ARTERIOSCLEROTIC, SENILE AND PRESENILE PSYCHOSES.

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MEDICINE has lately become conscious of the special problems set by the increase of life expectancy following the hygienic progress of the last half century. General interest has been roused in the modification of diseases when they occur in the middle-aged and elderly, and in the illnesses typical of the later periods of life. From this movement psychiatry has profited in so far as many problems of "geriatrics" prove to be psychological and psychiatric problems. Our knowledge of facts has been confirmed and widened and the contact with general medicine should prove fruitful (Malamud, 1941).

It seems, however, doubtful if the psychiatric implications are adequately dealt with in the latest edition of the American standard work, Cowdry's *Problems of Ageing* (1942). Among many contributors there is only one psychiatrist, G. V. Hamilton, writing on changes in personality and on psychosexual phenomena. Of the two contributing psychologists Miles provides a full survey of the results of experimental psychology in the different stages of human life, and Lawton gives psychological guidance for older persons based on recent investigations. Psychiatry of old age is included in the chapter on "ageing of the nervous system" by Critchley, who only briefly touches on the medical and social problems of the subject.

1. MODIFICATIONS OF PSYCHIATRIC ILLNESS IN THE AGEING.

Psychoneuroses and "functional psychoses" observed in 110 patients over 45 were the material on which a study on psychopathology of the ageing by Diethelm and Rockwell (1943) was based. Their positive findings are well-known facts: Anxiety, depressive and paranoid reactions are the predominant types of illness; confusion, peevishness, resentment and apathy are frequent features in all these types; narrowness of interests, lack of plasticity, rigidity of the ageing personality and feelings of insecurity are the main underlying mechanisms. "Dissociative" schizophrenia was rare except as an exacerbation of an old schizophrenic illness. Hysterical symptoms took the form of dramatic emotional reactions coloured by anxiety. Manic states were not uncommon. Ageing frequently "unveils" constitutional personality traits. It is interesting that the authors find it necessary to point to the frequency of cerebral arteriosclerosis as a causative facfor even in a material from which organic cases were deliberately excluded!

Doty (1942), describing 25 patients with manic-depressive psychoses occurring for the first time in later life, found signs of arteriosclerosis in over half of his cases. As he excluded involutional melancholia from his group, he found anxiety an uncommon symptom.

A clinical and pathological survey of the types of chorea occurring in aged people by Pauly (1939) contains much detailed observation. Later cases of Huntington's chorea are not rare, but the most frequent cause is softening or haemorrhage due to cerebral arteriopathy localized in the basal ganglia and their connecting pathways.

Syphilis in old age was investigated by Hilliard and Kirman (1941) among the patients of a hospital for seniles, all over 70. One tenth of 700 admissions had a positive Wassermann reaction in the blood, and 12·5 per cent. of the positives had abnormal reactions in the C.S.F. Clinical symptoms of the latter, which were obviously due to cerebral syphilis, did not differ from those of the rest in which the presence of syphilis—as proved by positive blood Wassermann—was coincidental. This affirms the well-known difficulty of diagnosing G.P.I. in the elderly. The frequency of syphilitic infection in survivors of the generation born before 1870 was approximately the same in non-psychotics tested by Raybaud and Orsini (1939) in France (14 per cent. positive blood Wassermann).

The difficulty in diagnosing a brain tumour in the ageing and of differentiating its symptoms from vascular disease has been restated in two articles. The average age of 25 cases in which a clinically undiagnosed tumour was discovered at autopsy was 56 (Hastings, 1939). Typical symptoms, such as headache, vomiting, and choked disc were absent in more than two-thirds of the cases which were patients of a general hospital. Even in the Mayo Clinic the diagnosis had been missed in 40 out of 100 tumour cases confirmed by Moersch and his collaborators post mortem (1941). Only one-third of their patients had papilloedema; only 15 complained of headache. On the other hand, signs of mental change or deterioration of more or less severe type were a prominent complaint, usually reported by the patient's relatives.

