ON PALILALIA.

BY MACDONALD CRITCHLEY, LONDON.

AMONG the more unusual disorders of speech the phenomenon of palilalia occupies, in spite of its great interest, an undeservedly meagre place in the neurological literature of this country. One understands by this term a variety of reiteration, whereby the patient repeats many times a word, phrase or sentence which he has just spoken. In addition the speech tends to be uttered more and more quickly and with less distinctness, so that the latter part may be almost inaudible. The patient is fully aware of his defect but cannot as a rule control it.

Palilalia (from *παλιν*, again, and *λαλία*, speech) was first described as such by Souques¹⁸ in 1908, although it is possible that the phenomenon spoken of earlier by Brissaud³ as "auto-echolalia" was of the same nature.* Since that date a score or so of cases have been recorded, occurring in two main types of disorder—in postencephalitic Parkinsonism and in cerebrovascular disease. In this present paper, seven more cases are added to the literature, together with anatomical verification in one instance.

PERSONAL CASES.

A. *The Postencephalitic Group.*

CASE 1. R.G., male, age 40, was admitted to the National Hospital, Queen Square, under the care of Dr. Kinnier Wilson, in March, 1927. In April, 1925, he had sustained his acute attack of epidemic encephalitis, and a few weeks after the febrile phase the symptoms of Parkinsonism gradually supervened. In September 1926, the speech disorder first appeared, with some suddenness. Whilst at the cinema he found himself reading aloud *over and over again* the captions of the films; his wife, getting annoyed, dug him in the ribs and said "For God's sake, Bob, shut up," but all the patient could reply was "I can't shut up—I can't shut up—I can't shut up—" This involuntary repetition of phrases persisted up to the time of his admission. When he was seen in hospital his physical state was one of Parkinsonism of moderate degree. There were no psychical changes, the patient in fact being interested in and even amused at his own defect.

His palilalia was entirely involuntary, and occurred during spontaneous speech as well as in replying to questions; it occurred equally during emotional utterances (swearing, etc.) and during propositionizing. The number of repetitions varied somewhat, being usually four to eight, but even when no words were audible his lips would be seen moving as if in silent utterance.

CASE 2. J. J., male, age 18, sustained an attack of acute encephalitis in August 1925. Since then he has become lethargic, and untidy in his habits. On attending the out-patient department of the National Hospital (Dr. Walshe) eighteen months later, he presented the characteristics of advanced Parkinsonism. He was kept under regular observation,

* " . . . leurs réponses sont souvent de simples monosyllabes ou de petits membres de phrase qu'ils répètent plusieurs fois de suite. On pourrait nommer *auto-écholalie* ce singulier phénomène."

his physical state remaining very much the same. On May 3, 1927, he was observed to show palilalia when talking. The patient himself was conscious of the defect and stated that it had been present for the past two weeks only. The palilalia was not marked, was inconstant, and was not present for any phrases other than monosyllables.

- CASE 3. W. D., male, age 33, attended the out-patient department of the National Hospital with postencephalitic Parkinsonism. His acute attack had occurred three and a half years previously. Both the patient himself and his family had noticed an increasing indistinctness in his speech, and also that he would frequently repeat several times a word or part of a sentence. This feature was not constant, being most marked for short phrases and monosyllables.
- CASE 4. D. B., age 43, out-patient under the care of Dr. Adie, Queen Square, sustained an attack of acute encephalitis in August 1925, which was gradually followed by Parkinsonism. "Speech difficulty" had been present for one year. On examination he revealed the characteristics of an advanced case of postencephalitic Parkinsonism. In addition he had a very gross palilalia which was constantly present during conversation. His voice was low and monotonous and became almost unintelligible towards the end of a sentence. The patient exhibited a definite difficulty in starting to speak—a difficulty of which he was very conscious. There were no repetitions when he counted aloud, and none when he swore in anger. Owing to his illiteracy reading tests could not be made satisfactory.

These four cases illustrate the association of palilalia with postencephalitic syndromes. They may be compared with other such cases quoted by Graziano, Marie and Lévy,¹² Sterling,¹⁹ Volpi-Ghirarderi and Tarozzi,²⁵ Agostini,¹ Vivaldo,²⁴ Rordorf and Cacchiararo,¹⁷ Thurzo and Katona,²¹ Giraud and Guibal.⁹ In the first and fourth cases the palilalia was constantly present, but in the other two it varied in degree from day to day and also during conversation, only certain phrases and words being repeated.

