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developed', and for the cryptic record that 'the hitherto separate "Entertainment Fund" Bank Account operated in the name of the Medical Superintendent is closed', an obviously retrograde step which naturally I deplore.

In 1884 Dr. Rutherford began an experiment whereby chronic female patients lived in a house without attendants, requisitioning their own supplies, and doing their own cooking, cleaning and mending; but in 1888 one of these patients died—not surprisingly—and the voice of authority forced the abandonment of this progressive measure. Very recently we had the same experience, when two 'hostel' wards ran without night nursing for some years, until departmental reaction to a fire in distant England forced us to re-staff them.

The Crichton Memorial Church dominates the centre of our hospital. Its cost was estimated in 1890 at £5,000, which rose to £12,500, and then £15,000, by the time it was begun in 1892. We were faced with strikes for more money, combined with complaints from the town that our high wages were drawing off settled labour; and the building was ultimately completed in 1897, years behind schedule, and at a final cost of £30,000. Those who have been concerned with hospital planning will find this story only too familiar.

The nearly final word is to comment on the hospital's teaching and scientific record. In 1909 the Directors established three Fellowships in Clinical Neurology and Psychiatry, in Pathology and Chemistry, and in Pathology and Bacteriology. These posts were, however, irregularly filled and it was Dr. P. K. McCowan—Easterbrook's successor—who expanded our research facilities. Dr. McCowan was—and is, I am glad to say—a man of many capacities, who revolutionized the treatment of our patients, by vigorously encouraging the early use of the new therapies which began to appear just before the Second World War. He was also, and this is not a common virtue, a man sufficiently secure to bring to his hospital colleagues of capacity and renown. In the period of his governance were established Departments of Clinical and Psychological Research, under Mayer-Gross and Raven; and these distinguished men helped to begin a new era in the work of the hospital.

In 1948, we entered the National Health Service. By retaining our private and amenity beds, we have kept something of our wider function. The rest is a matter of current affairs rather than history.

Dr. Tait's paper was followed by that of Dr. Robinson who discussed:

THE EVOLUTION OF GERIATRIC PSYCHIATRY

The recognition that insanity was due not to supernatural influences, but to disease of the brain was the major psychiatric discovery of the Greeks. That the contribution of psychodynamics to the problems of old age was also appreciated is apparent from Plato's famous dialogue between Cephalus and Socrates. 'The truth is . . . that these regrets, and also the complaints . . . are to be attributed to the same cause, which is not old age, but men's characters and tempers; for he who is of a calm and happy nature will hardly feel the pressure of age, but to him who is of an opposite disposition,

youth and age are equally a burden'. But this ancient medical wisdom did not survive the Dark Ages; demonology again replaced psychopathology.

Only one hundred years ago Daniel Maclachlan could write in his textbook on: *The Diseases and Infirmities of Advanced Life* that there was little in the English literature which contained any useful information on the hygiene and diseases of old age.

For it is a remarkable fact that the major organic psychoses were delineated clinically and histologically in the space of a dozen years only at the turn of the last century. Three factors were responsible for this sudden crystalization: the recognition of the cause of General Paralysis of the Insane; Kraepelin's separation out of Dementia Praecox and Manic-Depressive psychosis from the mess of symptomatic description which had previously served; and the rapid technological advances in neuropathological techniques.

To Esquirol (1845) is attributed the first careful clinical discussion of the mental changes associated with old age. It is clear too, that he was aware of the occurrence of functional syndromes in old age. After describing some of the modes of onset of senile dementia, including restless agitation, he says: 'We shall not confound this excitement with the mania which bursts forth at a very advanced age, in strong, robust and well sustained persons. Mania, attended even with fury, bursts forth after the age of 80 and is sometimes cured'.

In a famous passage Esquirol makes the distinction between dementia and amentia. 'A man in a state of dementia is deprived of advantages which he formerly enjoyed. He was a rich man, who has become poor. The idiot, on the contrary, has always been in a state of want and misery. The condition of a man in a state of dementia may change; that of the idiot is ever the same . . . the man who is in a state of dementia indicates in his organization, and even in his intelligence, something of his past perfection; while the idiot is what he always was; and is all that he can be'.

