THE SLEEPING-SICKNESS (*ENCEPHALITIS LETHARGICA*)

In the winter of 1916–17, in Vienna and other cities, a 'new' illness suddenly appeared, and rapidly spread, over the next three years, to become world-wide in its distribution. Manifestations of the sleeping-sickness[15] were so varied that no two patients ever presented exactly the same picture, and so strange as to call forth from physicians such diagnoses as epidemic delirium, epidemic schizophrenia, epidemic Parkinsonism, epidemic disseminated sclerosis, atypical rabies, atypical poliomyelitis, etc., etc. It seemed, at first, that a thousand new diseases had suddenly broken loose, and it was only through the profound clinical acumen of Constantin von Economo, allied with his pathological studies on the brains of patients who had died, and his demonstration that these, besides showing a unique pattern of damage, contained a sub-microscopic, filter-passing agent (virus) which could transmit the disease to monkeys, that the identity of this protean disease was established. *Encephalitis lethargica* – as von Economo was to name it – was a Hydra with a thousand heads.[16]

[15] The term 'sleeping-sickness' is used in America to designate both the African, parasite-borne, endemic disease *(trypanosomiasis)* and the epidemic, virus-borne, *encephalitis lethargica;* in England, however, the latter is often called 'sleepy-sickness.'

[16] Thus there arose the most baffling clinical and epidemiological perplexities. The first recognition in England that new and strange disease-syndromes were everywhere afoot, dates from the first weeks of 1918, and one may recapture the excitement of these early reports by looking at *The Lancet* for April 20th of that year and the extraordinary report put out by the Stationery Office in October 1918 (see His Majesty's Stationery Office, 1918). There had been earlier reports – from France, Austria, Poland, and Romania – as far back as the winter of 1915–16, but these were apparently unknown in England, due to the difficulties of disseminating information in wartime. One may see from the HMSO Report that confusion reigned, and how reports of the new and unidentified disease came in under the most various of names: botulism, toxic ophthalmoplegia, epidemic stupor, epidemic lethargic encephalitis, acute polioencephalitis, Heine-Medin disease, bulbar paralysis, hystero-epilepsy, acute dementia, and sometimes just 'an obscure disease with cerebral symptoms.' This chaos continued until the great clarifying and unifying work of von Economo, after whom we properly name this disease.

Cruchet, in France, described forty cases of 'subacute encephalomyelitis' ten days before von Economo; neither knew of the other's work, for Paris and Vienna were on opposite sides in the War and, as was often remarked in later years, communication about the disease was slower than communication of the disease itself. Questions of priority were fanned, not only by the discoverers themselves but by forces of national animus and pride; for some years the French literature spoke of 'Cruchet's disease' while the German literature spoke of 'von Economo's disease.' The rest of the world, neutrally, spoke of *encephalitis lethargica,* epidemic encephalitis, chronic encephalitis, etc. Indeed, almost every individual neurologist had their own name for it: for Kinnier Wilson it was 'mesencephalitis,' for Bernard Sachs it was 'basilar encephalitis.' For the public, it was simply 'sleepy sickness.'

Although there had been innumerable smaller epidemics in the past, including the London sleeping-sickness of 1672–3, there had never been a world-wide pandemic on the scale of that which started in 1916–17. In the ten years that it raged, this pandemic took or ravaged the lives of nearly five million people before it disappeared, as mysteriously and suddenly as it had arrived, in 1927.[17] A third of those affected died in the acute stages of the sleeping-sickness, in states of coma so deep as to preclude arousal, or in states of sleeplessness so intense as to preclude sedation.[18]

[17] There was some coincidence and overlap of the great encephalitis pandemic with the world-wide 'flu' pandemic – as thirty years earlier the Italian *'nona'* was preceded by a virulent if local influenza epidemic. It is probable, but not certain, that the influenza and the encephalitis reflected the effects of two different viruses, but it seems possible, and even probable, that the influenza epidemic in some way paved the way for the encephalitis epidemic, and that the influenza virus potentiated the effects of the encephalitis virus, or lowered resistance to it in a catastrophic way. Thus, between October 1918 and January 1919, when half the world's population was affected by the influenza or its consequences, and more than twenty-one million people died, the encephalitis assumed its most virulent form. If the sleeping-sickness was mysteriously 'forgotten,' the same is true of the great influenza (which had been the most murderous epidemic since the Black Death of the Middle Ages). In the words of H. L. Mencken, written in 1956: 'The epidemic is seldom mentioned, and most Americans have apparently forgotten it. This is not surprising. The human mind always tries to expunge the intolerable from memory, just as it tries to conceal it while current.'

[18] Absolute inability to sleep (agrypnia), in such patients, even without other symptoms, proved fatal in ten to fourteen days. The plight of such patients (in

Patients who suffered but survived an extremely severe somnolent/insomniac attack of this kind often failed to recover their original aliveness. They would be conscious and aware – yet not fully awake; they would sit motionless and speechless all day in their chairs, totally lacking energy, impetus, initiative, motive, appetite, affect, or desire; they registered what went on about them without active attention, and with profound indifference. They neither conveyed nor felt the feeling of life; they were as insubstantial as ghosts, and as passive as zombies: von Economo compared them to extinct volcanoes. Such patients, in neurological parlance, showed 'negative' disorders of behaviour, i.e. no behaviour at all. They were ontologically dead, or suspended, or 'asleep' – awaiting an awakening which came (for the tiny fraction who survived) fifty years later.