B. The Cerebral Arteriopathic Group.

- CASE 5. J. J. E., male, age 80, was first seen by me on March 1, 1927, at St. Marylebone Hospital, where he had been admitted some months previously for "senile decay, with epithelioma of mouth." He was a thin, cachectic old man, slow in cerebration and somewhat demented. His memory was grossly impaired and he was disoriented in time: his expression was wooden and "mask-like," with the mouth gaping in a vacuous fashion. There was a well-marked arcus senilis; his radials and temporals were hard and tortuous; B.P. 145/90. He showed some emotional instability, and laughed easily without adequate provocation. His pupils were small, equal and sluggish in their reactions; the eye movements were full but slow. Facial apraxia was evidenced by inability to protrude the tongue on command, although it could be done when not required. The limbs were rigid, especially proximally, a condition which varied somewhat in intensity according to the amount of passive force used in manipulating them. There was no muscular weakness; some tremor was present in the hands. The tendon jerks were brisk and the plantar responses were flexor. The patient was doubly incontinent.

Throughout the examination he betrayed an intense motor restlessness; he was continually fingering the lapel of my coat, or tugging at his bedclothes. The nurses testified to his agitation, and complained that he was continually "in and out of bed."

His speech was slow, indistinct and dysarthric, and there was definite palilalia. In reply to my greeting he said "Good morning, good morning, good morning . . ." and when questioned concerning his health, replied "pretty well, pretty well, pretty well."

The patient grew progressively weaker and finally died on May 9. An autopsy was obtained and the brain given me for examination.

The cerebrum as a whole was shrunken; there was an excess of subarachnoid fluid over the convolutions. The meninges were thick and milky but stripped easily. The gyri were somewhat atrophic, and there was some broadening of the sulci. A moderate degree of arteriosclerosis was present but was almost entirely limited to the basilar and posterior cerebral vessels; the other arteries were comparatively healthy. The total brain weight was 1,240 gm. (left hemisphere, 550 gm.; right hemisphere, 525 gm.; pons, midbrain and cerebellum, 165 gm.).

On section, the basal ganglia were seen to exhibit an *état criblé* in moderate degree; none of the lacunæ attained unusual dimensions: the cortical grey matter was not narrowed, but the central nuclei appeared shrunken, and were compressed by a marked dilatation of the lateral ventricles.

A small focus of old softening lay in the middle of the right external capsule; a second larger area was present in the left lenticular nucleus, close to, but not invading, the genu of the internal capsule. This softening had burst downwards and medially, so as to communicate with the ventricle (*see* Fig. 1). A third, very small focus, was seen in the right half of the upper pons, near the middle line.

No pathological change was detected in the cerebellum or midbrain, or in the corpus callosum.

CASE 6. C. O., male, age 60, was first seen by me at St. Pancras Hospital on March 2, 1927. His was a typical, advanced case of pseudobulbar palsy (spontaneous laughter and crying, facial overaction during movement with fixity during repose; bilateral rigidity and weakness of limbs, dysarthria, double Babinski responses). His blood pressure was 175/115, and his radials were grossly hardened and tortuous. His mentality was dull, cerebation was slow, and his memory obviously impaired. The speech was dysarthric and in addition was characterised by palilalia.

CASE 7. A. B., female, age 40, was admitted to the National Hospital, under the care of Dr. Kinnier Wilson. Her husband gave a story of two and a half years slow and progressive mental deterioration and emotional instability.

Her expression was immobile, her eyes staring; all her voluntary movements were slow and somewhat weak. There was rigidity in all four extremities. She found difficulty in swallowing solid foodstuffs. Some spontaneous laughter was noticed. The radial arteries were not diseased, but the retinae showed the characteristics of well-marked arteriosclerosis with a superimposed retinitis. Blood pressure 260/160.

Speech was high-pitched, monotonous and breathless; the words were not articulated distinctly and there was a slight superadded nasal element. The patient showed palilalia at times, particularly for monosyllables; phrases and sentences were rarely repeated.

Cases 5, 6 and 7 were obviously instances of diffuse vascular disease of the brain, characterised by many of the manifestations of the pseudobulbar syndrome. The presence of palilalia is on a par with that recorded by Trénel and Crinon,²² Dupré and Le Savoureux,⁵ Foix and Schulmann,⁷ Alexander, and Foix and Chavany.⁶ Souques' original case concerned a woman of 59, whose speech disorder had followed a sudden left hemiplegia. There was a definite mental enfeeblement but no manifestations of pseudobulbar paralysis.

