he thought he'd tickle me under the chin." She is wet and dirty in her habits, but is quiet and no trouble, and she takes her food well.

This patient died two and a-half months after admission in a condition of advanced dementia.

The chief details of importance which are exhibited by this case are the following:

(1) The patient does not know the time of year.

(2) She gives her first married name instead of her present one.

(3) She states that she is "getting on for forty," whereas she is seventy-five years of age.

(4) She confabulates readily, *but the psychic phenomena which are evolved are, on the whole, impossible as statements of fact, and are largely based on groups of memorial units dealing with her early life.*

(5) She has well-marked illusions of identity, *but she continually employs the same name, "P—r," in her identifications.*

In all these points the case differs from one of presumably

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recoverable mental confusion, and shows evidence of the mental confusion of progressive dementia. Other similar examples have also been cited and discussed in the section referred to—*e.g.*, Case 3, pp. 437–438, and Case 12, pp. 452–455.

As the various phenomena of lower association which frequently occur in cases of progressive senile dementia are, in reality, closely connected with, and, in fact, part of, the existing mental confusion, and as they are thus the symptomatological expression of active neuronic dissolution, the writer has not employed them as a basis for the elaboration of clinical types.

The cases falling into the group of progressive senile dementia have therefore been grouped as follows:

	M.	F.	T.
Sub-class (1): Melancholia with dementia .	3	2	5
Sub-class (2): Mania with dementia . .	–	5	5
Sub-class (3): Simple dementia . . .	6	8	14
Total . . .	9	15	24

The cases of progressive senile dementia thus form the small proportions of 5·4 *per cent.* of the 445 cases of dementia under consideration, and 3·3 *per cent.* of the total of 728 cases of amentia and dementia.

This point is interesting in view of the fact that the cases are derived from the largely agricultural population of East Sussex. Though the writer has no statistics at his disposal, he is nevertheless quite certain that progressive senile dementia is much more common amongst the insane derived from the great centres of population, and he is also inclined to think it more common in Lancashire than in the County of London.

As would be expected, very few cases of progressive senile dementia are capable of useful work. Of the nine males, seven were unemployed and two did a little work; and of the fifteen females, thirteen were unemployed and two did a little work.

Class (b).

Dementia Paralytica (*General Paralysis*).

Though earlier in this section the writer has indicated the existence of certain types of progressive dementia, which are, from the aspect of general pathology, homologous with dementia

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paralytica, these types are so unimportant from the clinical aspect owing to their rarity, and they are consequently at present so undefined, that he proposes to confine his attention in the following description to dementia paralytica alone.

It is not his intention to discuss, or even to enumerate, the various views which have been enunciated with regard to the causation and general pathology of this clinical entity.

The question as to whether dementia paralytica is primarily a meningo-encephalitis or a primary degeneration of the cortical neurones is now chiefly of historical interest, as it is very generally accepted that the essential histological features present consist on the one hand of a proliferation of the extra-neuronic elements, which is of different ages and of different degrees of severity according to the stage and type of the case, and on the other of a mixture of acute and chronic nerve-cell changes which also vary in type and extent in accordance with the clinical symptomatology manifested by the patient. It is probably quite unimportant to seriously discuss whether the former or the latter occurs the first, for, in the established morbid state, a "vicious circle" exists in which each factor in turn causes the other; and the writer hopes to make clear that there is every reason to believe that, under the influence of different exciting causes, either may originally form the starting point of the morbid process.

The opinion that syphilis, *i.e.*, *active infection by the Spirochæta pallida*, is the cause of dementia paralytica, owing to the frequency, or it may even be said the constancy, with which evidence is obtainable that the subjects of dementia paralytica have previously suffered from this disease, though widely held, is opposed by facts which, in combination, appear to be quite crucial. For example, on a liberal estimation probably only about 2 *per cent.* of the persons who have suffered from syphilis later on develop dementia paralytica. Again, general paralysis, even in the earliest stages, is quite intractable under anti-syphilitic treatment. Further, this symptom-complex develops at very variable periods after infection with syphilis, *e.g.*, from four to twenty-five years in the personal experience of the writer.

A serious attempt has recently been made by Ford Robertson to demonstrate that the essential cause of dementia paralytica is a type of diphtheroid bacillus to which he has applied the

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generic name of "*Bacillus paralyticans*," and of which he describes at least two varieties. The writer does not propose to discuss the views of Ford Robertson, as his investigations are not yet completed, as his conclusions are at present so entirely *sub judice*, and as these are diametrically opposed by the pathological, etc., considerations contained in the present paper. He thinks it desirable, however, without expressing any opinion, to draw the attention of the reader to the remarks he has already made with reference to Watson's experiments with abrin and ricin on the guinea-pig and rabbit, for these experiments have afforded results which, as far as it is possible to judge from Ford Robertson's descriptions, resemble those obtained by this investigator from his injection experiments.

The writer has already, earlier in this section, indicated his views as to the relationship, from the aspect of general pathology, which exists between dementia paralytica and progressive senile dementia, and as to the part played by a previous attack of syphilis in the development of the former of these types of progressive and secondary dementia.

In the following description, therefore, the ætiology of dementia paralytica will be considered mainly from the point of view of whether this clinical entity is a subdivision of mental disease, or is an organic disease of the cerebrum which merely in its symptomatology resembles insanity. If the latter were true the frequently expressed opinion that no anxiety need be felt regarding the future of the offspring of general paralytics would be justified, and dementia paralytica would bear no closer a relationship to mental disease than does cerebral tumour or cerebral abscess.

In the opinion of the writer, however, dementia paralytica is an integral part of mental disease, and, were syphilis non-existent, the majority of the existing cases of dementia paralytica would merely be replaced by cases of the primarily neuronic dementia which has already been considered. Of these cases the majority would remain in asylums as permanent inmates, and the rest would possess a sufficient remainder of intelligence to be discharged as "recoveries" or to the care of their friends. The writer thus hopes to demonstrate that the general paralytic is a lunatic who differs from the ordinary case of primarily neuronic dementia solely in having earlier in life suffered from syphilis.

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He believes that the ordinary sane individual and the ordinary psychopath or potential lunatic, who possesses cortical neurones of average durability, may suffer from syphilis with impunity as regards the later onset of dementia paralytica; and he would express the same opinion with regard to the syphilised lunatics with little or no dementia who are fairly common in asylums.

On the other hand, he thinks that a psychopath, who possesses cortical neurones of subnormal durability, and who, apart from an attack of syphilis, would develop a moderate grade of dementia as the result of one or more attacks of mental alienation, would, after an attack of this disease, sooner or later suffer from one or other of the types of dementia paralytica.

Further, since he considers, as has already been remarked, that the extra-neuronic reaction, which constitutes the essential feature of cases of dementia paralytica, is allied to, and only differs in type and degree from that occurring in cases of progressive senile dementia, he is of the opinion that whilst in ordinary life many psychopaths with deficiently durable cortical neurones manage to survive without the onset of an attack of insanity, all or nearly all such psychopaths would, if previously infected with syphilis, sooner or later develop (chronic) dementia paralytica.

This latter suggestion is founded on a basis of general pathology, but it is not contradicted by the estimate that about 2 *per cent.* of general paralytics occur amongst the former subjects of syphilis, as this probably roughly represents the percentage of psychopaths in the general population, this certainly being several times greater than the existing proportion of certified lunatics in England and Wales (1 in 282 in January, 1907).

Such an estimate is naturally not to be considered as other than suggestive, for even an approximate determination of the incidence of syphilis in England is impossible, and it is quite likely that the percentage of psychopaths amongst the subjects of syphilis may differ somewhat from that in the general population.

In the following description an attempt will be made to record in compact but intelligible form such data as the writer is able to produce with reference to the relationship of dementia paralytica to mental disease.

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The subject will be considered under the following headings:

(1) Evidence as to the existence of heredity of insanity and of parental and family degeneracy in the subjects of dementia paralytica.

(2) Evidence as to the relationship between dementia paralytica and mental disease, derived from the study of the death rates in mental disease (including and excluding dementia paralytica) at different ages, and from the comparison of these death rates with the homologous death rates in the corresponding general population.

(3) Pathological evidence as to—

(*a*) The relationship between the morbid anatomy and the regional cortical wasting of dementia paralytica and of progressive senile dementia.

(*b*) The existence of cerebral under-development in certain types of dementia paralytica.

(4) Evidence as to the relationship between dementia paralytica and mental disease, derived from a study of the clinical types of dementia paralytica.

(1) *Evidence as to the Existence of Heredity of Insanity and of Parental and Family Degeneracy in the Subjects of Dementia Paralytica.*

As has already been stated, the writer is of the opinion that the presence or absence of *heredity of insanity* in any case or series of cases possesses merely a relative value, as family and social conditions so largely decide whether any particular individual should be sent to an asylum or not. The equally and often more important evidence of *family or parental degeneracy* is frequently not available, and is usually not easy to obtain.

He thinks it more probable that isolated cases of insanity arise from the intermarriage of ill-assorted couples and mild degenerates, and that the severer grades of family degeneracy follow the intermarriage of definite degenerates, rather than that isolated examples of insanity in either parental stock will be followed by insanity in the offspring of such parents.

He would, in other words, place the percentage of heredity at 100 with regard to the offspring of either degenerate or "normal" individuals, and, without going so far as to assert that non-traumatic cerebral under-development or dissolution

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cannot occur in the absence of hereditary causes, would emphatically express his doubts with regard to its occurrence with any degree of frequency.

On the thesis that dementia paralytica were an integral part of mental disease, it would be expected therefore that a high percentage of heredity of insanity and of parental or family degeneracy would be obtained in a series of carefully taken cases, although this percentage, for the reasons stated, would necessarily fall far short of 100.

In a series of 85 cases of dementia paralytica which were published some years ago by the writer (*Arch. of Neurol.*, vol. ii), satisfactory family histories were obtained. These histories were the outcome of several hundred personal interviews with all the available relatives or friends of the patients, and of information collected by other means.

Much labour was expended on the subject, for the difficulties in the way of obtaining information concerning the family histories of cases of dementia paralytica are often very great, and particularly so in the case of patients of the male sex. The wife is frequently the only visitor, and it is quite common for the family of the patient to be unknown to her. This is more often the case with patients suffering from dementia paralytica than in other forms of insanity, for the former patients, owing to their previous dissipated and often wandering life, are frequently entirely out of touch with their relatives. The usual age of the subjects of dementia paralytica is again a serious drawback, as the older the patient is, the fewer are the available relatives who can give trustworthy information regarding the family history. Lastly, it is common for relatives to be informed that the disease is not insanity but is due to the former dissipated life of the patient, in order that their natural apprehensions concerning the future of the offspring may be relieved. They therefore tend to hide many facts of family history which they would otherwise have mentioned. The writer has in fact met with several instances in which the relatives, until definitely taxed regarding the correctness of some specific fact of history which had been accidentally acquired from other sources, stoutly denied the existence of any insanity in the family, and deliberately suppressed the name of the subject of this when first giving the history. Whilst such deliberate mis-statements are at times met with

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during ordinary history-taking, the writer is convinced that they are more frequently found in the case of the histories of general paralytics, the friends of whom are often only too anxious to accept former syphilis as the cause of the disease.

Of the eighty-five cases which will now be referred to, thirteen were private and seventy-two were rate-paid patients.