Though the problems of dementia and organic cerebral disease occupied a great deal of psychiatric attention during the nineteenth century they were not easily solved. On the one hand the subject was complicated by the classification of major psychiatric syndromes into either states of excitement or states of enfeeblement—many of the latter synonymous with 'dementia' (schizophrenia in today's terminology); and on the other by the presence of large numbers of general paralytics.

To Thomas Willis in 1672 is given the distinction of first recording the symptoms of general paralysis. But it was the end of the eighteenth century before John Haslam (1798) gave a clinical description in recognizably modern terms: 'The paralytic affections are a more frequent cause of insanity than is believed, and are also a very common sequela of mania . . . in the majority of patients, memory is materially weakened. These patients, as a rule, fail to recognize their condition. So weak that they can scarcely keep on their legs, they still maintain that they are extremely strong and capable of the greatest deeds'.

Bayle, another early writer also emphasized what he called 'the peculiarly ambitious' variety of mental illness by which the patients were dominated.

The Salpêtrière was the scene of many clinical and pathological studies by Esquirol and his pupils during the first half of the nineteenth century. As a result, it was slowly

established that the symptoms of general paresis were caused by organic brain disease. Calmeil (1826) first named the disease 'General Paralysis of the Insane' and finally towards the middle of the century the syndrome began to be recognized as a clinical entity. About this time Esmarch and Jessen suggested a syphilitic basis; but it was to be another fifty years before this hotly debated etiology gained general acceptance.

A quotation from Clouston, a recognized authority on the subject, as late as 1898, gives some indication of the psychiatric puzzlement of the times. 'I look on it [G.P.I.] as being equivalent to a premature and sudden senile condition, senility being the slow physiological process of ending, general paralysis the quick pathological one. . . . There are two causes that singly or combined, above all others, cause the disease, viz. sexual excess, especially if indulged in at or after middle life, and alcoholic intemperance, especially if bad or impure drinks are used. If hard work, muscular or mental, with a stimulating diet of flesh are combined with these, then we have an additional liability'. He accepted Wilson's (1892) description of a paretic diathesis. 'General intelligence, ambition and energy, sociability and a large capacity for enjoyment, a firm belief in one's self and a preference for handsome women are the good and sane characteristics of this diathesis, while a lack of the higher control, tendencies to excess, selfishness, vanity and restlessness are its weak points'.

But the essential gross pathological changes were already accepted and medical science was gathering momentum. After Quincke obtained cerebro-spinal fluid by lumbar puncture in 1890, laboratory tests began to be useful in diagnosis. Wassermann's complement fixation (1906) and Lange's colloidal gold reaction (1912) set the stage for the final proof by Noguchi and Moore (1913) that the *treponema pallidum* was responsible for the disease.

Though psychiatric pictures had been gradually rounded out during the century there was little resemblance to the classification we know today. Clouston's *Textbook of Psychiatry* (fifth ed., 1898), serves as an indication of standard psychiatric thinking towards the end of the century. Like other authors of the time, he tended to classify the insanities in terms of epochs, situations or symptoms. There were thus (among many others) adolescent, climacteric and senile insanities; anaemic, diabetic and post-conubial insanities. There was a strong tendency for any of these to end in 'dementia', and when he speaks of dementia it is usually this secondary type to which he refers.

He appreciated the role of cerebral arteriosclerosis in the causation of dementia but only distinguished such cases from senile dementia in the presence of hemiplegia, which he then termed 'paralytic insanity'.

It was clear that he was not satisfied with this arrangement for he says: 'The classification of the future will be won on a pathological basis, but we are far from that yet and any premature attempt to construct such a classification, not founded on a sound brain physiology and psychology must do more harm than good'.

In his Presidential address to the Royal Medico-Psychological Association in 1888, after reviewing somewhat disconsolately current psychiatric classifications and nomenclature, he indicated the method which was later to be used to such revolutionary effect by Kraepelin.