DIFFERENTIAL DIAGNOSIS.

There should be no real difficulty in accurately diagnosing palilalia if the essential features of the phenomenon—self-repetition, acceleration of speech and increasing inaudibility—are kept in mind.

Confusion is, however, conceivable with such conditions as *reiterative aphasia with recurring utterance*, and with certain cases of *stammering*. The explosiveness of the latter defect is, however, unlike palilalia, and the fact that the repetition is one of syllables rather than of words or phrases should be a clear differential point.

Palilogia is the term sometimes applied to that form of rhetoric whereby a word or sentence is deliberately repeated for the purpose of emphasis, or as a subconscious act of mental irritation or peevishness; here again confusion is scarcely possible. In cases of *verbal perseveration*, words or phrases may be repeated, but in this case it is the *same* word or phrase which is repeated, as though the original idea was persisting for an undue length of time in the patient's mind to the exclusion of fresh incoming ideas. The persistence of the same reply to different questions at once distinguishes verbal perseveration from palilalia. In *echolalia* the patient repeats the statements or questions put to him, with or without a change of pronoun. There is no attempt on the patient's part at mental co-operation with the interlocutor. As pointed out by Brissaud, palilalia is really an *auto-echolalia*.

Some psychotics—especially cases of dementia præcox—may repeat the same sentence over and over again *ad nauseam*: such a phenomenon, spoken of by alienists as *verbigeration*, is an instance of verbal stereotypy. With such cases there is usually a wealth of accompanying gesture so that the speech takes on the appearance of a rhetorical declamation; in other cases again the words are intoned in an affected litanical or ritualistic manner.

THE CLINICAL ASPECT.

Palilalia entails the involuntary repetition two or more times of a word, phrase or sentence just uttered. It occurs equally during spontaneous speech and in reply to questions. With succeeding repetitions the words tend to be clipped and abrupt, the voice to become lower and less audible, while the rate of speaking accelerates. The normal articulation may, however, be preserved.

A point of great interest and importance lies in the fact that the palilalia (as with stammering) disappears during pre-formed speech automatisms, as for instance, when the patient reads aloud, sings or recites. Thus if the patient is ordered to repeat from memory some well-known speech pattern, such as the Lord's Prayer, the alphabet or a nursery rhyme, there is no trace of repetition. Palilalia may occur not only during so-called intellectual speech but also in emotional, interjectional speech; thus oaths and exclamations may also show the characteristic palilalic reiterations. On the other hand, the palilalia may be completely absent from emotional utterances, as in Case 4.

Palilalia may be constant in appearance, but frequently it varies in intensity from time to time. Particularly is this true of the postencephalitic group (*vide* Cases 2 and 3). Sterling emphasizes the fact that when variability exists it seems to depend not so much upon the kaleidoscope of the emotions as upon the physical state, for when his patients felt better the palilalia was

less, and conversely it was more evident when the tremor, rigidity and helplessness were greater. Agostini has noted that the speech disorder is always intensified when the patient is taken off hyoscine treatment. Similarly in the arteriopathic group the palilalia may abate when the patients' physical state improves (*vide* Case 6). While it appears then that the influence of the emotions plays a minor part, it is equally certain that the speech disorder can, in some measure, be checked for a time when the patient's attention is focussed upon it, and when a strong effort of will is exercised.

The number of repetitions varies with individual cases, and with the length of the phrase repeated; in general, the shorter the phrase the greater the number of repetitions. Sometimes as many as twenty-one reiterations may be heard, whilst the lips are seen to continue forming additional but inaudible repetitions (*palilalie aphone*). The palilalia is particularly noticeable when the patient's utterances are confined to one sentence. Where his replies or spontaneous diction comprises two or more sentences, usually—though not invariably—it is the last sentence alone which is repeated.

In all the recorded cases, in which particular search has been made, there has never been found any evidence of writing-repetitions (paligraphia) or of repetitions of movements executed by the patient himself (palikinesia). In a few instances, particularly in the pseudobulbar cases, there has been a hint of echolalia (in that the patient seemed to imitate and repeat several times what was said to him). Pick¹⁵ indeed classified palilalia into two groups, according as echolalia coexisted or not. This feature is never a marked or a constant one, however. Very rarely the patient tends to imitate two or three times the actions of the bystanders (echopraxia). Thus Foix and Schulmann have noticed a suggestion of this phenomenon in the patient's handshake.