In 8 of the 13 private cases there was direct or collateral insanity; in 3 there were allied disorders, including epilepsy; in the twelfth the father died of cerebral hæmorrhage, and the mother of paralysis, and the patient was the youngest of a family of eight; and in the thirteenth the mother and sister died of phthisis, a brother was delicate, and eleven out of fourteen in the family were dead.

In one instance the parents were first cousins, and a paternal uncle and two female cousins were insane; four patients were the youngest in the family; and in three families there was a very high death rate. In four cases there was phthisis in the family (mothers and sisters), in two diabetes, and in one asthma.

Of the 72 rate-paid cases, *actual insanity* existed in 45 families (62·5 *per cent.*), and in 4 of these true epilepsy also existed, apart from the cases of insanity. *True epilepsy* existed in 5 other families without insanity (6·9 *per cent.*), though in one case it was probably associated with melancholia of pregnancy. Histories of insanity and epilepsy consequently existed in 50 of the 72 families (69·4 *per cent.*). In these fifty families, as further evidence of family degeneracy, there were disorders allied to insanity in at least sixteen instances (nervous diseases not being included).

In the 45 histories containing actual insanity there existed 65 insane relatives. These included 10 brothers, 10 sisters, 11 mothers, 7 fathers, 3 maternal grandfathers, 1 maternal grandmother, 2 paternal grandfathers, 1 maternal great-grandfather, 2 maternal uncles, 5 maternal aunts, 4 paternal uncles, 1 paternal aunt, and 8 collaterals (1 half-sister, 1 half-brother, 4 cousins, 1 father's maternal cousin, and 1 sister's son). Several of these insane relatives suffered from fits, and a few may have been cases of general paralysis, but no stress can be laid on this point, as the details available are insufficient.

Of the remaining 22 of the 72 cases, *psychopathy* (equals "border-land cases," and does not include examples of nervous

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disease) existed in 9 (12·5 *per cent.*), 2 brothers, 2 sisters, 3 mothers, and 3 sons being affected.

Of the remaining 13, there was *an abnormally high death-rate* amongst relatives in no less than 7 cases (9·7 *per cent.*).

Finally, of the remaining 6, in 3 there was *paralysis*; in one the patient was the *delicate child of the family* and did not walk until he was four years of age; and in the remaining 2 there was merely a history of alcoholic excess in the parents.

Hence, of the 72 histories of rate-paid patients, there was *psychopathic heredity* in no less than 81·9 *per cent.*, and an abnormally high family death rate in another 9·7 *per cent.* In the remaining 8·4 *per cent.* less important ætiological factors existed.

Phthisis existed in 19 of the 72 families (26·4 *per cent.*), in 8 affecting brothers and sisters, and in 6 the father's, in 4 the mother's, and in 1 both families.

Intemperance in alcohol existed in 26 of the 72 families (36·1 *per cent.*), both sides of the family suffering from the disorder in 7 cases, the paternal side in 12, and the maternal in 5; the remaining 2 cases occurred in sisters of the patients.

General or nervous diseases were ascertained to have existed in 19 cases (26·4 *per cent.*).

In 13 families (18 *per cent.*) there was an *abnormally high death rate*, it being so high in 7 of these that very few family details could be obtained.

The figures given above illustrate the high percentage of heredity of insanity and of parental and family degeneracy which occurs in dementia paralytica, and form the first part of the evidence which the writer is able to produce in support of the thesis under consideration.

He is, of course, aware that comparative observations on normal individuals and on ordinary cases of mental disease would be necessary in order that exact conclusions might be drawn regarding the respective degrees of degeneracy in these two classes and in dementia paralytica. Such observations would, however, necessarily have had to be made on exactly similar samples of population, and for these he had neither the time nor the opportunity. It is also extremely doubtful whether the results would have been of sufficient value to justify the expenditure of the necessary time and labour, even if it had been possible, as for practical purposes the only question it was necessary to settle was whether or not a high heredity of

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insanity and of parental and family degeneracy were obtainable in dementia paralytica.

The writer considered it desirable to carry out this investigation as there are few subjects on which greater differences of opinion exist than with regard to the question of the percentage of heredity of insanity in dementia paralytica.

That the figures he has given are higher than those published by most observers he admits, but he judges this to be due to the fact that in, at any rate, the majority of cases, the histories employed are such as are provided by ordinary case-book entries, and are not obtained as the result of laborious individual investigation.

In Table XXII of the *Sixty-first Report of the Commissioners in Lunacy* are given the proportions (*per cent.*) of the yearly average number of the total patients admitted to the asylums in England and Wales during the five years, 1901–5, in which certain assigned causes of insanity were found to exist. Those data which bear on the question under consideration are as follows:

Causes of insanity.	Proportion (*per cent.*) to the yearly average number admitted during the five years.	
	M.	F.
Hereditary influence ascertained . .	19·8	25·3
Congenital defect ascertained . .	6·3	4·1

As at least a large proportion of the histories of admissions to asylums are not taken at all, these figures are naturally much lower than such as would be obtained from the data provided by asylum case-books.

In his presidential address (*Journal of Mental Science*, October, 1902) Dr. Wiglesworth provides statistics of great interest in this connection. "My statistics deal with a series of 3,445 insane patients who have been admitted into Rainhill Asylum under my care during a period of twelve years, 1,693 of these patients being males and 1,752 females. It has not been practicable to include all cases that have passed through the asylum in the course of that period, as many patients come in of whose antecedents it is impossible to obtain any trace, but every patient

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has been included of whose family history any details whatever were obtainable."

These are shown in the following table:

Form of insanity.	Number of cases.			Number of these showing heredity.			Percentage of hereditary cases on total numbers.		
	M.	F.	T.	M.	F.	T.	M.	F.	T.
Congenital insanity (idiocy and imbecility) with or without epilepsy . .	35	33	68	13	17	30	37·14	51·51	44·11
Epileptic insanity . .	77	43	120	15	23	38	19·48	53·48	31·66
General paralysis . .	363	70	433	60	22	82	16·52	31·42	18·93
Ordinary insanity (non-congenital)—mania, melancholia, dementia, etc.	1,218	1,606	2,824	331	484	815	27·17	30·13	28·85
All cases together .	1,693	1,752	3,445	419	546	965	24·74	31·16	28·01

It will be noted that the percentage of hereditary cases amongst the female general paralytics is slightly higher than that amongst the examples of ordinary insanity, whereas amongst the male general paralytics it is much lower. From the considerations already adduced it seems to the writer of the present paper to be at the least probable that this sex difference is largely or entirely due to the less satisfactory nature of the information which is usually obtainable regarding the personal and family history of male general paralytics. This explanation is supported by the fact that no such sex-difference existed in the case of the eighty-five histories referred to above. The private patients, for example, were all of the male sex.

It is true that in the above table a similar sex-difference exists in the case of the epileptics, the hereditary cases forming an extremely high percentage in the case of the females and a low one in the case of the males, in comparison with the respective percentages in the cases of ordinary insanity. Epileptic insanity, however, is so frequently the result of organic and traumatic causes that it falls into a different category from other cases of mental disease, and the number of cases (120), even when recruited from the cases of congenital insanity, forms

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an unusually low proportion of the total of 3,445 cases. Dr. Wiglesworth expressed this opinion as follows: "One cannot but think that this great difference must in part be due to accidental causes, and that if larger numbers were taken, the real disparity would be found not so great. Still, the figures certainly lead one to suppose that epilepsy in the male is far more of an acquired affection than it is in the female."

The percentages given in the case of congenital insanity are also of relatively slight value, for "the number of cases of congenital insanity admitted into Rainhill Asylum is a small one, due in part to the fact that, the asylum having been overcrowded for many years past, a restriction has been put upon the admission of this class of cases," and Dr. Wiglesworth adds "These cases have, however, been taken indiscriminately, and no endeavour has been made to exclude 'accidental' idiots from the list, whose idiocy may have been occasioned by accidents occurring during the process of birth. On account of the superior size of the male head, it is probable that there are more cases of this class amongst males than amongst females, and if all these cases (in which one might expect an absence of hereditary taint) were excluded, it would tend to make the difference between the two sexes somewhat less pronounced."

The important sex difference in the table, therefore, concerns the general paralytics, and this is, in the opinion of the writer, susceptible of the explanation he has given.

Hence Dr. Wiglesworth's statistics may be considered not to contradict the conclusions drawn from the personally obtained data which the writer has provided with reference to the existence in dementia paralytica of a high percentage of heredity of insanity and of parental and family degeneracy. Further, they are susceptible of the interpretation that the percentage in dementia paralytica does not substantially differ from that in ordinary mental disease.

(2) *Evidence as to the Relationship between Dementia Paralytica and Mental Disease, derived from the Study of the Death Rates in Mental Disease (including and excluding Dementia Paralytica) at Different Ages, and from the Comparison of these Death Rates with the Homologous Death Rates in the corresponding General Population.*

The writer has calculated the death rates at different ages

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amongst the insane of a certain asylum population. These death rates on the one hand refer to the whole of the asylum population, and on the other to the subjects of mental disease apart from dementia paralytica.

The object of the writer is two-fold. He proposes in the first place to compare the death rates of the insane with those of the corresponding general population, and in the second to determine what modifications of the former rates result from the exclusion of the cases of dementia paralytica.

The data employed with reference to the general population are the corrected death rates per 1,000 living in the County of London for the year 1905 (*Sixty-eighth Report of the Registrar-General*, Tables 16 and 17). The margin of error owing to the use of these data is inconsiderable, as the rates for neighbouring years are practically constant. It is necessary to assume that the death rates for the whole County of London and for its several larger sub-districts are the same, and this assumption may appear likely to cause a serious error. Such an error, however, if it exists, cannot be considerable, as the death rates in the County of London do not, as regards the present purpose of the writer, differ greatly from those of the total general population of England and Wales.

The data regarding the insane refer to the inmates of the London County Asylum, Claybury. The deaths employed are those included in Series B (*Journal of Mental Science*, April, 1905), together with the cases of dementia paralytica dying during the same period of twenty months, from October, 1901, to May, 1903, inclusive.

The number of deaths during the period under consideration was 311. Of these, 16 special cases (8 male and 8 female) suffered from gross lesions, etc., and were unclassified. These deaths are excluded, and an error of about 5 *per cent.* is thereby introduced throughout the death rates, as it is impossible to correct these by also excluding the living portion of the asylum population from which such "accidental" cases arise. This general lowering of the death rates, however, applies to all the tables, and is fairly evenly distributed through the decades, the eight male cases dying at ages varying from 21 to 77 years, and the eight female cases dying at ages varying from 27 to 67 years. This error, therefore, as will be seen, does not pre-

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judicially affect the conclusions which will be drawn from the death rates.

The cases employed thus include 127 males, of whom 83 are ordinary cases and 44 are general paralytics; and 168 females, of whom 150 are ordinary cases and 18 are general paralytics.

The annual death rates per 1,000 living at the eight age-periods which are dealt with are worked out from the estimated average population of the London County Asylum, Claybury, at these age-periods during the twenty months referred to.

In the first instance the rates are prepared from the total numbers of male and female deaths respectively and from the estimated total average male and female populations respectively, at the eight age-periods under consideration.

Further death rates are then prepared from the numbers of non-general-paralytic male and female deaths respectively, and from the estimated non-general-paralytic average male and female populations respectively, at these age-periods.