2. Arteriosclerotic and Senile Psychoses.

Attention was directed to the circulatory factor in mental illness not only because of the greater frequency of arteriosclerotic psychoses with increasing longevity—the number of such cases in the New York State Hospitals was five times greater in 1936 than in 1912 (Clow, 1940)—but also because of the role psychological factors are supposed to play in causation and treatment of hypertension. Some of the recent work on this psychosomatic problem has a bearing on our topic. In a symposium on hypertension Katz and Leiter (1939) discussed the possible psychological influence of Western civilization on the incidence of hypertensive illness. Collaboration of psychiatry and general medicine is suggested in this field. The relation between blood pressure and emotional tension is the subject of two psychoanalytical case studies (Alexander, 1939; Saul, 1939). In cases of essential hypertension Saul established a condition of "strong, chronic hostility," inhibited, but near to consciousness. Similar views are put forward in an article by Riemer (1941). A series of 193 depressive and paranoid patients could be divided into three groups according to the strength of "inhibited aggressiveness" they displayed (Miller, 1939). When their blood pressure was tested its height was significantly correlated with the degree of hostile emotion.

However important the psychological approach in general medicine may be, there is much doubt if progress can be achieved by such pseudo-quantitative methods applied to unconscious or semi-conscious emotions. Winding up the symposium, Weiss (1939) surveyed the various therapeutic approaches to hypertension, and pointed out the pitfalls as well as the achievements of the psychological method.

S. K. Robinson (1940) made a plea for medical and psychological treatment of hypertension, based on 93 cases, of which 70 responded well to conservative therapy; his paper is also an attack against operations like splanchnic resection, adrenalectomy, etc., applied to relieve hypertension. Good results after surgical procedure are probably explained by indirect mechanisms, such as shock, bed rest, or psychogenic factors, according to Robinson.

A comprehensive study of the brain in malignant hypertension (Rosenberg, 1940) disclosed severely destructive cerebral lesions in 12 out of 17 cases examined post mortem. Cerebral oedema, multiple miliary haemorrhages and infarcts as well as larger destructive lesions were found, obviously the result of profound changes in the arterioles of the brain. In the past many of the clinical phenomena in these patients were attributed to spasms of cerebral vessels. "These spasms may . . . occur and perhaps are responsible. . . . However . . . such an assumption may lead the clinician to an unjustified sense of security concerning the brain. A history of cerebral symptoms was found to be associated constantly with more or less widespread cerebral destruction." The problem of hypertension—angiospasm—haemorrhage is discussed in all its theoretical aspects in a paper by Scheinker (1940).

One hundred arteriosclerotic psychoses diagnosed clinically have been collected by Clow. Their medium age was 64, the ratio of males to females 1·2 to 1, which corresponds to the average sex incidence found by former workers. There was a marked family background of cardiovascular and senile disease, but manic-depressive heredity and depressive attacks in the patients themselves were also frequent. Alcohol and tobacco seemed without aetiological importance, but excessive taking of sedatives was admitted or suspected in many patients. Precipitating emotional causes were elicited in three-fourths of the patients. Hypertension was found in 79, retinal arteriosclerosis in 76 cases. The most frequent presenting symptom was delirium. More than half of the patients had vascular accidents and signs of focal cerebral lesions. These cases were treated according to the principles of general medicine, with emphasis on psychological re-adjustment through routine exercises and occupational therapy. Almost half of them returned home.

The idea that delirious states in old age are of toxic nature and often due to barbiturates or bromides given for insomnia has been taken up with great zest by G. W. Robinson (1939, 1941, 1942). From favourable results he obtained, using daily infusions of 10 per cent. glucose, he draws far-reaching conclusions about the pathology of the condition based on relatively little factual evidence.