With regard to the concomitant manifestations of palilalia, it is important to note one negative and one positive feature; firstly, the absence of aphasia in the accepted sense of the word. Thus there is no difficulty in the comprehension of spoken or written speech; internal speech is intact, the power of propositionizing is retained, and the patient may have complete insight into the nature of his disorder. Secondly, the frequency with which certain psychological disorders coexist, viz., emotivity and intellectual impairment. The emotivity is evidenced both in the encephalitic and arteriopathic cases by rapid vacillations in the emotional content and particularly in its physical affect—laughter or tears. Intellectual loss has been present in most of the recorded cases; in the pseudobulbar group there is usually a global dementia with concentric narrowing of the mental field. In the encephalitic group, there too has been noted a slowing-up of cerebration and a restricted mental horizon—so-called 'mental viscosity' or 'bradyphrenia.'

The physical accompaniments always take the form—in the post-encephalitic instances—of the Parkinsonian syndrome. Other features may also be present, such as oculogyric crises or respiratory disorders, but such

phenomena are always superimposed upon a state of Parkinsonism, when palilalia coexists. In the case of cerebrovascular disease, the commonest associated feature is the pseudobulbar syndrome; a history of multiple strokes may or may not be obtained; there is sometimes an associated unilateral or bilateral weakness of the extremities; the earlier instances of palilalia occurred more particularly in pseudobulbars with predominant left-sided hemiplegia, and it was at one time supposed that palilalia was evidence of uncompensated activity of the left brain. The record of subsequent cases (Foix and Schulmann and others) and the advent of the postencephalitic cases, disproved the theory and at the same time demonstrated that palilalia might occur with predominant right-sided weakness.

LOCALISATION.

Although, as mentioned above, the majority of the recorded cases have occurred either in pseudobulbars or in postencephalitics, it must be pointed out that it has also been met with in Alzheimer's disease (Frey⁸), the relationship of which to cerebral arteriopathy is still not beyond suspicion. Palilalia has also been described in the 'idiopathic' variety of paralysis agitans (Pick, Maillard¹⁰), arising independently of vascular disease or epidemic encephalitis. Foix and Chavany have, moreover, recorded a variety of palilalia (*palilalie syllabique*) in a man of 72, whose brain shewed disseminated foci of sclerosis throughout the cerebrum, which the authors suggest were ischæmic in origin. This case, however, can scarcely be included within the present category as the speech disorders differed in several minor though important particulars from the classical description.

Owing to the paucity of pathological evidence, the anatomical basis of the disorder has been largely speculative. Furthermore, the very nature of the disorders in which palilalia is encountered are those in which we expect to find multiple or diffuse pathological lesions. Marie and Lévy indeed went as far as to state that palilalia of necessity indicated a diffuse pathological process.

Some of the writers upon this subject have been inclined to look to the basal ganglia for the site of the causative lesion. Following the views of Oppenheim and the Vogts, who attributed to the anterior putamen the functions of a speech regulator, Pick and Sterling have postulated lesions in this situation. Marie and Lévy, and also Sterling in his later paper, are less definite in their statements and suggest that palilalia is an expression of disease in the central grey nuclei and subthalamic centres.

Autopsy evidence has been forthcoming in but few cases, viz., in Trénel and Crinon's case, in three of Pick's, and in Case 5 of the present series.

In 1912 Trénel and Crinon showed before the Société de Neurologie a woman, age 51, with a pseudobulbar syndrome and palilalia. There was a tendency, moreover, towards echopraxia. Two years later at a meeting of the same Society, in which a similar case was shown, Trénel²³ referred briefly to

his patient who had since died. There had been found post-mortem a sclerosis strictly limited to one cerebellar hemisphere ; in addition, the corpus callosum was definitely diminished in volume and contained numerous scattered *foyers* of degeneration.

Two out of Pick's three cases were examples of cerebral vascular lesions, in subjects aged 78 and 35 respectively. In the former, there were several foci of softening, one in the right putamen, another in the head of the left caudate, and a third in the left subthalamic region. A fourth foyer lay in the anterior portion of the corpus callosum ; a hæmorrhagic cyst was situated at the foot of F¹ and F², implicating the adjoining precentral gyrus. The latter case showed clinically a right-sided hemiplegia due to a large softening in the territory of the left Sylvian artery. The right hemisphere showed some convolucional atrophy and ventricular dilation. Pick's remaining case was one of paralysis agitans occurring in a man of 65. Autopsy revealed no microscopic abnormality, but there was a generalized outfall of cells from the right caudate and lenticular nuclei.