The estimated average population of the London County Asylum, Claybury, at the eight age-periods during the twenty months under consideration, is based on the returns of patients resident on December 31st, 1902.

The average general paralytic population during these twenty months is estimated by the writer, from data at his disposal, to be 121, of which 90 (or 9 *per cent.* of estimated average total male population) are males, and 31 (or 2·2 *per cent.* of estimated average total female population) are females.

Of the 90 males, 12, 29, 37, 8, 3, and 1 are estimated to belong to the second to the seventh age-periods respectively; and of the 31 females, 7, 15, 7, and 2 are estimated to belong to the second to the fifth age-periods respectively.

The estimated average general paralytic male and female populations are subtracted from the estimated average total male and female populations, in order to obtain the average non-general-paralytic male and female populations, which are employed for the preparation of the series of death rates on the fourth lines of Tables I and II respectively.

These death rates are shown on the following tables:

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TABLE I.—*Death-rates per* 1,000 *Living Males.*

Age periods.	15-	20-	25-	35-	45-	55-	65-	75-	85 and upwards.
A. London County, 1905	2·8	3·7	5·8	10·6	19·4	35·8	70·3	135·9	313·7
B. Series B + G.Ps. on estimated total average population at the several age-periods . . .	26		58	55	64	121	129	280	600
Ratio of B to A . .	About 8		10	5·2	3·3	3·4	1·8	2·1	1·9
C. Series B, on estimated total average population less estimated G.P. population at the several age-periods . . .	26		44	26	25	110	126	257	600
Ratio of C to A . .	About 8		7·6	2·45	1·3	3·1	1·8	1·9	1·9

TABLE II.—*Death-rates per* 1,000 *Living Females.*

Age periods.	15-	20-	25-	35-	45-	55-	65-	75-	85 and upwards.
A. London County, 1905	2·3	2·8	4·2	7·8	14·0	25·9	55·1	117·1	288·0
B. Series B + G.Ps. on estimated total average population at the several age-periods .	27		41	48	48	59	199	315	600
Ratio of B to A . .	About 10·6		9·8	6·2	3·4	2·3	3·6	2·7	2·0
C. Series B, on estimated total average population less estimated G.P. population at the several age-periods . . .	27		31	35	40	57	199	315	600
Ratio of C to A . .	About 10·6		7·4	4·5	2·9	2·2	3·6	2·7	2·0

It will be noticed that the death rate in the normal population nearly doubles itself at each decade, the rise being slightly more rapid throughout in the case of the males than in that of the females.

In the case of the total male insane population, as is shown both by the death rates (Table I, B) and by their ratios to the normal death rates, which are shown on the next line of the table, a rise also occurs throughout the age-periods, but this

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rise after the second decade becomes progressively less in comparison with the normal.

In the case of the total female insane population (Table II, B), a similar rise in the death rates occurs throughout the decades, but it is somewhat less than in the males in the earlier decades, and somewhat greater than in the males in the later.

The average total insane male death rate (not shown in the table) is 76·5, and the average female is 72·2. These thus differ slightly only, but this difference is in the same direction as that of the normal population, though it is less marked.

The death rates of the total insane of both sexes thus resemble those of the general population in forming an ascending series, the increments to which are, however, much greater than in the latter at first, but become proportionately less marked as the ages increase, this being especially evident in the case of the male sex.

In line C of the tables the death rates of the non-general-paralytic male and female insane population are given.

In the case of the females (to whom in the total number the general paralytics form a relatively small proportion), the ascending series of death rates (Table II, C, and also the ratios on the following line) is not markedly affected, though the death rates in the second and third, and to a less extent in the fourth, of the given decades are decidedly low.

In the case of the males, however (to whom in the total number the general paralytics form a relatively large proportion), the ascending series is entirely broken up at the third and fourth of the given decades, and the unexpected and curious result appears that *male lunatics have an extraordinarily low death rate between the ages of* 35 *and* 54. (Table I, C, and also the ratios on the following line.)

This result is so grossly marked that the only possible inference from the figures is that exclusion of the general paralytic members of the lunatic population is unjustifiable, and that therefore *cases of general paralysis form an integral part of mental disease.*

In other words, it is impossible to avoid the conclusion that, whatever be the cause of the particular symptom-complex known as general paralysis, the cases which exhibit this are nevertheless lunatics, and not merely the subjects of a disease of the brain of microbic or syphilitic origin.

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(3) *Pathological Evidence as to* (a) *the Relationship between the Morbid Anatomy and the Regional Cortical Wasting of Dementia Paralytica and of Progressive Senile Dementia, and* (b) *the Existence of Cerebral Under-development in Certain Types of Dementia Paralytica.*

In its essentials the morbid anatomy of dementia paralytica is that of ordinary progressive dementia. Certain important differences, however, exist, and these the writer considers to be largely or entirely due to the higher degree of reparative proliferation on the part of the non-neuronic elements of the encephalon which is present in dementia paralytica.

As in progressive senile dementia, so here, the grossly obvious features are cerebral wasting, and replacement of the lost cerebral tissue to some extent by reparative proliferation of the non-neuronic elements, but chiefly by a large quantity of intra-cranial fluid.

The following description will largely be confined to those appearances in which the morbid anatomy of dementia paralytica differs from that of progressive senile dementia, with the view of illustrating how these are due to differences in the immediate ætiology and in the course of these different types of dementia.

The cerebra of certain selected cases will then be illustrated and described with the object of demonstrating (*a*) that the wasting in dementia paralytica bears a close resemblance in distribution and degree to that occurring in progressive senile dementia, such a resemblance, in fact, as renders it impossible to avoid the conclusion that in both cases this wasting is the result of dissolution of the last evolved and functionally highest regions of the cortex, and (*b*) that cerebral under-development occurs in dementia paralytica just as it exists in ordinary mental disease, and that the naked-eye anatomy of the cerebrum gives as important evidence regarding the unity of dementia paralytica and ordinary mental disease as will next be produced with regard to the clinical types of dementia paralytica, and as has already been detailed from other aspects in the preceding sub-sections.

(A) *Morbid anatomy of dementia paralytica.*—In early and moderately developed, but less often in very advanced cases, the venules, etc., of the intra-cranial membranes and encephalon

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are intensely congested, a morbid appearance usual in *status epilepticus*, but not often seen in progressive senile dementia.

Even in relatively early cases the excess of *subdural fluid* is large, and in advanced cases it is as great as, or greater than, occurs in ordinary gross dementia.

In the 44 male cases already referred to, great or very great excess existed in 37, excess just over the tentorium in 2, moderate excess in 2, slight excess in 1 early case, and no excess in 2. Of the two without subdural excess, one was very recent and acute, and in the case of the other the subdural space contained 205 grm. of recent blood-clot.

Of the 18 female cases already referred to, a very great excess of subdural fluid existed in all.

This sex-difference is in all probability associated with the greater chronicity of the female cases.

A similar sex-difference exists with regard to the frequency of the *subdural deposits*, which are somewhat more common in dementia paralytica than in progressive senile dementia, except in the case of the severer grades.

Of the 44 male cases, subdural deposits occurred in 12, or 27 *per cent.*, and of the 18 female in 2, or 11 *per cent.*, the percentage in the case of the total of 62 being 22·6.

Of 85 male cases published in a previous paper, these deposits occurred in 25, or 29·4 *per cent.*, and of 38 female in 4, or 10·5 *per cent.*, the percentage in the case of the total of 123 being 23·6.

The percentages in these two series of cases are thus substantially the same. In both series also in each sex, half of the deposits were of recent date and the remaining half were more or less organised, and in several instances multiple.

In progressive senile dementia subdural deposits are rather less common, except in the very advanced cases.

In the case of the combined series A and B referred to in the first part of this paper, and including in all 433 cases of ordinary mental disease, these deposits existed in 17·4 *per cent.* of the 92 cases in Group IV (severe dementia) and in 22·8 *per cent.* of the 79 cases in Group V (gross dementia), the latter percentage being about the same as that indicated above as occurring in dementia paralytica.

In early cases there is little or no excess of *sub-arachnoid fluid*; in slow chronic cases there is often considerable excess,

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frequently in the form of scattered "arachnoid cysts"; and in advanced cases there is great excess, the prefrontal pia-arachnoid being in many instances ballooned out by the subjacent fluid.

In early cases the *pia-arachnoid* may superficially present few abnormal characters beyond a larger or smaller amount of congestion; in later cases it is, as a rule, immensely thickened and opaque, and stretches as a continuous sheet over and often entirely hides the subjacent sulci. The thickening and opacity are, usually, most marked over the fronto-parietal regions and the neighbouring median parts of the hemispheres, and also over the first temporal gyri. They are often not so marked in the prefrontal region, where the pia-arachnoid is raised up by subjacent fluid. The opacity may, however, be more widespread, and may even occupy the whole cerebrum except, apart from rare cases, the orbital surfaces of the frontal lobes, the lower and inner occipito-temporal regions, and the posterior poles of the hemispheres.

In progressive senile dementia the distribution of the opacity and thickening is similar to that described, but the fibrosis (and also the contraction) of the pia-arachnoid is commonly much less marked than in dementia paralytica.

Even in early cases of dementia paralytica the pia-arachnoid is granular in the mid-line prefrontal region below the falx cerebri, and pia adheres more or less firmly to pia in this position, the actual area of adhesion depending on the size and shape of the falx cerebri. In more advanced cases the adhesions in this region are dense, and the hemispheres cannot be separated without tearing the subjacent cortex. It is worthy of note in this connection that the region under consideration is the only part of the encephalon where pia meets pia, as elsewhere the pia lies in contact with the dura, to which, however, it only rarely forms adhesions.

Particularly in early cases, but also in later ones in the regions into which the morbid process is extending, the pia is adherent to the subjacent cortex. Later on, when the pia-arachnoid has become much thickened and the cortical wasting is pronounced, the membrane strips like a glove from the underlying cortex. Decortication on stripping is usually laid much stress on in descriptions of dementia paralytica, but it is an uncertain sign. The more chronic or the more advanced the case happens to be, the less is the decortication, and *vice-*

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versâ. Decortication, on the other hand, is much increased by œdema of the brain, and especially by *post-mortem* decomposition. It is largely obviated by the absence of these latter factors, but in early cases it very often occurs in the mid-line prefrontal region below the falx cerebri.

The peculiarly localised areas of adhesion between the pia and the cortex, which are usually situated on the flat external surfaces of the convolutions and do not reach to the fissure lips, strongly suggest a vascular causation. This is almost conclusively proved by the occasional occurrence, particularly in acute cases, of fairly extensive areas of adhesion which approximate very closely to known areas of arterial distribution.

This is finally proved by the fact that exactly similar regions of pial adhesion occasionally occur in cases of gross senile vascular degeneration (with or without dementia) and also in cases of progressive senile dementia. (*Archives of Neurology*, vol. ii, pp. 483–4, Case 201.)

The *lateral ventricles* in dementia paralytica are dilated, and often extremely so, and, with the *third*, are much more frequently granular than are these regions in progressive senile dementia. The *fourth ventricle*, however, exhibits, as a rule, the most characteristic naked-eye sign of dementia paralytica. Granularity of the lateral sacs of the fourth ventricle is common in all varieties of insanity, and in progressive senile dementia granules also at times exist on each side of the mid-line in the upper half of the lozenge. They, however, rarely or never occur in the lower half or calamus except in dementia paralytica, in which, even if the granularity is general, it is usually most marked in this situation.