Rothschild's neuropathological study of arteriosclerotic psychoses (1942) dealt with 28 cases in which the clinical diagnosis was confirmed post

mortem and the brain fully examined. Although various focal lesions were found in all cases, there were many discrepancies between the extent of damage and the degree of intellectual deterioration. Structural damage, for instance, in patients with severe intellectual impairment was sometimes less pronounced than that observed in better preserved patients. As differences in localization of the cerebral lesions could, according to Rothschild, not account for these divergencies, he proposed an explanation which he had used before for a similar problem in senile dementia (1937). Taking the cardiac weight as a measurement of cardiovascular equipment and resistance, he found that it differed considerably among the patients. It might be expected that those with stronger resistance were able to compensate for even severe cerebral lesions, while those less well equipped showed severe symptoms, even after small damage. His figures bore out this theory; but it seems difficult to follow him further when he eventually linked up cardiovascular with psychological resistance of the personality, thus paying his tribute to the "psychobiological" trend in present-day American psychiatry.

Greenfield (1938) proposed a relatively simple classification of the pathological findings in cerebral vascular disease intended to replace the numerous variants distinguished by former workers: (1) All cases with softenings and haemorrhages, the most common form, localized cortically or subcortically, often with arteriosclerotic changes of the basal arteries. (2) Verrucose or granular atrophy of the cortex, usually associated with a similar change in the cortex of the kidneys and hyperpiesis. Both cells and fibres of the cortex are degenerated and replaced by neuroglial overgrowth. (3) Widely scattered areas of degeneration or rarefaction of nerve cells with little glial reaction. These so-called "areas of paling" are invisible to the naked eye, brain substance and vessels appearing normal until examined microscopically. This type is according to Greenfield closely allied to findings in mental deterioration associated with epilepsy.

A statistical survey of psychotic manifestations in 1,000 cases of acute cerebral vascular lesions confirmed at autopsy was published by Irish (1940, 1941). Only 17 per cent. of these patients afflicted by embolism, thrombosis or haemorrhage of the brain showed marked psychotic symptoms, including signs of mental deterioration. This figure can only be regarded as a minimum, since psychiatric symptoms are so frequently passed by in general hospitals, especially in elderly patients.

Our knowledge of the pathology of senile psychoses and dementia has made little progress in the last five years. E. D. Cameron, who put forward a research programme in this field at a round table discussion in 1941, has attacked it from many aspects, together with his collaborators. He confirmed the low ascorbic acid level in blood and spinal fluid of seniles established by Plaut and Bülow, and found that it could be raised by addition of sufficient vitamin C to the diet (Remp, Rosen, Ziegler and Cameron, 1940). The controversial nature of these findings is discussed by Mueller-Deham (in Cowdry, 1942). Cerebral oxygen consumption was found diminished in senile psychoses (Cameron, Himwich, Rosen and Fazekas, 1940). In both papers one misses control experiments in non-psychotic seniles. Greatly increased tendencies to

perseverate during the intake of memorized material and to elaborate it afterwards were found in psychological experiments; the poverty of recent memory in senile patients could be explained from these anomalies (Cameron, 1940).

Nocturnal delirium of seniles was reproduced when the patients were placed in a dark room during daytime. Thirteen out of 16 patients showed a severe distortion of their spatial image within an hour after they had been blindfolded. Cameron (1941) suggested that the delirium was based upon an inability to maintain a spatial image without repeated visualization.

Horgrefe (1939) tried to differentiate the various shapes and forms of senile plaques in the brains of five patients, and to relate the histopathological picture to the stage of development and to the clinical course of the mental deterioration in each case. Such attempts to differentiate certain types within the large and varied group of senile dementias are very much in line with the ideas of Kraepelin, who, for instance, separated clinically the hyperactive, lively and socially well-preserved cases with marked retention defect from the rest under the term "presbyophrenia." Bostroem explained the hyperactivity and sociability of the prebyophrenics as due to a previous hyperthymic personality. Against this view Sabass (1939) showed that it is impossible to predict the type of senile psychosis from the premorbid personality.

Tompkins (1939) collected data on the frequency of convulsions and other epileptiform signs in senile and arteriosclerotic patients. He found epileptic fits in 7, episodic attacks which might be epileptoid in 6, and lasting changes of consciousness in 3 out of 100 cases.