Are we in a position to say which is the typical form of mental disease? Can we select out of all the varieties and sequences of mental symptoms . . . that which, built up into a clinical whole,

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would form an ideal case? I am not sure but that it is as yet an impossible task. How can one get a common character out of emotional elevation in one case and emotional depression in another? Out of stupor on the one hand and mania or impulsive insanity on the other. . . . How can we get a common type out of mild, curable melancholia on the one hand and general paralysis or organic dementia on the other? . . . Mental diseases are most various in symptoms and course, and the tendency has been strongly, the more closely they are studied, to multiply varieties and name each of them. This, is no doubt, a necessary stage in all scientific studies. The question is—have we arrived at the stage in our special study at which we can be synthetic? Can we combine varieties and species into true classes?

It seems certain that if we confine our attention to the purely psychological view of our cases, we shall fail to arrive at any typical form. But if we take the *whole clinical history and course of a large number of cases*, and especially if we look at their *terminations* we shall, I think, get within sight of a true type of mental disease.

The contribution of Britain to the development of nineteenth-century psychiatry was not so much scientific as in the pursuit of the humanitarian concept of treating mental illness. Non-restraint, moral treatment and the organization of efficient hospitals and policies, essentially British concepts, were the background against which the French and Germans were able to develop their diagnostic skills.

It was Kraepelin (1904) who finally, on the basis of continuing and systematic observations on a vast number of patients, applied the brilliant principle which transformed the nosologic chaos of the times. By his painstaking work, he was able to show that the apparently diverse entities of the four-stage insanity of Zeller, the catatonia of Kahlbaum, the hebephrenia of Hecker, the folie circulaire of Falret and the multitude of enfeeblements and excitements, could be resolved into the twin abstractions of Dementia Praecox and Manic-Depressive psychosis. Only with the acceptance of these concepts was it possible to construct a rational framework within which the other syndromes rapidly each began to find its place. But that even such a profound evolutionary step was not without its inherent defect has been pointed out by Zilboorg (1941). 'Essential to his discovery was the "prognostic attitude". One diagnosed by prognosis as it were, and if the prognosis proved ultimately correct, the diagnosis was considered correct. . . . This was a departure from a vital and sound principle of general medicine. One cannot say that because a disease ends in a certain definite way, that it is a certain definite disease. And in this flaw in his otherwise monumental discovery lay the seeds of near-disaster, as we know to our cost today. For therapeutic efforts were to become based, as a result, on the complacent, expectant attitude that if the disease was a manic-depressive psychosis the patient would get well, at least from the attack, and if it was a dementia praecox the patient would deteriorate'.

Kraepelin's work covered the whole field of psychiatry, but until general paresis and the two major functional psychoses were isolated, little progress could be made in differentiating the other organic psychoses. Within a few years he and his school at Munich had established all the major organic syndromes. This became possible because of the rapid blossoming of neuropathology in the closing years of the century.

Grmek (1958) attributes the first use of the term 'arteriosclerosis' to Lobstein in his *Textbook on Pathological Anatomy* published in 1833; though the condition was recognized even by ancient medical writers, by whom it was regarded as one of the essential degenerative processes of ageing. Leonardo da Vinci showed in some of his

drawings that he appreciated the significance of the hardened tortuous vessels of the elderly. In 1677 Francis Bayle described calcification of the cerebral arteries and pointed to its association with apoplexy. The first good clinical descriptions of arteriosclerotic syndromes date from the second half of the nineteenth century. As a result, many neuropathologists turned to the investigation of cerebral arteriosclerosis as the possible cause of insanity in later life.

Neuropathological skills were, of course, dependent on existing laboratory techniques. The development of the science during the nineteenth century has been divided, on the basis of the staining methods available, into three distinct phases. During the earliest period, from the invention of the microscope by Jansen in 1609, until 1854, staining was not employed and progress was slow. In spite of the primitive methods in use—teasing, maceration and, much later, hardening and sectioning—some notable achievements were recorded. Ehrenberg described nerve cells and fibres in 1833; in 1838 Schwann extended the ‘cell theory’—already accepted by botanists—to the animal kingdom. The work of Virchow and his school, culminating in the publication of his *Cellular Pathology* (1858) brought this era to a close.

The carmine period, 1854–1884, marked the introduction of staining techniques and saw a much more rapid advance. As carmine was readily taken up by myelinated fibres, the emphasis during this phase was on the identification of the main fibre tracts. Charcot and his school particularly, were absorbed by the correlation of lesions of the tracts and nuclei, with clinical signs observed during life. In this way a functional neuro-anatomy was gradually built up.