The smaller *cortical arteries*, even in very early cases of dementia paralytica, are invariably fibrous, and in toughness resemble strands of thread or fine wire.

The *basal vessels* at times show no obvious naked-eye abnormality, but in many cases, and especially in such as have acquired syphilis at or after maturity, they are dilated and irregularly thickened owing to patches of pearly white fibrosis. They are occasionally small and fibrous. They are less frequently calcareous.

Of the 44 male cases of dementia paralytica referred to above, the basal arteries were affected to a greater or a lesser degree in 28, or 64 *per cent.*, and were apparently normal in 16, or 36

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per cent. In the 28 cases the affection was slight in 11, moderate in 10, and severe in 7. The average age of these 28 cases was 49 years, whereas in the case of the remaining 16 the average age was 38 years.

Of the 18 female cases, the basal arteries were more or less affected in 9, or 50 *per cent.* In these the affection was slight in 4, moderate in 2, and severe in 3. The average age of these cases was 47 years, whereas in the case of the 9 with apparently normal basal vessels the average age was 38 years.

The basal vessels were therefore more frequently affected in the case of the male sex. In the case of both sexes the average age of the patients with affected basal vessels was about ten years higher than that of the patients with apparently normal vessels.

These morbid appearances in the cerebral arteries resemble in their characters the dense, almost cartilaginous, pearly-white fibrosis of the often-dilated aorta which frequently occurs in dementia paralytica, and particularly so in cases over the age of forty years. These vascular changes, and particularly that in the aorta, are, in the experience of the writer, practically diagnostic of a former attack of syphilis, in that they represent an intense reparative reaction to previous severe injury, and at the same time are found in syphilised but not in non-syphilised subjects.

The vascular degeneration and nodular atheroma of the cerebral arteries which occur in senility and premature senility show, on the other hand, no such fibrotic appearances. In these cases, in association with and probably owing to the exceedingly feeble reaction of repair which they possess, a deposition of lime salts occurs and results in the better known calcareous degeneration.

As has already been stated, this condition of the cerebral arteries is practically constant in progressive senile dementia. On the other hand, in dementia paralytica, though naked-eye pearly fibrosis is not a constant feature, it is, when present, a highly characteristic morbid appearance.

The *cerebral wasting in dementia paralytica* often differs in degree from that found in progressive senile dementia. In fulminating cases of dementia paralytica death frequently occurs so rapidly that no time is allowed for the removal of the products of neuronic dissolution. On the other hand, in more

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chronic cases of dementia paralytica, the relative finality of the dissolution of the region of higher association and the organisation of the results of extra-neuronic reparative reaction more often result in very marked grades of cerebral wasting than is the case in progressive senile dementia.

As a preliminary to the description of certain selected cases of dementia paralytica which illustrate the more important appearances found in its different types and stages, the writer proposes to briefly refer to four cases which exhibit in the different sexes the essential features presented by the ordinary rapid and slow types of dementia paralytica.

These cases were published in full in the second volume of the *Archives of Neurology* as Nos. 212–215, and their morbid anatomy, in summary, is as follows:

The encephala of the females (Nos. 213 and 215) weighed respectively 985 and 782 grm. (average normal 1,275 grm.), and both in their remarkably low weights and in the simplicity of their convolutional patterns were markedly the brains of degenerates. Those of the males (Nos. 212 and 214) were much below the average normal weight of 1,400 grm., scaling respectively 1,205 and 1,225 grm., but they differed from the brains of the females in being convoluted in a fairly average manner.

Nos. 214 and 215. Rapid spastic cases with very small and alternating pupils.—In both cases there existed relatively little opacity and thickening of the pia-arachnoid, this occupying the fronto-parietal region; and there was only a moderate amount of wasting, which was chiefly visible in the prefrontal region.

Nos. 212 and 213. More chronic cases without knee-jerks.

No. 212. Male.—There was considerable fronto-parietal opacity and generally marked thickening of the pia-arachnoid, except at the occipital pole and on the under and inner part of the temporo-sphenoidal region and the orbital surface of the frontal lobe. In the region of the marked thickening, the pia-arachnoid stripped like a glove from the brain. The wasting was extreme in the *prefrontal region*, marked in the *first temporal gyrus and the inferior and superior parietal lobules*, rather less marked in *Broca's gyrus, and the posterior thirds of the first and second frontal gyri*, moderate in the *ascending frontal gyrus*, slight only in the *outer part of the temporo-sphenoidal and pre-occipital regions*, and almost absent in the *occipital lobe, the lower and*

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inner part of the temporo-sphenoidal region and the orbital surface of the frontal lobe.

No. 213. *Female.*—The pia-arachnoid showed considerable opacity and marked thickening, which was most obvious in the frontal lobe, the first temporal gyrus, and the superior and inferior parietal lobules. The wasting was extreme in the *prefrontal region*, marked in the *first temporal gyrus and the superior and inferior parietal lobules*, moderate in *Broca's gyrus and the posterior thirds of the first and second frontal gyri*, much less in the *ascending frontal gyrus*, and slight or absent elsewhere.

[*Note.*—The terms employed for the cortical regions in the above summary overlap somewhat, but are convenient for brevity of description.]

The above-mentioned morbid appearances agree closely with the clinical course of the dementia paralytica in the several cases. In the two which rapidly broke down, the total amount of intra-cranial fluid, the morbid state of the pia-arachnoid, and the grade of the cerebral wasting were all much less marked than in the two cases of chronic type, in which it may be presumed had occurred a more complete removal of the results of neuronic dissolution, and a greater degree of finality, as regards cell-death, in the degenerative process.

Though such morbid appearances as those just referred to are in average cases very definite and quite readily visible, the determination of regional grades of wasting, and also of the type of convolutional pattern as regards relative simplicity or complexity, is at times attended with much difficulty. This is especially the case when sub-development and wasting occur together, though experience and practice enable error to be largely eliminated.

The writer has, therefore, illustrated on Plates V to VIII, figs. 6 to 11, certain hemispheres of cases of general paralysis, which demonstrate the chief types of wasting and under-development which have come under his notice. In order that they may be the more readily compared with one another and also with the hemispheres of under-developed cerebra and of cases of progressive senile dementia which have already been illustrated, all the photographs are of exactly the same relative size.

As has already been insisted on in both the present and pre-

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vious papers, the most useful preliminary criterion of both the existence and the approximate amount of wasting is the quantity of intra-cranial fluid which is present, as this is practically non-existent in the normal cranium, and as it can only occur in quantity in association with loss of cerebral tissue. The writer here excludes certain grossly obvious pathological conditions unassociated with mental disease in which the blood normally contained in the cerebral vessels and sinuses is largely replaced by intra-cerebral fluid, and also ordinary local gross lesions of the cerebrum in which loss of brain tissue is replaced by intra-cranial fluid, as none of these morbid states form part of the subject which is under consideration.

In the cases figured on Plates I and II, for example, there was no excess of intra-cranial fluid, and there is no cerebral wasting. In the first of these plates are shown the small and very simply convoluted hemispheres of an imbecile with epilepsy, and in the second the very small and simply convoluted hemispheres of a normal degenerate.

In the case figured on Plate III, some excess of fluid existed in association with slight cerebral wasting, and, from the clinical aspect, a mild grade of dementia in an intelligent individual suffering from presenile melancholia. The cerebrum is very large, and is extremely well convoluted, in these details markedly contrasting both with the preceding and the following illustrations.

In the case figured on Plate IV, fig. 4, there existed in the subdural space moderate, and in the sub-arachnoid considerable, excess of fluid. The case is one with a marked grade of progressive dementia, and the wasting depicted in the photograph is obvious.

In the case shown on Plate IV, fig. 5, there existed in the subdural space enormous, and in the sub-arachnoid great, excess of fluid. The case is one of gross progressive senile dementia, and the wasting exhibited in the photograph is grossly obvious. In the preceding case the encephalon weighed 1,150 grm. and the cerebellum, etc., 135 grm., and in the present one the encephalon weighs 1,045 grm. and the cerebellum, etc., 145 grm. It is, therefore, likely that the latter brain was, in its original condition, greater than the former. If, on this basis, the hemispheres in Figs. 4 and 5 were remodelled, the latter would be the larger, and the apparent

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great complexity of the prefrontal region which shows so clearly in the photograph would become less obvious.

These hemispheres clearly illustrate the first of the important points to which the writer wishes to draw attention, namely that *gross wasting markedly increases the* APPARENT *complexity of the convolutional pattern.* If they are compared gyrus by gyrus with the hemisphere shown on Plate III, fig. 3, it will be evident at once that neither possesses anything like either the frontal or the parietal complexity which is exhibited by this case, although on casual inspection they both appear to be more complex.

A further important fact is also shown by the comparison of these hemispheres, namely that LARGER *actual size of a hemisphere decreases the* APPARENT *complexity of the convolutional pattern.* Hemispheres, when studied one by one as they occur at *post-mortem* examinations, are unconsciously inferred to be of about the same size, unless they should happen to be grossly large or very small. Even if certain selected hemispheres are later on compared side by side, the smaller and more wasted examples often *appear* to be the more complex, although actual detailed examination may demonstrate the reverse to be the case. The writer has, therefore, systematically supplemented his examination of cerebral hemispheres by the study of a series of photographs, all taken exactly to scale and of such a size as enabled several to be examined at the same time.

As might be expected, the converse of the last point to which attention has been drawn is also true. SMALLER *actual size of a hemisphere increases the* APPARENT *complexity of the convolutional pattern.* This detail is well exhibited by certain hemispheres, which will now be referred to.

The writer would finally draw attention to the point that whilst in many hemispheres wasting is associated with an unfolding of the affected convolutions, which results in relatively little apparent increase of complexity, *in other cases which exhibit gross wasting the convolutions may lie so closely together that great* APPARENT *increase of complexity results.* This detail is especially evident in the hemispheres illustrated on Plate VIII, fig. 11. These, though very simply convoluted, appear, through the gross wasting and close packing of the convolutions, to be, on first inspection, little inferior in complexity of pattern to the well-developed hemisphere illustrated on Plate III, fig. 3. It is difficult, indeed, even when the fact is known, to believe that

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the magnification is exactly the same and that the convolutional pattern is so grossly different in the two cases. In reality the hemisphere shown in Fig. 3 weighed 680 grm., whereas the hemispheres illustrated in Fig. 11 scaled but 330 and 290 grm. respectively, their combined weight being less by 60 grm. than that of the first hemisphere.

After the above preliminary remarks, the necessity of which will be seen when the different illustrations are compared with one another, the writer will now proceed to describe the hemispheres of the cases of dementia paralytica which are shown on Plates V to VIII.

On Plate V, figs. 6 and 7, are figured the right hemisphere of a chronic case, and the left hemisphere of a more acute case of dementia paralytica. In the former of these (Fig. 6) the pia-arachnoid stripped readily from the hemisphere illustrated, whereas in the case of the left hemisphere, which was the less severely affected of the two, this membrane stripped with considerable difficulty on the postero-inferior aspect. In the hemisphere illustrated in the figure both the regional distribution and the degree of the cortical wasting are well shown.

In the latter (Fig. 7) the pia-arachnoid was very adherent to the subjacent cortex, and decortication occurred on the second temporal gyrus and the pre-occipital region. The distribution and the degree of the wasting, which are stated in the description, are readily visible in the illustration.