Cameron (1941) has emphasized the part played by loneliness and lack of social contact in the causation of breakdown in the elderly; he opened a series of social clubs in connection with a special clinic to overcome their social isolation. The idea of lack of social integration as a factor in senile psychoses is supported by a study by Williams and his co-workers (1942) on the significance of what they called extrinsic factors in seniles and arteriosclerotics. They went into the histories of 47 carefully diagnosed cases of each group and contrasted the social settings prior to the onset of illness. 79 per cent. of the arteriosclerotics lived with their wives or children in favourable social conditions against only 15 per cent. of the seniles. Only 21 per cent. of the latter lived under financial circumstances which could be regarded as satisfactory, while 68 per cent. of the arteriosclerotics had had financial security. The authors appeal for "well conducted, socially acceptable institutions for the aged" as a preventive measure against senile psychoses.

That it is still worth while trying to distinguish senile from arteriosclerotic psychoses clinically was proved by Rothschild (1941) in a critical paper on differential diagnosis. He compared the symptoms and the post-mortem findings in 60 patients, half of which were clinically diagnosed seniles and the other half arteriosclerotics. Although anatomically pure forms of each were less frequent than mixtures of the two processes, one or the other predominated in most cases; and clinical pictures of a mixed nature were not nearly as frequent as might have been anticipated from the pathological findings. The criteria generally used in clinical psychiatry were confirmed. Senile psychoses occur later in life, are gradually progressive and last longer; paranoid pictures

are more common in seniles. Arteriosclerotic psychoses have usually a sudden onset, show less intellectual impairment, more fluctuation of symptoms, transient neurological signs, etc. Rothschild also warned against labelling senile-arteriosclerotic any type of psychosis when it affects an elderly person, and gave striking illustrations of cases in which the diagnosis was disproved at autopsy.

3. Presenile Dementias.

This subject, which has attracted more workers than any other topic of the present chapter, has been admirably surveyed by McMenemey (1941). His article covers not only the "essential" dementias like Alzheimer's and Pick's diseases, but also deals with Huntington's chorea, Jakob's disease and the other rarer types in which neurological symptoms are predominant. The differential diagnosis of dementias of middle age due to tumour, trauma, syphilis, arteriosclerosis, are fully discussed, as well as Gower's concept of abiotrophy, frequently used as an explanation of senile and presenile disease and as a "cloak for ignorance." The concept is meant to signify an inborn constitutional tissue defect manifesting itself at a certain age of the individual.

Spatz (1938) has taken up this idea in a somewhat modified form. He speaks of premature ageing of certain cerebrospinal systems as the hypothetical cause of a number of diseases which he proposed to combine under the term "systemic atrophies." In such a group Pick's disease would be classed with Huntington's chorea, Jakob's disease, and with ponto-olivary, cerebellar, and spinal "degenerations" like spastic spinal paralysis and amyotrophic lateral sclerosis; Leber's hereditary optic atrophy was also considered as a probable member of the group. This fusion of hitherto unconnected disease entities is mainly based on pathological similarities; it would make it easier to account for the relatively frequent combination of symptoms from the different diseases in the same patient and for co-occurrence of different forms in the same families.

A. *Alzheimer's Disease.*

This is not included in the "systemic atrophies" by Spatz because its pathology is not atrophy of nervous elements alone, but also includes characteristic tissue reactions with formation of senile plaques and fibrillary changes. Also it is not confined to one neuronic system, and finally its hereditary nature is still under debate. However, combinations of Alzheimer's with other forms of the presenile group are not infrequent. Further instances of such mixtures have been published, e.g. Liebers, 1939—with Pick's disease; Worster-Drought and others, 1940, van Bogaert and others, 1940—with spastic paraplegia; Hemphill and Stengel, 1941—with cerebellar atrophy. While all these atypical cases show familial incidence, new familial cases of pure Alzheimer's disease have also been reported (Gruenthal and Wenger, 1939/40). On the other hand, nothing about heredity could be established in six patients by McMenemey (1940); in only one out of a series of seven cases collected by English (1942) was there heredity of the same type; and in nine patients recently communicated by Stengel (1943) the hereditary factor was found

insignificant. One has however to remember the difficulties in assessing hereditary trends in elderly people who have often been lonely inmates of homes and hospitals for many years. A completely unexplained feature of Alzheimer's disease is the prevalence of females. All patients of McMenemey's and English's series were women, as also seven out of the nine cases in Stengel's material.