The detailed study of the cortex had to await the more complex techniques by Exner, Weigert, Nissl, and finally in 1903 Cajal and Bielchowsky. In this ‘elective’ period, it was for the first time possible to select and examine, by the use of the appropriate stain, specific elements of nervous tissue. A complete histological picture was at last available.

In the last decade of the century, Pick, Binswanger and Alzheimer described and differentiated the appearances of lobar atrophy and cerebral arteriosclerosis from general paralysis on the one hand and senile dementia on the other. In another few years Alzheimer, Perusini, Fischer and Barrett had delineated the classical histological components of senile dementia—senile plaques and neuro-fibrillary change—and had established the presenile form of the disease which bears Alzheimer’s name, as a separate entity.

But latterly the pace of advance had been too swift for many. These concepts were not readily accepted everywhere. There were those who could echo Osler’s (1904) words: ‘Within the lifetime of some of us, science—physical, chemical and biological—has changed the aspect of the world, changed it more effectively and more permanently than all the efforts of man in all preceding generations. Living in it we cannot fully appreciate the transformation and we are too close to the events to realize their tremendous significance’.

But by 1906, Farrar, speaking at the British Medical Association Meeting in Toronto, was able to summarize magnificently the century’s psychiatric progress and to indicate the way ahead. Of necessity, the symptomatic method had come first; in its most primitive form it recognized but few distinct diseases of the mind. These were

assumed to be determined by the most conspicuous symptom in the disease picture and naming the symptom constituted the diagnosis. Etiology, course and outcome of the psychosis were of secondary importance, if they were considered at all. This symptomatic method was, nevertheless the birth of the scientific approach in psychiatry. The growth of the clinical method was the best thing which nineteenth-century psychiatry had to offer. It had been recognized that the external appearances of, for example, mental depression or exaltation or of psychic enfeeblement such as had stood for the earlier symptomatic entities were not sufficient for the identification of individual diseases, and that moreover, these states might occur in the most varied conditions, distinguishable from one another in etiology, course and outcome. Clinicians began to take a biographic perspective of mental illness. As a result so-called typical disease forms began to emerge.

When it was found by observers all over the world that large numbers of patients fitted more or less into these categories, the hope was not unnatural that as knowledge accumulated and differentiation became still more accurate, new and independent forms would gradually be discovered, as a result of which the great bulk of undiagnosed material would finally be used up, each case being eventually assigned to its proper pigeon-hole, each with its label attached. Such sanguine expectations were not entirely to be realized.

But to classify is not in itself the aim and end of psychiatry. The excursion of the pendulum has covered 50 years, and there are signs that the point of the return swing is near. Already clinicians are taking a broader view of mental pathology than that which attempts to discover in every patient some definite disease form.

This broader view may be described as the biologic method. It comprehends the advances that have been made by both the symptomatologic and clinical methods, but its standpoint is less dogmatic. It looks at diseases of the mind not so much as stationary or transitory symptom complexes, nor indeed alone as protracted manifestations of individual pathologic processes, it looks rather at the diseased personality in its entirety. . . . It is neither a cross section (the symptomatic method) or a longitudinal section (the clinical method) but embraces both and takes the cubic measurements into the bargain.

The biologic method studies personality first and disease second; and not despairing at the ultimate futility of absolute clinical differentiation, it turns rather to the minute analysis of the perverted functions of individual minds, comparing them with each other, point by point, both in health and disease. Under the influence of this conception, whatever further growth the symptomatologic and clinical methods are capable of will proceed to the best advantage.

But Farrar's hopes too were to be only partially fulfilled. Freud's discovery and application of a dynamic psychology was contemporaneous with the development of modern clinical psychiatry. His concepts have continued to fertilize psychiatric thought throughout this century. But he believed that psycho-analysis was not appropriate to the older patient, whose emotional disturbance could often be interpreted as a reaction to a reality situation which was unmodifiable. Freud (1924) was pessimistic about the application of classical psycho-analytical methods of treatment: 'Near or above the fifties the elasticity of the mental processes, on which the treatment depends, is as a rule lacking—old people are no longer educable . . . the mass of material to be dealt with would prolong the duration of the treatment indefinitely . . . with persons who are too far advanced in years it fails because owing to the accumulation of material so much time would be required that the end of the cure would be reached at a

period of life in which much importance is no longer attached to nervous health'.