A cursory examination of these hemispheres by no means suggests that the former (Fig. 6) possesses a greater complexity of convolutional pattern than the latter.

These cases indicate what is still more clearly demonstrated in the cases illustrated in the next plate, namely, that the degree and the regional distribution of the wasting are the same as is found in progressive senile dementia, and figured on Plate IV, figs. 4 and 5. They also show that, in the absence of *post-mortem* decomposition, the pia-arachnoid is especially adherent in those regions in which, at the time of death, recent and active dissolution of the cortical neurones is occurring, whereas this membrane becomes less adherent to, or readily strips from, the convolutions which have already undergone considerable dissolution, and in which more or less complete organisation of the proliferated non-neuronic elements of the cerebrum has occurred.

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On Plate VI are shown two cases, which are unique owing to the clearness with which they illustrate these details.

Fig. 8, in fact, depicts the different functional regions of the cerebrum with almost diagrammatic clearness. The anterior centre of association is grossly wasted, and the pia-arachnoid over it stripped very readily. This is the region in which, in mental disease, dissolution first occurs, and in which, in advanced cases of dementia, it is the most marked. Further, as has already been stated, it is the only region of the convex aspect of the cerebrum in which, in very early cases of dementia paralytica uncomplicated by *post-mortem* decomposition, adhesion of the pia-arachnoid to the cortex occurs. The psycho-motor area shows some, but much less, wasting, and the pia-arachnoid over this area stripped readily. In the case of the parietal, temporal, and insular centres of association externally, and of the precuneus and the inner part of the temporo-occipital region internally, the pia-arachnoid was very adherent to the cortex, and extreme decortication resulted from stripping (*post-mortem* four and a half hours after death). There was also decortication scattered in irregular patches throughout the callosal convolution, but the visuo-sensory area (projection sphere) was practically intact. It was, unfortunately, impossible to obtain a clinical history of this case for a longer period than one month before death.

On Fig. 9, the same distribution of the cortical wasting, and also a similar distribution of the decortication, are visible, but the differences in the appearance of the several regions are still more gross. The prefrontal region, especially, was to all intents and purposes little more than a firmly organised scar, and cut like soft wood. The case presented an unusual course in having started with a long series of epileptiform convulsions after which the patient rapidly became grossly demented; and this history exactly agrees with the morbid appearances exhibited in the cerebrum. The unusually severe involvement of the sensori-motor area has its clinical counterpart in the series of convulsions which ushered in the disease. In the majority of the cases of dementia paralytica which have come under the writer's observation, the temporal and parietal centres of association are, however, more severely affected than in the sensori-motor area, and this distribution agrees with the usual clinical course of the cases. Such a distribution has also been

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PLATE V. Fig. 6.

Chronic dementia paralytica.

Photograph of the right hemisphere of a case of chronic dementia paralytica, who died after a series of 198 epileptiform convulsions. The figure shows wasting, which is very marked in the prefrontal region (anterior two-thirds of the first and second, and anterior part of the third frontal gyri); marked in the first temporal gyrus, the inferior parietal lobule, Broca's gyrus, and the lower part of the ascending frontal gyrus; fairly marked in the remainder of the sensori-motor area and the superior parietal lobule; and relatively slight in the remainder of the hemisphere, including the orbital surface of the frontal lobe.

History.—Male, æt. 53. Married eighteen years, no children. No family or personal history. In Claybury Asylum suffering from chronic dementia paralytica for nearly three years, during the greater part of which time he was lost to time and place, and wet and dirty in his habits. During the last two years of his illness he had several series of convulsions, and eventually died after a succession of 198 epileptiform fits. Knee-jerks absent. Left pupil greater than right, and both inactive to light. Tremor.

Post-mortem.—Dura: Slightly thickened. S.D.: Recent subdural hæmorrhage and excess of blood-stained fluid. Pia: Fronto-parietal opacity and marked thickening and congestion; strips readily except on the postero-inferior aspect of the left hemisphere. S.A.: Excess. Vents.: L., markedly dilated and granular; IV, granular throughout. Vessels: Considerable thickening of basal arteries. Encephalon: 1,225 grm. Cerebellum, etc.: 145 grm. R.H.: 500 grm.; stripped 460 grm. L.H.: 520 grm.; stripped 480 grm. The right hemisphere was more severely affected than the left. The aorta was exceedingly dilated, of cartilaginous density, and contained a large amount of pearly-white fibrosis and some calcareous deposit. Liver, spleen, and kidneys dense.

Fig. 7.

More acute dementia paralytica.

Photograph of the left hemisphere of a more acute case of dementia paralytica. The figure shows wasting, which is very extreme in the prefrontal region; extreme in Broca's and the first temporal gyri and the inferior parietal lobule; marked in the rest of the sensori-motor area and the superior parietal lobule; and less marked elsewhere, including the orbital surface of the frontal lobe. Decortication exists in the second temporal gyrus and the pre-occipital region, into which parts the morbid process appears to be rapidly spreading.

History.—Female, æt. 38, married. No family or personal history. In Claybury Asylum suffering from dementia paralytica for thirteen months. On admission she was quiet and somewhat lost. She collected rubbish, and she was dirty in her habits. During her residence she had several (chiefly left-sided) convulsions. The pupils were unequal. The right knee-jerk was absent and the left was exaggerated. Facial and lingual tremors. Speech slightly slurred. Died in the last stage of dementia paralytica.

Post-mortem.—Dura: Natural. S.D.: Great excess. Pia: Fronto-parietal opacity; extremely thickened and gelatinous, and very adherent to the cortex. S.A.: Excess. Vents.: L., immensely dilated, the left more than the right, and very granular; IV, dilated and granular, especially in the lower part. Vessels: Natural. Encephalon: 1,045 grm. Cerebellum, etc.: 160 grm. R.H.: 393 grm.; stripped 360 grm. L.H.: 355 grm.; stripped 320 grm. The left hemisphere was more severely affected than the right. Cause of death: Chronic tuberculous pneumonia. Aorta natural. Liver, spleen, and kidneys dense.

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PLATE V.

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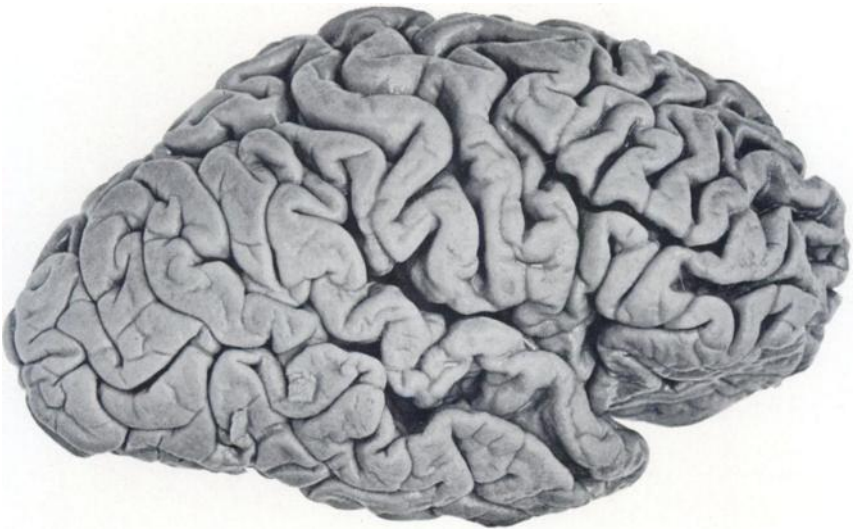

FIG. 6.

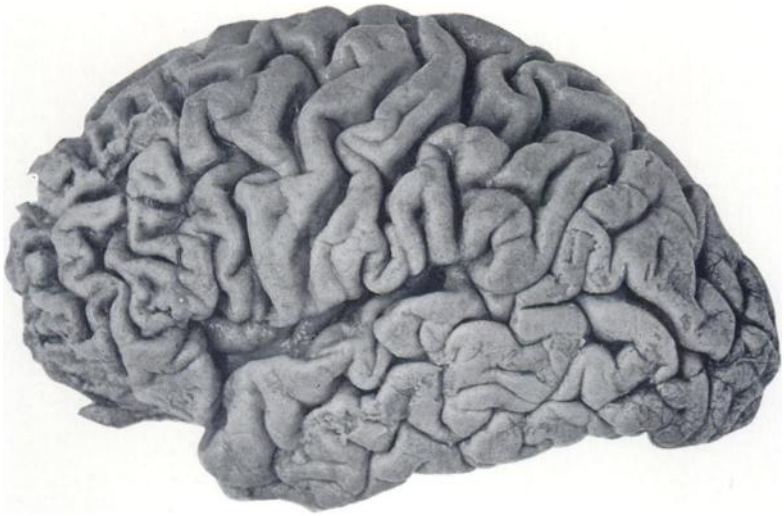

FIG. 7.

To illustrate Dr. J. S. BOLTON's paper.

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PLATE VI.

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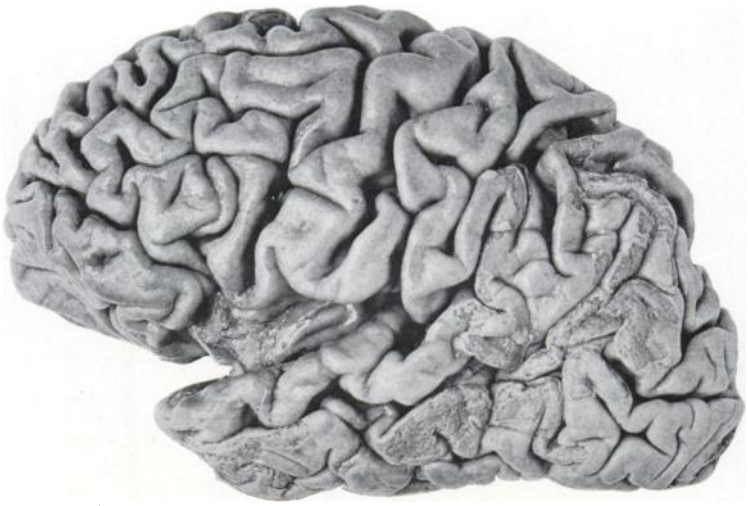

FIG. 8.

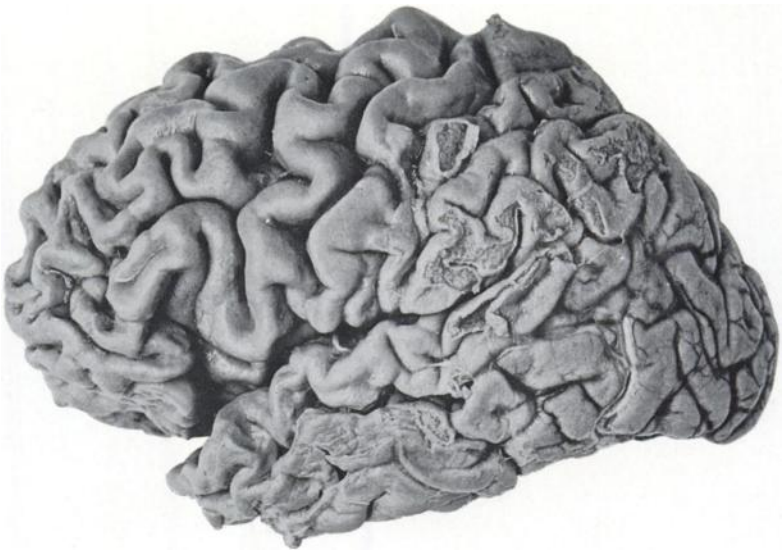

FIG. 9.