If these 'negative' states or *absences* were more varied and severe than those seen in common Parkinson's disease, this was even truer of the innumerable 'positive' disorders or pathological *presences* introduced by the sleeping-sickness: indeed, von Economo, in his great monograph, enumerated more than five hundred distinct forms or varieties of these.[19]

whom the cerebral mechanisms for sleep had been destroyed) showed, for the first time, that sleep was a physiological necessity. Sometimes these insomniac states were accompanied by intense drive, driving those affected into a veritable frenzy of body and mind, a state of ceaseless excitement and movement, until their death (from exhaustion) a week or ten days later. Although terms like 'mania' and 'catatonic excitement' were sometimes used, these wild states more closely resembled rabies (for which they were sometimes mistaken).

Above all they resembled the states of intense cerebral excitement, with tremendous pressure of thought and movement, which may be seen in acute ergot poisoning: an amazing picture of this, as it affected an entire French village convulsed by accidental ergot poisoning (due to contamination of their bread), is given by John G. Fuller in *The Day of St. Anthony's Fire.* His picture of those affected, unable to sleep, talking excitedly all day and all night, making faces, making noises, constantly, compulsively moving and ticcing, driven by a rush and energy which gave no respite, until death from exhaustion came a week later, immediately made me think of those who were stricken by a hyperkinetic-insomniac form of *encephalitis lethargica.*

[19] The enormous range of post-encephalitic symptoms – particularly its unique disturbances of sleep, of sexuality, of affect, of appetite – fascinated physiologists as well as physicians, and led, in the 1920s and 1930s, to the founding of behavioural neurology as a science. Yet in this booming, buzzing confusion (which McKenzie called a 'chaos'), there seemed to von Economo to be three

Parkinsonian disorders, of one sort or another, were perhaps the commonest of these disorders, although their appearance was often delayed until many years after the acute epidemic. Post-encephalitic Parkinsonism, as opposed to ordinary or idiopathic Parkinsonism, tended to show less in the way of tremor and rigidity – indeed, these were sometimes completely absent – but much severer states of 'explosive' and 'obstructive' disorders, of akinesia and akathisia, push and resistance, hurry and impediment, etc., and also much severer states of the compliant-perseverative type of akinesia which Gowers had compared to catalepsy. Many patients, indeed, were swallowed up in states of Parkinsonian akinesia so profound as to turn them into living statues – totally motionless for hours, days, weeks, or years on end. The very much greater severity of these encephalitic and post-encephalitic states revealed that *all* aspects of being and behaviour – perceptions, thoughts, appetites, and feelings, no less than movements – could also be brought to a virtual standstill by an active, constraining Parkinsonian process.

Almost as common as these Parkinsonian disorders, and frequently co-existing with them, were *catatonic* disorders of every sort. It was the occurrence of these which originally gave rise to the notion of an 'epidemic schizophrenia,' for catatonia – until its appearance in the encephalitis epidemic – was thought to be part-and-parcel of the schizophrenic syndrome. The majority of patients who were rendered catatonic by the sleeping-sickness were *not* schizophrenic, and showed that catatonia might, so to speak, be approached by a direct physiological path, and was not

main patterns of involvement, or 'types' of disease: somnolent-ophthalmoplegic, hyperkinetic, and myostatic-akinetic (in his terms), corresponding to three main patterns of neuronal involvement (the first of these arising from involvement of the brainstem, of what were later to be delineated as 'arousal-systems' in this area; the last of these – which corresponds to Parkinsonism – to the involvement of the substantia nigra; and the most complex disorders of all – the impulsive and emotional hyperkinetic-tourettic ones – to involvement in the diencephalon and hypothalamus).

Hess's great studies of subcortical function (for which he was later awarded the Nobel Prize) were stimulated in the first place by his wonder at the novel symptoms of the *encephalitis lethargica* (this is described in the preface to his monograph, *Diencephalon,* 1954).

always a defensive manoeuvre undertaken by schizophrenic patients at periods of unendurable stress and desperation.[20]

The general forms or 'phases' of encephalitic catatonia were closely analogous to those of Parkinsonism, but were at a higher and more complex level, and were usually experienced as subjective states which had exactly the same form as the observable behavioural states. Thus some of these patients showed automatic compliance or 'obedience,' maintaining (indefinitely, and apparently without effort) any posture in which they were put or found themselves, or 'echoing' words, phrases, thoughts, perceptions, or actions in an unvarying circular way, once these had been suggested to them (palilalia, echolalia, echopraxia, etc.). Other patients showed disorders of a precisely antithetical kind ('command negativism,' 'block,' etc.) immediately preventing or countermanding any suggested or intended action, speech, or thought: in the severest cases, 'block' of this type could cause a virtual obliteration of all behaviour and also of all mental processes (see the case of Rose R., for example). Such constrained catatonic patients – like constrained Parkinsonians – could suddenly burst out of their immobilized states into violent movements or frenzies: a great many of the tics seen at the time of the epidemic, and subsequently, showed themselves to be interchangeable with 'tics of immobility,' or catatonia (Ferenczi, indeed, called tics 'cataclonia').