As a result, the problems of the aged were not subjected to the scrutiny which other syndromes evoked. The vigorous debates which characterized the early decades of the century, when psycho-analysis was codifying its discoveries, did little to stimulate interest in the psycho-dynamics of ageing.

Psychiatrists were at first fully occupied in the application of Kraepelinian concepts to diagnosis and classification. The mental reactions precipitated by the First World War; Wagner-Jauregg's discovery of an effective treatment for general paralysis of the insane (1917); Meduna's introduction of convulsive therapy (1933); Sakel's insulin coma therapy for schizophrenia (1938); all combined to concentrate attention and effort on the diseases of early and middle life. In general, the problems of the elderly continued to be neglected because of their presumed irreversibility.

The status of the psychiatric disorders of later life, in the first forty years of this century, have been well summarized by Roth (1962).

That a small proportion of cases of affective psychosis and even schizophrenia begins in old age, was of course, well known, but accounts of mental disease among the aged implied that the commonest cause of persistent affective and paranoid symptoms among the aged was cerebral disease. According to this viewpoint, functional syndromes, whether depressive or paranoid, as also delirious states, were thought to be the indicators of progressive decline in intellect and personality. The frequent occurrence in the aged of mild impairment of memory, of physical illness and disability of various kinds, and of inconspicuous neurological changes tended to foster views of this nature. It was consequently common practice when depressive or paranoid psychosis appeared for the first time in old age, to search for neurological signs or for some evidence of a decline in intellectual efficiency which might account for the illness in terms of organic disease of the brain. Weight was thus often given to clinical findings of a quite subtle character, to mild memory impairment, the presence of hypertension or peripheral and retinal arteriosclerosis or the results of deterioration tests whose validity as measures of early degenerative change in the brain is far from established.

These views underwent a gradual refinement in the years during which modern physical treatments began to be extensively used in psychiatry. Though even after electro-convulsive therapy had been accepted as the treatment of choice for depressive illnesses, there was reluctance to use it in the elderly because of its possible dangers. Mayer-Gross (1945) was one of the first to demonstrate its effectiveness, with reasonable precaution, even in advanced old age. Finally, with the arrival of simpler anaesthetics and relaxants, it became generally accepted that elderly depressives could respond quite as dramatically and completely as did their juniors. More accurate diagnosis and classification became profitable. Affective disorder—depressive or manic; and paranoid states were added to the geriatric nomenclature.

The great value of Roth's classic study: *The Natural History of Mental Disorder in Old Age* (1955) was to define more clearly this grouping and to show that each had a distinct pattern of outcome. Moreover, he showed that there was little overlap between the different categories and that affective disorder, late paraphrenia and acute confusion were each entities largely independent of the two main causes of progressive dementia in old age, senile and arteriosclerotic psychosis. His findings have not been challenged save for his concept of late paraphrenia, and that mainly on the grounds that he accorded it a frequency of incidence in excess of that found by most other investigators.

This rebirth of clinical interest has been maintained by other workers, notably Post and Kay, whose contributions have been principally on the affective side. Further study and refinement, particularly within the organic divisions, is urgently required. A monograph by Allison (1962), a review of 200 patients with organic mental states, marks a notable step in this direction.

An illustration of the general neglect of the organic psychoses is the fact that sixty years after their histological delineation, there was still no general agreement about the nature of the relationship between cerebral degeneration and dementia. Wolf (1959) after reviewing the literature could conclude: 'No good correlation exists between the degree, the distribution and the character of the various abnormal changes and the age and state of neuro-function of the individual'. But the recent studies of Corsellis (1962) and of Blessed and Tomlinson (1966) using modern methods of assessment, have shown a high degree of clinico-pathological correlation.