To illustrate Dr. J. S. BOLTON's paper.

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PLATE VI. FIG. 8.

Dementia paralytica, showing the order of progress of the morbid process.

Photograph of the left hemisphere of a case of dementia paralytica. The anterior centre of association is grossly wasted. The psycho-motor area shows some, but much less, wasting. The temporal and parietal and insular centres of association are acutely changed (*post-mortem* four and a half hours after death). The upper part of the temporal centre of association, and the anterior part of the parietal, show less acute change and more wasting. The visual projection centre, and the visuo-psychic cortex around it, are intact.

History.—Male, single, private in rifle brigade, stated to be æt. 26. No satisfactory personal or family history, but heredity of insanity denied. Died in Claybury Asylum after a residence of four weeks. Was admitted in a feeble and helpless condition. Tongue tremulous. Right pupil greater than left, and both irregular. The pupils react neither to light nor to accommodation, undergo irregular rhythmic movements, and become eccentric at irregular intervals. Knee-jerks + +. All the limbs undergo clonic contractions, and at times enter into a pseudo-clonus. There are continual masticatory movements of the lower jaw. Patient is somewhat resistive, grossly demented, and wet and dirty.

Post-mortem.—Dura: Natural. S.D.: Remarkable excess. Deposit: Non-hæmorrhagic film, the thickness of tissue paper, on the right vault. Pia: Extremely opaque and almost universally thickened; marked mid-line prefrontal adhesions below the falx cerebri; strips very readily over the frontal region. S.A.: Great excess, especially in the pre-frontal region. Vents.: L., immensely dilated, granular; III, granular; IV, very granular throughout, but especially so in calamus. Vessels: Apparently natural. Encephalon: 975 grm. Cerebellum, etc.: 157 grm. R.H.: 395 grm. L.H.: 395 grm.; stripped 350 grm. Density of liver, spleen, and kidneys increased. Cause of death: Right lobar pneumonia, dementia paralytica.

FIG. 9.

Dementia paralytica, showing the order of progress of the morbid process.

Photograph of the left hemisphere of a case of dementia paralytica. The wasting is very extreme in the prefrontal region, and extreme in the whole sensori-motor region (posterior thirds of the first and second frontal, Broca's, and the ascending frontal gyri), and in the first temporal gyrus, the superior parietal lobule, and the ascending parietal gyrus. The acute degeneration is most marked in the outer surface of the temporo-sphenoidal lobe, the inferior parietal lobule, and the pre-occipital region, but it is marked elsewhere. This distribution shows fairly well in the photograph, but it was much more clear in the actual hemisphere.

History.—Male, æt. 41, clerk. Uncle insane. Mother died of phthisis. Family intemperate. Married six years, no children. Suffered from syphilis in early life, and "took enough mercury to kill a horse." One year before admission to Claybury Asylum patient had a series of convulsions, and was unconscious after the first for twenty-four hours. He had forty-two in four days, and he had fifty or more during the year. He has been in two asylums, with an interval of two weeks at home, during this time. Slight hypospadias. Old scar on glans penis. Resists examination as if afraid of being hurt. Is grossly demented. Does not speak. Is wet and at times dirty. During his residence he rarely spoke, and suffered at times from auditory and visual hallucinations. He had several right-sided and mixed convulsions. He continued helpless and resistive, and wet and dirty, until his death fourteen months after admission.

Post-mortem.—Dura: Natural. S.D.: Great excess; a little lymph between pons and occipital bone. Pia: Marked fronto-parietal opacity and thickening; extreme mid-line prefrontal adhesions. Both on the median surface in the pre-frontal region, to some extent in the prefrontal region externally, and also in the post-central region and the whole of the temporo-sphenoidal lobe, there is marked decortication on stripping. In the remainder of the fronto-parietal region the pia-arachnoid is ballooned out with fluid and strips like a glove from the subjacent cortex. S.A.: Great excess. Vents.: L., considerably dilated; many scattered granulations; IV, granular throughout. Vessels: Apparently natural. The prefrontal region, after hardening in formalin, cuts like soft wood. Encephalon: 1,280 grm. Cerebellum, etc.: 198 grm. R.H.: 535 grm. L.H.: 527 grm.; partially stripped 475 grm. Cause of death: Broncho-pneumonia, dementia paralytica.

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PLATE VII. Fig. 10.

Dementia paralytica. Cerebrum very small and very simply convoluted.

Photographs of the hemispheres of a case of dementia paralytica. Duration about two and a half years. There is much wasting of the fronto-parietal region and of the first temporal gyrus, but this is imperfectly shown in the figure. The hemispheres are very small and very simply convoluted.

History.—Female, æt. 37, married. Father intemperate. Father and sister committed suicide. Patient suffered from tingling of the hands and feet for six months before her admission to Claybury Asylum, where she died of dementia paralytica after a residence of two years. On admission she exhibited marked physical signs of dementia paralytica, and was dull and lethargic and lost to her surroundings. She soon became defective in her habits. She had her first convulsions a year after her admission. She died helpless and grossly demented.

Post-mortem.—Dura: Natural. S.D.: Great excess. Pia: Much fronto-parietal opacity and thickening; marked mid-line prefrontal adhesions. S.A.: Moderate excess. Vents.: L, somewhat dilated, granular; III, granular; IV, markedly granular throughout. Vessels: Natural. Encephalon: 985 grm. Cerebellum, etc.: 138 grm. R.H.: 415 grm.; stripped 385 grm. L.H.: 400 grm.; stripped 373 grm. Wasting: Chiefly marked in the prefrontal region, less severe and fairly general in the rest of the fronto-parietal region and in the first temporal gyrus, and slight or absent elsewhere. Cause of death: Dementia paralytica, cystic kidneys, and secondary morbus cordis.

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PLATE VII.

JOURNAL OF MENTAL SCIENCE, JANUARY, 1908.

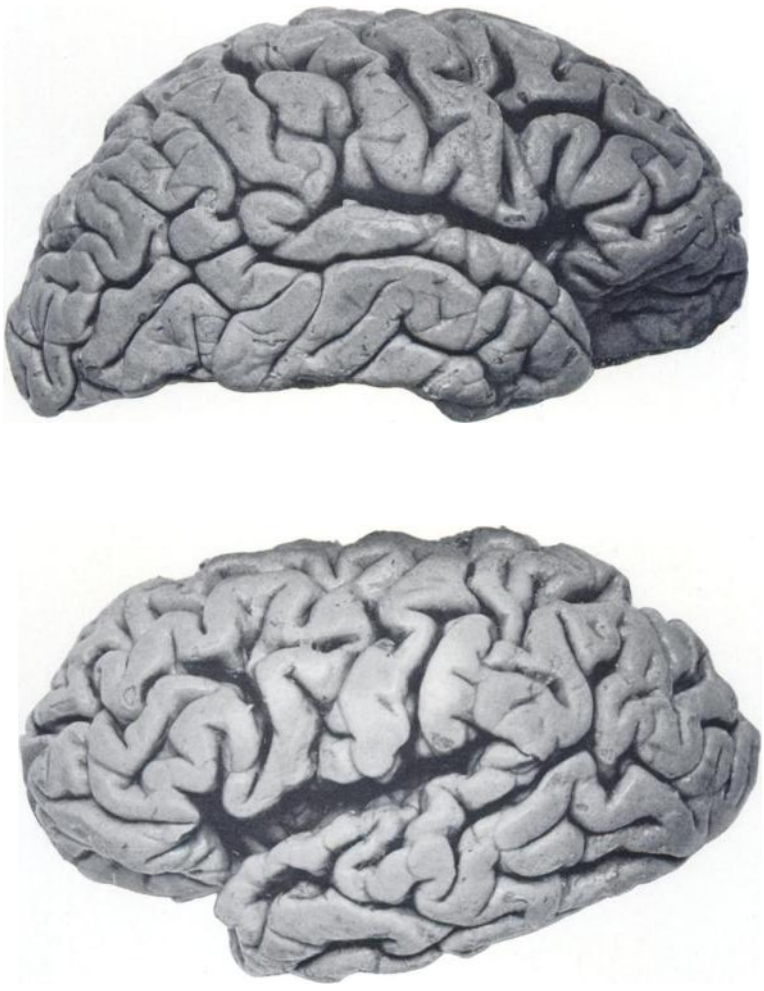

FIG. 10.

To illustrate Dr. J. S. BOLTON's paper.

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PLATE VIII.

JOURNAL OF MENTAL SCIENCE, JANUARY, 1908.

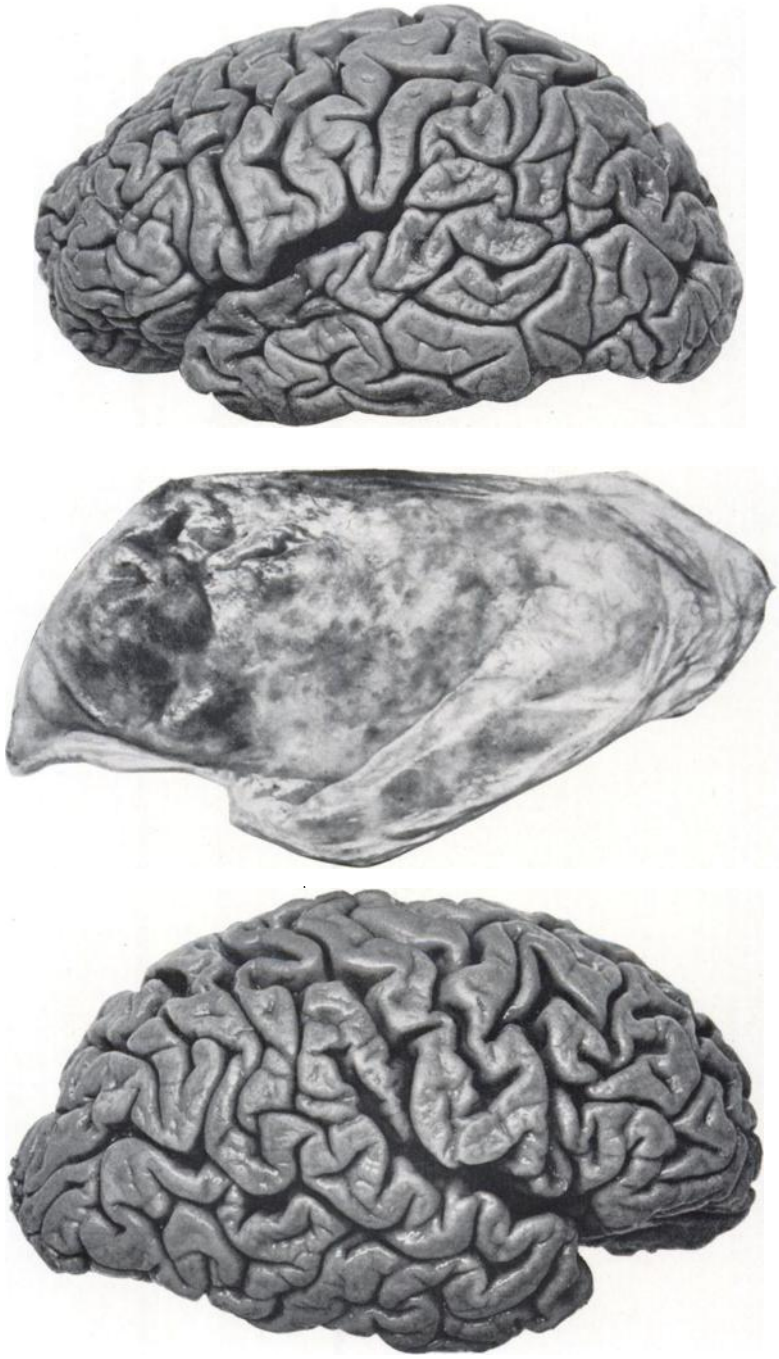

FIG. 11.