The cerebral changes in Alzheimer's disease are so characteristic that it was suggested (McMenemey, 1940) that the diagnosis be based on pathological findings only, thus including rare cases starting in adult life and without the typical clinical symptoms. Unfortunately the characteristic changes—plaques and neurofibrillary alterations—are also present in ordinary senile dementia, the only distinctive sign being a greater degree of severity in the findings in Alzheimer's disease. Surveying the literature on early cases, Ferraro and Jervis contributed two observations with autopsies of patients whose illness started at 33 and 38 respectively. Their clinical symptoms and course were those typical of Alzheimer's disease. From the newly published series of cases mentioned before, these symptoms seem well established. The most prominent are, in the approximate order of their appearance, impairment of memory, prolonged periods of restlessness, anxiety and depression, speech disorders, syncopal and epileptic attacks, muscular hypertonicity, and focal signs like apraxia, agnosia, cortical blindness, facial paresis, etc. (Rothschild, 1934; English, 1940). The significance of restlessness and over-activity and of the emotional features was fully discussed by Stengel (1943). The diffuse focal symptoms and the muscular rigidity have attracted less attention among recent workers. All this refers to cases in which the illness was already in a rather advanced stage because only then are they usually seen by a psychiatrist. Prodromal depressive or paranoid symptoms may disguise the organic nature of the disease; rapid deterioration occurs in typical cases, but arrests and remissions and a prolonged course over twenty years have been observed.

Differential diagnosis from senile dementia can be very difficult, especially if the patient approaches 60 at his first visit to a doctor. Absence of fabrication, greater intellectual deterioration and the presence of focal symptoms have a certain distinctive value. There are more signs of general senility in the senile, while in Alzheimer's disease only the central nervous system shows deterioration (English, 1940). Air encephalography shows dilatation of ventricles and general cortical atrophy, but only in relatively advanced cases. Electroencephalography was employed by Longridge (1939) and Stengel (1943); the former found a well-marked delta focus in one fronto-temporal region, while the latter's patient had "small irregular potential changes with no sign of any rhythmic activity whatever." Further observations are wanted before any conclusions are possible.

Differentiation from Pick's disease will be discussed later.

All workers in this field agree that Alzheimer's disease is not rare, and that many cases pass undiagnosed in mental homes for the elderly and in other institutions. It is certainly much more common than Pick's form of presenile dementia.

B. *Pick's Disease.*

Workers in Britain and America have only lately paid more attention to this form of presenile dementia first described by Arnold Pick in 1892. By way of contrast there seems at present a tendency to over-estimate its frequency. The fact that according to Ferraro and Jervis (1940) some 50 publications had appeared in the continental literature within six years is somewhat misleading, because the great majority of these publications deal with one or two cases only.

As the name appears unfortunate because of possible confusion with Pick's polyserositis or Niemann-Pick disease, "idiopathic circumscribed presenile cerebral atrophy" has been suggested as a comprehensive and accurate term (Nichols and Weigner, 1938).

In spite of many instances in which no positive family history is reported, there is good reason to regard the hereditary factor in Pick's disease as important, perhaps even as satisfactorily established (Benedek and Lehoczky, 1939). Many recent case-studies deal with families in which an identical heredity is found, or more often, because of lacking autoptic evidence in relatives, suspected. Friedrich (1940) has proved the presence of Pick's disease in mother and son by post-mortem examination of the brains of both. Lowenberg, who had reported a number of typical cases before (1936), distinguished, in a recent article with Boyd and Salon (1939), two familial trends operating in Pick's disease, viz. occurrence of the same illness in near relatives, and the tendency of Pick's disease to appear in families with other degenerative changes of the central nervous system, e.g. senile psychoses, paralysis agitans, and mental deficiency. Related views of Spatz have been already mentioned (p. 321).