The case (No. 5) reported in this paper also exhibits multiple lesions of a vascular nature scattered throughout the hemispheres and brainstem. There were three small areas of softening, one in the right external capsule, one in the right half of the upper pons, and a third and large focus in the left lenticular nucleus, communicating with the lateral ventricle. In addition there were many lacunes of disintegration in both hemispheres ; the whole cerebrum was shrunken, there was a slight degree of convolucional atrophy and some internal and external hydrocephalus. The cerebellum and corpus callosum were normal.

The lack of sufficient anatomical evidence does not warrant, therefore, any dogmatic statement as to the underlying pathology of the condition, but the above case—viewed in conjunction with Trénel's and Pick's—supports the surmise that palilalia is the expression either of a diffuse morbid process, or of the summation of numerous isolated lesions.

PATHOLOGICAL PHYSIOLOGY.

Many views have been put forward to explain the pathogeny of palilalia. Some writers have regarded it as a psychotic manifestation by bringing it into line with the well-known instances of verbal stereotypy or catatonic speech. Other writers, amongst them Meige,¹⁴ have suggested that palilalia is a variety of stammering (*bégaiement*). Much more attention has been paid to the hypothesis of Trénel and Crinon, who suggested that palilalia bears the same relationship to normal speech as spasmodic laughter or crying does to the normal emotional content. "Nous croyons qu'il s'agit d'un parler spasmodique relevant d'une même cause que le pleurer et le rire spasmodiques dont cette malade est également atteinte."

It is necessary at the outset to emphasize that palilalia is a disorder of speaking rather than speech. ("Un trouble, non pas du langage mais de la

parole"—Dupré and Le Savoureur). There is no disorder of internal speech, the defect being purely in the motor or effector mechanism.

Before we enquire further into the nature of palilalia it is necessary to recall the points in common between pseudobulbar palsy and postencephalitic Parkinsonism, the two disorders par excellence with which palilalia is associated. Both these syndromes are characterised by generalised rigidity of greater or less degree, by a more or less asthenic muscular state and by an upset in the ratio of automatic to volitional movement. Thus in both conditions we find the play of volition or quasivolitional activity reduced to its minimum, as exemplified by the fixed and wooden facies, the statuesque pose, the poverty of spontaneous movement and lack of play of gesture. Movements themselves are slow in initiation and execution (bradykinesia).

With regard to the movements concerned with the more automatic activities such as speaking, walking and running, we may trace in some cases evidence of exaggerated activity. There is, moreover, an obvious delay in checking the movement at will, which contrasts with the delay in the genesis of spontaneous movement. It is as though a certain static energy had to be generated in order to overcome the primary inertia or 'unwillingness' to move; once overcome the movement proceeds automatically with more and more ease so that it becomes checked voluntarily only with difficulty. These features are well illustrated in the gait; there is a preliminary difficulty in 'getting off the mark'; once started, however, the patient progresses with more and more rapid steps over which he may actually lose control, hence the propulsion and festination characteristic not only of the Parkinsonian but also of the arteriopath.

Somewhat analogous to this phenomenon is palilalia. Babinski and Mlle. Lévy have pointed out that many of these patients exhibit a variety of mutism alternating with their palilalia, as though loath to embark upon the act of speaking. Once this unwillingness is mastered the speech is emitted in monotonous form; the rate of utterance tends to accelerate, and as in the case of walking, the patient finds difficulty in ceasing. It is easier to go on talking than to make the effort of stopping.

Palilalia tends to be absent when there is a smooth and orderly progression of successive concepts; thus, when the patient spontaneously utters a speech composed of two or more sentences it is only the last sentence which becomes implicated in the palilalia. The same is true of replies comprising two or more sentences. It is as though the act of speaking can proceed normally only so long as the patient 'has something to say'; as soon as his thought is expressed palilalia puts in an appearance; for the same reason short uniphrastic utterances are the most likely to be repeated, whilst reading aloud or recitations are enunciated normally.

Such a view is closely analogous to that of Claude, who in describing a somewhat allied speech phenomenon, employed the term "verbal antepulsion and festination." Claude described, under the title of progressive paroxysmal

tachyphemia, a condition in which there was an acceleration of speech with repetition of a word or fragment of a sentence, in cases of paralysis agitans. Unlike palilalia, however, these repetitions were also present during pre-formed speech automatisms as when counting or reciting from memory.