The psychiatrist faced with a diagnostic problem cannot yet rely too firmly on psychological aid. Dementia is one of those words, freely but loosely used, and difficult to define accurately in useful clinical terms. In the past, too much emphasis has been placed on its purely intellectual implications. In some recent studies, even fairly simple clinical assessments—including personality and behavioural variables—have given better correlations with performance and prognosis than have conventional psychometrics. Zangwill (1964) has commented on this situation which perturbs psychologists no less than psychiatrists. The studies of Welford (1958) have thrown new light on the changes in memory, learning and sensory motor performance in the ageing brain. Studies by Kendrick and Post (1967), Irving (1970), and others indicates the increasing interest among clinical psychologists in these processes, and the hope that this will lead to the development of techniques providing more accurate definition of the expression of cerebral changes associated with dementia.

Though the electro-encephalogram has been a disappointment to some in the elucidation of brain pathology, evidence is accumulating that it is indispensable for diagnosis and treatment. In research studies, correlations have been as good as the companion clinical work deserved. In a subject where we have too few diagnostic tools, one which gives us an additional clinical sign cannot be ignored.

In therapy, apart from the drug treatment of depression and schizophrenic illness, most of our advances have been in the socio-medical area. Milieu, occupational and recreational therapies and the therapeutic community all have their place in the handling of the organic patient. Here, drugs have in general been disappointing save for their symptomatic effects. Earlier referral, more powerful antibiotics and a closer search for nutritional and bacterial causes, have resulted in increasingly successful treatment of the toxic confusional state. But though mortality has fallen, this has not been completely reflected in discharge rates; for florid psychosis may resolve only to reveal an underlying dementia.

The psychodynamics of the ageing process and psychotherapeutic methods are still a neglected area of study, though the publications of the Boston Society for Gerontologic Psychiatry (1963, 1965) mark the beginnings of a comprehensive examination of the problem. Rechtschaffen (1959) in a review of the literature concludes: 'If, at best, social reality is cruel, what can therapy hope to accomplish? There is genuine despair

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when a therapist himself cannot visualise a happier life for the geriatric patient who is physically ill, mentally clouded, socially ostracised and approaching death. When the values of our culture are largely those of productivity and vigour, what satisfactions can psychotherapy produce for the old person? This is more than a rhetorical question, for it may be the principal obligation of geriatric psychotherapy to investigate the gratifications that can be evolved for older persons.'

The welfare and social services are contributing much to preventive and supportive psychiatry. The recent H.M.S.O. publication *Services for the Elderly with Mental Disorder* marks official recognition of some of the tasks ahead and gives clear advice for their solution.

Attitudes to ageing and indeed the behaviour of the ageing are cultural phenomena. Will traditional attitudes continue to serve in an era when survival to old age is the rule rather than the exception?

Sjogren's epidemiological study (1963) suggests that the morbidity risk for senile dementia increases tenfold from aged seventy to eighty-five. It is likely that other brain syndromes show similar trends. Advances in the medical care of the elderly have been substantial. Is the future prospect then to be an unhealthy mind in a healthy body?

These are some of the questions facing geriatric psychiatry today.

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News, Notes and Queries

THE MELBOURNE PHARMACY

A CORRECTION

In the October 1971 number of *Medical History* a note appeared over my name entitled 'A notable pharmacy comes to Melbourne' (pp. 401-2). In this it was stated that the shop was opened in London at 29 Chapel Street, Belgravia, in 1915. This was the date given to me by Savory & Moore Ltd. although it was questioned on the grounds of style and obvious age of the fittings; in fact the date is not correct. Savory & Moore opened their branch in Chapel Street about 1849 and they are listed as being at that address in the *London Directory* of 1853. The *Registers of Pharmaceutical Chemists and Chemists and Druggists* of 1869 lists the shop with Charles Hunt as manager, a position which he held until 1910 when he retired. The date of 1849-50 is suggested by many of the pre-*British Pharmacopoeia* drugs listed on the labels of the drawers and by the type of nails, screws and details of joinery used in the fittings. I had no doubt as to the approximate age of the fittings, but in view of the dogmatic date given to me by Savory & Moore Ltd. I suggested that the furniture had come from another, older shop. It now appears that this is original to the shop when it opened in 1849-50 although some modifications to the layout were made at a later date.

I am most grateful to Dr. J. K. Crellin of the Wellcome Institute of the History of Medicine for providing me with the information which cleared up the confusion. My faith in primary sources has received a slight jolt.

K. F. RUSSELL