Pick's disease has a predilection for females similar to that of Alzheimer's. If Rothschild's (1938) assertion that it is twice as frequent in women as in men proves correct, this would be a theoretically and practically important distinction from cerebral arteriosclerosis, which has the opposite ratio of sex incidence. As in Alzheimer's disease the age incidence has extended more and more, the youngest case confirmed by autopsy being only 21 (Lowenberg, Boyd and Salon, 1939). Cases up to 80 have been reported. Cases of Alzheimer's disease reported in patients over 60 are somewhat doubtful because of the identity of its pathology with senile dementia.

Pick's original interpretation of the pathology as precocious ageing of association centres is still valid in spite of many attempts to supplement or replace it. The atrophy is systemic in so far as the gross macroscopically visible changes involve preferably the frontal lobes, but temporal, parieto-temporal and occipital forms have been observed; it is symmetrical, but often the left hemisphere is more affected. On histological examination atrophic areas, with corresponding glial reaction, are found all over the cortex, and in some cases in the basal ganglia and substantia nigra. Davison (1938) stressed the localization of atrophy around blood vessels, but Ferraro and Jervis (1940) who had favoured this idea before, could not confirm it in their recent case. In fact, as McMenemey (1941) points out, the cerebral vessels are singularly free from histological evidence of disease. The white matter of the hemispheres is always reduced—a fact much emphasized by Freeman and recently by Stengel (1943).

The clinical picture is less uniform than that of Alzheimer's disease, as one would expect from the differing localization of the main atrophic region in different patients. Loss of judgment, reduction of associative power, emotional abnormalities like facile hilarity, flattening of affect and other personality changes precede the disturbance of memory, which in many cases was found surprisingly intact (Bouton, 1940; Malamud and Boyd, 1940). Disturbance of the waking-sleeping rhythm has been observed as an initial symptom. Disturbances of speech are prominent when the illness progresses, e.g. echolalia (Nichols and Weigner, 1938) and all types of aphasia, especially a complete sensory aphasia. In later stages there is loss of spontaneity, increasing apathy, and severe mental decline; the patient usually succumbs to some intercurrent infection. The duration varies between 2 and 12 years.

Diagnosis is often very difficult. "There is hardly any other syndrome in the field of neurology and psychopathology that represents such a testing ground for diagnostic acumen and is so instructive as that of the presenile dementias" (Bouton). While this worker published four cases only diagnosed post mortem, Urechia (1940/41) reported his case because it was diagnosed clinically. Ferraro and Jervis (1940), on the other hand, insist that half of the 50 patients from the European literature had been correctly diagnosed in lifetime.

As to differential diagnosis from Alzheimer s disease, Critchley (1938) remained sceptical about the possibility of distinguishing the two forms on purely clinical grounds; but on the same occasion he surveyed the fundamental ideas of Goldstein's method of examination, from which many workers expect much help in the analysis of the psychological disturbances in Pick's disease. Because of the small number of patients observed by one author, unfortunately no one has been able to compare Goldstein's method in a group of early cases belonging to Alzheimer's and Pick's forms. Convulsions and muscular rigidity are rare in Pick's disease; the focal symptoms are more diffuse in Alzheimer's, more definite in Pick's form; initial restlessness and depression are more frequent in Alzheimer cases. A resemblance between Pick's disease and general paralysis has been noticed as against a similarity of Alzheimer's to senile dementia (Nichols and Weigner, 1938). Early impairment of insight in Pick's disease forms a useful diagnostic sign of distinction from cerebral arteriosclerosis which, in contrast to the steady progressing decline in Pick's disease, usually shows considerable fluctuations. Diagnosis by air-encephalography is only possible in cases of severe localized atrophy, i.e. in a rather advanced stage of the illness. According to Romano and Miller (1940) the characteristic pneumo-encephalographic data are: "Bilateral dilatation of the lateral ventricles, increase of intragyral air spaces more marked in the frontal and temporal areas, and accumulations of air over these areas in the nature of confluent masses."

c. *Other Forms of Presenile Dementia.*

Jakob-Creutzfeld disease.