This latter proviso must be regarded as a criterion of palilalia; it dissociates clearly voluntary from automatic speech: whilst in the former case the speech is characteristically festinant and repetitive, speech automatisms on the other hand present none of the marks of palilalia. In this way palilalia may be compared to the paradoxical kinesia of the Parkinsonian who, unable to walk without the utmost difficulty, can nevertheless run, dance or cycle with comparative ease. If we regard walking as an instance of a 'most automatic' or 'least voluntary' movement, then running, dancing and cycling may be regarded as pre-formed automatisms, or movement-patterns. Similarly, reading aloud and singing must be considered as speech automatisms, easier of execution from the mechanical standpoint than spontaneous conversational speech.

*For permission to report these cases I beg to thank the Physicians of the National Hospital, Queen Square; it is a pleasure, moreover, to acknowledge the kindness of Dr. Hood and Dr. Feldman, the medical superintendents of St. Mary-le-Bone and St. Pancras Hospitals, in permitting me to quote Cases 5 and 6.

REFERENCES.

- ¹ AGNOSTINI, C., *Ann. dell' ospedale psichiat. provin. in Perugia*, 1925, xix, 111.
- ² ALEXANDER, *Rev. neurol.*, 1925, xxxii, 319.
- ³ BRISSAUD, *Lecons*, 2me série, 1899, xxxii, 310.
- ⁴ CLAUDE and BROUSSEAU, *L'Encéph.*, 1922, xvii, 395.
- ⁵ DUPRÉ, E., and LE SAVOUREUX, *Rev. neurol.*, 1914, xxvii, 453.
- ⁶ FOIX CH., and CHAVANY, J. A., *Rev. neurol.*, 1926, xxxiii, 61.
- ⁷ FOIX, CH., and SCHULMANN, *Rev. neurol.*, 1914-15, xxvii, 779.
- ⁸ FREY, *Zeit. f. d. g. Neurol. u. Psychiat.*, 1914-15, xxvii, 397.
- ⁹ GIRAUD, G., and GUIBAL, *Rev. neurol.*, 1927, xxxiv, 723.
- ¹⁰ MAILLARD, *Thèse de Paris*, 1907; quoted by Pick (ref. 15).
- ¹¹ MARIE, P., and LÉVY, G., *Rev. neurol.*, 1922, xxix, 66.
- ¹² MARIE, P., and LÉVY, G., *Monde méd.*, 1925, xxxv, 329.
- ¹³ MARIE, P., and LÉVY, G., *Rev. neurol.*, 1925, xxxii, 260.
- ¹⁴ MEIGE, H., *Rev. neurol.*, 1913, xxvi, 108.
- ¹⁵ PICK, A., *Abhandl. a. d. neurol.*, 1921, xiii, 178.
- ¹⁶ PILCZ, *Wien. med. Woch.*, 1910, lxi, 626.
- ¹⁷ RORDORF, R., and CACCHIARARO, G., *Rev. neurol.*, 1926, xxxiii, 144.
- ¹⁸ SOUQUES, *Rev. neurol.*, 1908, xvi, 340.
- ¹⁹ STERLING, W., *Rev. neurol.*, 1924, xxxi, 205.
- ²⁰ STERLING, W., *Neurol. Polska*, 1926, ix, 29.
- ²¹ THURZO, and KATONA, T., *Gyogyaszat.*, 1927, lxvii, 54.
- ²² TRÉNEL, and CRINON, *Rev. neurol.*, 1912, xxiii, 676.
- ²³ TRÉNEL, *Rev. neurol.*, 1914, xxvii, 454.
- ²⁴ VIVALDO, J. C., *Rev. di criminol. psiquiat. y. med. leg.*, 1926, xiii, 280.
- ²⁵ VOLPI-GHIRARDENI, and TAROZZI, G., *Rev., neurol.*, 1925, xxxii, 506.

*I must express my indebtedness to the Medical Research Council under whose auspices this work has been carried out.

FIG. 1.

Photograph of right and left hemispheres from brain of Case 5.

The right half of the cerebrum is characterized by numerous lacunes situated mostly in the region of the basal ganglia. The middle section also illustrates the softening in the external capsule.

The left half shows the large softening which has burst into the ventricle. Note the internal hydrocephalus which has compressed the caudate nucleus and optic thalamus.