To illustrate Dr. J. S. BOLTON's paper.

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PLATE VIII. FIG. 11.

Dementia paralytica. Cerebrum simply convoluted and very small. Subdural deposit.

Photographs of the cerebral hemispheres and of a subdural deposit from a case of chronic dementia paralytica. The illustrations exhibit the essentials of the following description. The right hemisphere shows extremely marked wasting in the fronto-parietal region and in the first temporal gyrus. Of these parts the prefrontal region is the most wasted, and the psycho-motor area, with the exception of Broca's gyrus, is the least. The left hemisphere, which was compressed by the deposit photographed below it, exhibits scattered bronzing and very extreme wasting in the prefrontal region. The anterior part of the first temporal gyrus shows much bronzing and marked wasting. The wasting is otherwise as in the right hemisphere. The hemispheres, apart from the wasting, are very small and simply convoluted (R.H. = 330 grm., L.H. = 290 grm., average normal male = 589 grm.), and the small size, in association with the wasting and the close packing of the convolutions, tends in the photographs to obscure the simplicity of the convolutional pattern.

History.—Male, æt. 41, insurance agent. Intemperance on paternal side. Father's cousin is at present in an asylum. Patient had syphilis at the age of twenty years. Married seven years, six children. The first, fourth, and fifth were stillborn, the second is alive, and the third and sixth died when infants. For two years before his admission patient was excitable and curious in his behaviour, and talked and raved about his business. He had been intemperate, but latterly he was often queer and erratic, although he had had no drink. He slept badly during the last four months, and was eventually certified owing to sudden violence. He resided in Claybury Asylum for three and a half years, and then died of dementia paralytica. On admission he was excited and grandiose and confused, and thought that he was Emperor of the World. Knee-jerks absent. Speech characteristic. Pupils irregular, and left greater than right. A year afterwards he was cheerful and industrious, but was beginning to go downhill. A year later he still exhibited delusions of wealth and strength, but was demented and wet and dirty. He gradually became lost, untidy, destructive, shaky, and feeble, and died in an advanced stage of dementia paralytica.

A monthly record of reflexes and pupillary changes was taken from the sixth to the fifteenth month of his residence. The knee-jerks were absent. Hypotonus (85°) developed in the fourteenth month and continued. Both pupils were very irregular and were absolutely fixed to light. The right gradually decreased during the above period from 3½ to 2½ mm., accommodating to 3 and 2 mm. respectively, and the left gradually decreased from 5 to 4½ mm., accommodating to 4½ and 3 mm. respectively. At this time his tongue was only moderately tremulous and his speech was not grossly characteristic. By the twelfth month of residence patient was distinctly more stolid in his behaviour, but he continued to be grandiose. He devoured pheasants, partridges, bullocks' brains, jellies, honey, and port wine daily. He was as strong as a lion. His mother was a beautiful lady and his father a lawyer's clerk with a carriage and pair.

Post-mortem.—Dura: Natural. S.D.: Enormous excess of clear fluid. Deposit: When the dura is reflected it tends to adhere over the left side, but strips readily; the whole left hemisphere, except for the median half inch about the posterior half, is covered with an old greyish-green deposit, which is ballooned out anteriorly by fluid; the right hemisphere possesses a large amount of loculated S.A. fluid, but there is less on the left side. On removing the brain the deposit readily separates from the dura at the base. It is very loosely attached to the pia. The deposit contains fluid in its anterior part and is here in places hæmorrhagic. The weight of the deposit and contained fluid is 45 grm.; it is dense and pale and fibrous, and behind the cystic cavity it varies from ⅛ to $\frac{1}{16}$ in. in thickness. Pia: Almost generalised opacity and extremely marked mid-line prefrontal adhesions. Vents.: L., enormously dilated, granular throughout; III, granular throughout; IV, extremely granular, especially in calamus. Vessels: Slightly fibrous and very small. Encephalon: 812 grm. Cerebellum, etc.: 108 grm. R.H.: 362 grm.; stripped 330 grm. L.H.: 310 grm.; stripped 290 grm. Cause of death, etc.: Gangrene of right lung; dementia paralytica; very chronic tuberculosis of the small intestine and the mesenteric glands. Severe pigmented scars on the left shin and unpigmented papery scars on the right shin; extremely marked scar on the glans penis just to the left of the urethral orifice. Glands in groins very shotty.

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independently described by Schaffer and by Watson. On the other hand, in ordinary cases of dementia, in which the process of neuronic dissolution is neither so fulminating nor so severe, and in which the centres of lower association are frequently less severely affected than is the pre-Rolandic portion of the cortex, it is less common to meet, during their clinical course, with the grossly aberrant psychic phenomena of lower association which are common in dementia paralytica, and which have already been considered in the present paper (*Journal of Mental Science*, July, 1906, pp. 456–465).

(B) *Cerebral under-development in dementia paralytica.*—In the remaining cases to which reference will be made and which are illustrated on Plates VII and VIII, the question of cerebral under-development in relation to dementia paralytica will be considered. All the cases of dementia paralytica which so far have been described may, for practical purposes, be considered to have possessed cerebra of, at the least, average development.

For comparison with the clinical account of the varieties of dementia paralytica in which it will be shown that not only "normal" individuals, but also high grade aments and even imbeciles exhibit this symptom-complex, it is now necessary to produce cases of dementia paralytica which possess the small and simply convoluted cerebra of mental degenerates.

In Plate VII, fig. 10, are shown the right and left hemispheres of a woman possessing a markedly under-developed cerebrum. These hemispheres are very small and also very simply convoluted, and these details become especially obvious when the photographs are compared with the equal-sized illustration on Plate III, fig. 3. The weights of the right and left hemispheres, after stripping, are respectively 385 and 373 grm., whereas the weight of the average normal stripped hemisphere of the female (based on Huschke's ratio and on F. Marchand's statistics) is about 534 grm.

The hemispheres of this case are so simply convoluted that the marked wasting which exists is not obvious, in spite of the fact that in any, but particularly in small, hemispheres the apparent complexity of convolutional pattern is increased by wasting. Were the hemispheres of this case in their original condition, it is not an exaggeration to remark that the simplicity of their convolutional pattern would appear extraordinary.

In Plate VIII, fig. 11, are shown the hemispheres of a similar

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(male) case of dementia paralytica with an under-developed cerebrum. The central photograph is that of a subdural deposit of long standing, which entirely covered the left hemisphere. Though the patient was of the male sex, the stripped hemispheres weighed respectively but 330 and 290 grm., the pair thus totalling less by 60 grm. than the 680 grm. scaled by the single hemisphere illustrated on Plate III, fig. 3 (average male normal 589 grm.).

Here, again, the simplicity of convolutional pattern is so marked that the very gross wasting which exists is far from obvious. On the other hand, the close packing of the convolutions, in association with the gross wasting, tends to hide the remarkable simplicity of convolutional pattern, and makes it difficult to conceive that the hemispheres are of exactly the same relative size as is that illustrated on Plate III, fig. 3.

(4) *Evidence as to the Relationship between Dementia Paralytica and Mental Disease, derived from a Study of the Clinical Types of Dementia Paralytica.*

In the preceding sub-sections evidence has been adduced as to the frequency of heredity of insanity and of family and parental degeneracy in dementia paralytica, and as to the modification of the death rates of the insane at different ages which results from the exclusion of the cases of dementia paralytica. Further, the writer has indicated the relationship which exists between the morbid anatomy of dementia paralytica and that of progressive senile dementia, and he has drawn attention to the existence of cerebral under-development in certain types of dementia paralytica.

The final evidence, which he purposes to produce in support of the thesis that dementia paralytica is an integral part of mental disease, is derived from a comparison of the clinical types of dementia paralytica with the homologous types of ordinary mental disease, which have already been considered under the heading of "Primarily Neuronic Dementia."

Further experience has confirmed him in the opinion that the following classification of the varieties of dementia paralytica, which was suggested in a previous paper (*Archives of Neurology*, vol. ii), is on the whole satisfactory. It is based on the different grades of cerebral degeneracy which are presented

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by the several types, and thus follows on the lines already adopted in the description of primarily neuronic dementia, though for convenience the order is inverted.

The classification is as follows:

(1) *Dementia paralytica.*

(*a*) Juvenile dementia paralytica.

(1) In imbeciles (low-grade aments).

(2) In high-grade aments, etc.

(*b*) Ordinary chronic dementia paralytica in adult high-grade aments.

(*c*) "Tabetic general paralysis" or dementia paralytica associated with extensive degeneration of (usually afferent) lower neurones.

(*d*) Acute or subacute dementia paralytica in the highest grade degenerates (general paralysis of the text-books).

(2) *General paralysis without mental symptoms.*

Cases of dementia paralytica form a small proportion only of the series of cases under consideration, there being but 23, of whom 14 are males and 9 are females. They thus amount to 5·17 *per cent.* of the 445 cases of dementia, or 3·16 *per cent.* of the total of 728 cases of amentia and dementia.

These cases fall into the following classes:

	M.	F.	T.
Dementia paralytica.			
(*a*) Juvenile.			
(1) In imbeciles	1	—	1
(2) In high-grade aments, etc. .	—	—	—
(*b*) Ordinary chronic in high-grade aments	9	8	17
(*c*) Tabetic general paralysis . .	—	—	—
(*d*) Acute or subacute in highest grade degenerates . . .	4	1	5
Total . . .	14	9	23

In spite of the small number of cases it will be seen that in the acute or subacute type there is the usual preponderance of male cases, whereas the chronic cases show but a slight difference in sex-frequency.

The practically equal sex-incidence in chronic dementia paralytica was noted some years ago by the writer in a previous

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paper. Whilst dementia paralytica is more common in the male than the female sex owing to the greater frequency of syphilis in the former, "stress" is also a more important factor in the male sex, owing to the conditions of civilised life. Acute cases of dementia paralytica therefore preponderate in the male sex and chronic cases in the female sex, with the accidental result that an approximately equal number of chronic cases exist in the two sexes.

On the other hand, the approximately equal sex-incidence in juvenile general paralysis, which was first noted in 1893 by Dr. Wiglesworth, is the natural consequence of the equal sex-incidence of "congenital" syphilis and of the more equal sex-incidence of "stress" in such juvenile cases.

As typical examples of the several varieties into which dementia paralytica has been classified were published by the writer in the paper already referred to, and as their existence is now probably proved beyond dispute, his purpose will be served by an explanatory amplification of the classification, without the insertion of any cases beyond those already briefly summarised in the illustrations.