A few more instances of this type of presenile dementia, which is characterized by additional neurological signs, have been reported within the last five years, almost all of them with a slightly different clinical picture and correspond-

ing different pathology. A. Meyer (1938) demonstrated two cases at a meeting of the Royal Society of Medicine, one of which showed a combination with amyotrophic lateral sclerosis. A patient in which the neurological symptoms were of the paralysis agitans type was subsumed under this group by Brown and Buckle (1939). Davison and Rabiner (1940) used Jakob's original name, "spastic pseudo-sclerosis," for their cases, because spastic paresis with pyramidal and extra-pyramidal features was a prominent symptom, while the mental changes were slight or appeared only in the later stages of the illness. After a thorough discussion of all possible classifications, Jervis and his collaborators (1942) found the clinical and pathological features of their patient well in keeping with Jakob's original description. Conversely, McMenemey and Pollock (1941), refrain from classifying their case, but emphasize its resemblance to the Jakob-Creutzfeld syndrome and to a modification of it first described by Hallervorden.

Kinneir Wilson (1940) criticized the term "spastic pseudo-sclerosis" as inaccurate and misleading. Cortico-pallido-spinal degeneration and similar hyphenated names have been suggested in which the terms can be exchanged according to the parts of the C.N.S. involved. Many workers favour retaining the names of the first observers on the grounds that it is still doubtful if the condition represents a nosological entity. In fact, it serves at present as a convenient "dumping ground" (McMenemey, 1941) for all instances of atypical presenile dementia combined with various neurological disturbances, especially of the motor system. The illness runs a rapid course, lasting 1–2 years. Its histology is a parenchymatous atrophy of nervous tissue in the affected regions with a more or less marked glial hyperplasia.

Relations to the so-called primary cerebellar atrophies are highly probable, especially in cases like those recently published by Akelaitis (1938), Richter (1940), Weber and Greenfield (1942), in which the cortex was involved and the patients showed mental deterioration in the presenile age.

Familial incidence is only reported in a few patients of the Jakob-Creutzfeld type. Several workers have discussed the similarity of the symptoms and pathology with those of pellagra. Vascular and infectious origins have also been debated.

Stern's (1939) patient with a bilateral symmetrical degeneration of the thalamus may be mentioned in this connection because he also showed a rapidly dementing course during the fourth decade. In other respects the observation differed from all known types of presenile illness.

Kraepelin's Disease.

Gruenthal, whose important work in the field of presenile dementia is generally recognized, suggested this name for atypical depressive psychoses with catatonic features starting after the age of 40 and followed by severe organic deterioration. The organic nature of these late catatonias had been pointed out by Kraepelin. Best (1940/41) contributed another example of this group examined in Gruenthal's laboratory. Histologically a severe affection of nerve cells in cortex, basal ganglia and cerebellum was found in the patient, who died from an accident.

In conclusion it seems appropriate to remember that the " essential " presenile dementias form only a small percentage of the psychoses with intellectual deterioration of this age, and to quote, in a slightly modified form, the " working classification based on a clinico-aetiological foundation " of these psychoses as given by Critchley (1938) :

Syphilitic dementias (including " syphilitic encephalitis," luetic cerebrovascular disease, G.P.I., etc.).

Arteriosclerotic dementias.

Traumatic dementias (including sequelae of cerebral laceration, subdural haematoma, etc.).

Cerebral tumour and abscess.

Epileptic dementias.

Dementias following chronic psychotic states like schizophrenia.

Dementias complicating chronic neurological disorders (disseminated sclerosis, epidemic encephalitis, subacute combined degeneration, Huntington's chorea.)

Dementias after chronic, toxi-infective states (uraemia, alcohol, lead, hashish, barbiturates, pellagra).

Essential dementias of unknown origin: Pick's, Alzheimer's, Jakob-Creutzfeld's disease, etc.

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