Juvenile dementia paralytica.—In the *imbecile type* the patient is a well-marked degenerate of congenitally deficient intelligence. At or before the age of puberty a slowly progressive dementia develops under the influence of the "stress" of normal environment. In spite of the deficient durability of the cortical neurones of these cases the development of the dementia is usually slow, as the "stress" which has determined their incarceration in an asylum is so slight that a relatively small amount of immediate injury to the cortical neurones has been produced. It is probable that accident of environment or physical disease has a good deal to do with the exacerbation of symptoms which at times occurs. In one case, for example, the writer has little doubt that the exertion of acting as a golf caddie was the exciting cause of a more rapid progress of the disease, for the patient had for a long time previously remained in a practically stationary condition. In the writer's experience cases of this type frequently suffer from degeneration of the lower neurones and exhibit optic atrophy and tabetic symptoms. They are the probable juvenile homologues of the "ordinary chronic" and "tabetic" types of dementia paralytica.

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In the view of the writer patients suffering from the imbecile type of juvenile dementia paralytica would, if they had not previously suffered from syphilis, have become ordinary examples of the (stationary) premature dementia of marked degenerates.

In the *high-grade ament form* of juvenile dementia paralytica, the patient has originally been of at least average intelligence, and at times appears to have been distinctly well endowed mentally. It is, however, common to find that such patients become "backward" in their studies about the period of puberty. Under what at times seems to be the "stress" of normal environment, but is usually distinctly more severe, *e.g.*, in some cases prolonged over-study, the patient, about the period of puberty or adolescence, develops acute and progressive dissolution of the higher cortical neurones, which, when the morbid process has once got under way, often runs a rapid course. The symptomatology presented by cases of this type is at times identical with that given in ordinary text-book descriptions of adult general paralysis. The writer has formed the opinion, though he expresses it guardedly owing to the lack of statistical evidence, that degeneration of the afferent systems of neurones is less common in such cases than in the imbecile type of juvenile dementia paralytica.

In the view of the writer, the subjects of the form of general paralysis under consideration would, if they had not previously suffered from syphilis, have become ordinary examples of (stationary) premature dementia. In consequence, however, of former infection with syphilis, these cases become the premature homologues of the rapidly progressive adult variety of dementia paralytica, in which, at the period of greatest mental and physical activity, fulminating dissolution of the higher cortical neurones is precipitated under the influence of excessive mental and physical "stress."

Ordinary chronic dementia paralytica.—Whilst any of the well-known types of symptomatology, including epilepsy, may occur in the subjects of the chronic form of dementia paralytica, progressive dementia is the prominent clinical feature, and the course of the case is often so slow that, were it not for the existence of the ordinary physical signs, the condition would undoubtedly often be undiagnosed. Many such cases, in fact, probably die unsuspected at home or in workhouses, for only the cases which cause trouble are likely to be sent to asylums.

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Ideas of grandeur often exist, and the writer has seen several cases which still exhibited this symptom after a residence in an asylum of ten or twelve years. Such cases, as a rule, neither exhibit the acute symptomatology nor provide the antecedent history which occur in cases of the ordinary text-book description, and they are often admitted to asylums when already in an advanced stage of dementia. Two examples of this type of dementia paralytica are described and figured on Plates VII and VIII.

These cases are commonly, if not invariably, high-grade aments, who often exhibit marked stigmata of degeneracy. It is probable, therefore, that, especially in the examples who do not suffer from convulsions, the often prolonged course of the case is due to the same cause as has already been stated with reference to the imbecile variety of juvenile dementia paralytica, namely the readiness with which the cortical neurones are affected by "stress." In such cases there is consequently less immediate dissolution of the higher cortical neurones than occurs in the more fulminating types whose breaking-strain is not readily reached. Hence, when the slight "stress" which has precipitated their breakdown is removed by their being placed under asylum *régime*, the symptoms largely subside, and, unless they should be discharged "recovered" and consequently relapse, these cases usually run a prolonged course.

In the opinion of the writer, such cases would, had they not previously suffered from syphilis, have become examples of the ordinary chronic lunatic with moderate (stationary) dementia, and a general symptomatology appropriate to their mental constitution.

"*Tabetic general paralysis.*"—In this form of dementia paralytica dissolution of the higher cortical neurones is associated with a more or less extensive degeneration of (usually afferent) systems of lower neurones. Owing to the definite neurological symptomatology in well-marked cases, it is desirable that these examples should be considered a special type, as otherwise dementia paralytica would require to be artificially sub-divided into (*a*) dementia paralytica and (*b*) dementia paralytica with involvement of lower neurones. This is, however, undesirable, as many, if not the majority of, cases of dementia paralytica exhibit some slight affection of these neurones when they are

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submitted to systematic histological examination. As a rule, however, when the affection of lower neurones is well marked, the cases are either the rare examples of the imbecile variety of juvenile dementia paralytica or are fairly high-grade degenerates who, apart from involvement of the lower neurones, would fall into the class of "ordinary chronic dementia paralytica."

The writer thinks it probable that, had they not previously suffered from syphilis, certain of these cases would have become examples of ordinary chronic insanity with moderate (stationary) dementia, and the remainder would have suffered from a chronic process of dissolution of certain systems of lower neurones, and would have thereby come under the purview of the neurologist rather than of the alienist.

Acute or subacute dementia paralytica in the highest grade degenerates (general paralysis of the text-books).—It is unnecessary to refer here to the classical symptomatology of this form of dementia paralytica, and particularly so as it has already been critically discussed under the subject of "Mental Confusion and Dementia" in an earlier section of this paper (*Journal of Mental Science*, July, 1906, pp. 456–465).

It may, however, be pointed out that cases of this type are, by cerebral development, frequently so little prone to suffer from dementia, that only the severest forms of "stress" (mental and physical over-strain, business worries, alcoholic and other excesses, etc.) are able to precipitate the onset of dissolution of the higher neurones of the cortex. In such cases, where highly evolved cortical neurones have long been strained to breaking-point, fulminating dissolution occurs when this has been overstepped, and a rapid case of dementia paralytica ensues.

In the view of the writer, if cases of this type had not previously suffered from an attack of syphilis, they would either have become temporarily insane, or would have developed a more or less marked grade of non-progressive dementia.

General paralysis without mental symptoms may perhaps be considered the very highest (and non-certifiable) grade of the form of mental disease under consideration. Though well known to neurologists, this condition does not fall into the sphere of alienism, although certain rare cases of arrested or recovered general paralysis might be included under the term. Such latter cases are, however, more likely to be examples of what might be called a premature onset of dementia paralytica, in which the

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"stress" to which the cerebrum had been subjected had sufficed for the production of symptoms, but had not been severe enough to cause an appreciable degree of dissolution of the higher cortical neurones. In these cases definite dementia paralytica would be expected to ensue at some future time, provided that the patient were again subjected to "stress" beyond the resistance of his cerebrum.

If, however, it be taken for granted that *no* mental symptoms, rather than *no certifiable* mental symptoms, exist in such cases, it is preferable to employ the term "general paralysis in the sane."

The writer has necessarily excluded *senile dementia paralytica* from his classification, as cases of this kind are usually complicated by the existence of senile or prematurely senile degeneration of the cerebral arteries of a grade which might in itself result in the development of progressive senile dementia. Such cases, in other words, as a rule, combine both the morbid anatomy and the symptomatology of dementia paralytica and of progressive senile dementia.

In these cases, in the experience of the writer, the attack of syphilis has usually occurred at or after maturity, and its influence has chiefly been in the direction of a gross exacerbation of normal senile vascular degeneration. This is shown by the presence of well-marked dilatation and pearly fibrosis of the aorta and of the larger and medium arteries, together with extensive fibrosis of the smaller arteries (particularly in those of the cerebrum), and a moderate amount of calcareous deposition in the arteries generally.

The cerebral morbid anatomy of such cases, whilst suggesting dementia paralytica, is frequently that of progressive senile dementia, probably in consequence of a lesser capacity for reparative reaction on the part of the non-neuronic elements of the encephalon than exists in ordinary dementia paralytica. The clinical symptoms presented by such cases agree with the morbid anatomy in being chiefly those of progressive senile dementia, although dementia paralytica is suggested both by the physical signs which are present and by the type of mental confusion which is exhibited.

SUMMARY.

It is beyond the expectation of the writer that the evidence

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which he has collated should finally settle the vexed question of the relationship of dementia paralytica to mental disease. He, however, hopes that he has at any rate stated a case which will justify the attitude he has adopted.

In brief, he considers that dementia paralytica is a branch of mental disease, and that the subjects of this form of mental disease would, if they had not been syphilised, have suffered from one or other of the types of primarily neuronic dementia. He is further of the opinion that former syphilis is a necessary antecedent to dementia paralytica.

With regard to the first question, he has shown, by a study of the death rates in mental disease at different ages, and by a comparison of these death rates with the homologous death rates in the corresponding general population, that the exclusion of the general paralytic population of an asylum leads to the result that lunatics (particularly those of the male sex) have an extraordinarily low death rate between the ages of thirty-five and fifty-four. If, on the other hand, the general paralytic population is included in the total lunatic population, this result is not apparent.

He has also pointed out that the morbid anatomy and the pathology of dementia paralytica do not differ in their essential features from those of progressive senile dementia. He has further shown, by a classification of the types of dementia paralytica and a comparison of these with the varieties of primarily neuronic dementia, that the two series are homologous.

On these various grounds he has based his contention that dementia paralytica is a branch of mental disease. As confirmatory evidence he has pointed out the high percentage of heredity of insanity and of parental and family degeneracy which can be obtained in cases of dementia paralytica, and he has shown that cerebral under-development occurs in certain types of this form of mental disease.

With regard to the second question, he has indicated his reasons for considering that former syphilis is a necessary antecedent to dementia paralytica. He is of the opinion that the ordinary sane individual and the ordinary psychopath or potential lunatic, if possessed of cortical neurones of average durability, may suffer from syphilis with impunity as regards the later onset of dementia paralytica, and he considers that the same statement may be made with regard to the syphilised

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lunatics with little or no dementia, who are fairly common in asylums. On the other hand, he holds that a psychopath who possesses cortical neurones of subnormal durability, and who, apart from an attack of syphilis, would develop a moderate grade of dementia, would, after an attack of that disease, sooner or later suffer from one or other of the types of dementia paralytica.

He thinks that the important feature in which dementia paralytica differs from progressive senile dementia consists in the possession, by the subjects of former syphilis, of a permanently enhanced capacity of reparative reaction on the part of the non-neuronic elements of the encephalon. In both cases neuronic dissolution and non-neuronic reparative reaction occur *pari passu*. In the case of dementia paralytica the latter is more or less intense, and vascular degeneration is relatively slight; in the case of progressive senile dementia the latter is relatively feeble and vascular degeneration is relatively severe. He would illustrate this point by a coarse analogy, comparing dementia paralytica to certain types of progressive renal cirrhosis and progressive senile dementia to senile renal cirrhosis.

On these grounds he includes dementia paralytica and progressive senile dementia under the common group of "Progressive and Secondary Dementia."

(*To be continued.*)

Observations on the Opsonic Index to Various Organisms in Control and Insane Cases.

By C. J. SHAW, M.D., Senior Assistant Medical Officer, Montrose Royal Asylum, formerly Assistant Medical Officer, Perth District Asylum, Murthly.

IN Wright's earliest researches on the opsonic body in human blood serum he used various strains of staphylococci. He found in various forms of staphylococcal invasion, such as acne, furunculosis, and sycosis, that the index of the patient so infected was lower than that of an ordinary healthy individual to the particular organism causing the disease. By his method of inoculation of a vaccine made from the infecting organisms

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