An immense variety of involuntary and compulsive movements were seen during the acute phase of the encephalitis, and for a few years thereafter: myoclonic jerks and spasms; states of mobile spasm (athetosis), dystonias and dystonic contortions (e.g. torticollis), with somewhat similar functional organizations to that of Parkinsonian rigidity; desultory, forceless movements dancing from one part of the body to another (chorea); and a

[20] Post-encephalitic patients, when they can speak – which in the severest cases was not rendered possible until half a century later, when they were given L-DOPA – are thus able to provide us with uniquely detailed and accurate descriptions of states of catatonic 'entrancement,' 'fascination,' 'forced thinking,' 'thought-block,' 'negativism,' etc., which schizophrenic patients, usually, are unable or unwilling to do, or which they will only describe in distorted, magical, 'schizophrenic' terms.

wide spectrum of tics and compulsive movements at every functional level – yawning, coughing, sniffing, gasping, panting, breath-holding, staring, glancing, bellowing, yelling, cursing, etc. – which were enactions of sudden *urges*.[21]

At the 'highest' level the *encephalitis lethargica* presented itself as neurotic and psychotic disorders of every kind, and a great many patients affected in this way were originally considered to have 'functional' obsessional and hysterical neuroses, until the development of other symptoms indicated the encephalitic aetiology of their complaints. It is of interest, in this connection, that 'oculogyric crises' were considered to be purely 'functional' and hysterical for several years after their first appearance.

Clearly differentiated forms of affective compulsion were common in the immediate aftermath of the sleeping-sickness, especially erotomanias, erethisms, and libidinal excitement, on the one hand, and tantrums, rages, and destructive outbursts on the other. These forms of behaviour were most clearly and undisguisedly manifest in children, who sometimes showed abrupt changes of character, and suddenly became impulsive, provocative, destructive, audacious, salacious, and lewd, sometimes to a quite uncontrollable degree: such children were often labelled 'juvenile psychopaths' or 'moral aments.'[22] Sexual and destruc-

[21] In Thom Gunn's poem 'The Sense of Movement,' there occurs the following pivotal line:

'One is always nearer by not being still.'

This poem deals with the basic *urge* to *move*, a movement which is always, mysteriously, *towards*. This is not so for the Parkinsonian: he is *no* nearer for not being still. He is no nearer to anything by virtue of his motion; and in this sense, his motion is not genuine movement, as his lack of motion is not genuine rest. The road of Parkinsonism is a road which leads nowhere; the land of Parkinsonism is paradox and dead end.

[22] Among the many eminent physicians who were deeply concerned with the changes in character which might be wrought by the sleepy-sickness was Dr G. A. Auden (father of the poet W. H. Auden). Such changes, Dr Auden stressed, could not always be regarded as purely deleterious or destructive in nature. Less zealous to 'pathologize' than many of his colleagues, Dr Auden noted that some of those affected, especially children, might be 'awakened' into a genuine (if morbid) brilliance, into unexpected and unprecedented heights and depths. This notion of a disease with a 'Dionysiac' potential was often

tive outbursts were rarely outspoken in adults, being 'converted' (presumably) to other, more 'allowable,' reactions and expressions. Jelliffe,[23] in particular, who undertook lengthy analysis of some highly intelligent post-encephalitic patients, showed unequivocally how accesses of erotic and hostile feeling could be and were 'converted,' not only into neurotic and psychotic behaviour, but into tics, 'crises,' catatonia, and even Parkinsonism. Adult post-encephalitic patients thus showed an extraordinary ability to 'absorb' intense feeling, and to express it in indirect physiological terms. They were gifted – or cursed – with a pathologically extravagant expressive facility or (in Freud's term) 'somatic compliance.'

Nearly half the survivors became liable to extraordinary crises, in which they might experience, for example, the simultaneous and virtually instantaneous onset of Parkinsonism, catatonia, tics, obsessions, hallucinations, 'block,' increased suggestibility or negativism, and thirty or forty other problems; such crises would last a few minutes or hours, and then disappear as suddenly as they had come.[24] They were highly individual, no two patients

discussed in the Auden household, and formed an enduring theme in W. H. Auden's thought. Many other artists at this time, perhaps most notably Thomas Mann, were struck by the world-wide spectacle of a disease which could – however ambiguously – raise cerebral activity to a more awakened and creative pitch: in *Doctor Faustus* the Dionysiac fever is attributable to neurosyphilitic infection; but a similar allegory of extraordinary excitement, followed (and *paid for*) by attrition and exhaustion, could as well apply to post-encephalitic infection.

[23] Smith Ely Jelliffe, a man equally eminent as neurologist and psychoanalyst, was perhaps the closest observer of the sleeping-sickness and its sequelae. This was his summing-up, looking back on the epidemic: 'In the monumental strides made by neuropsychiatry during the past ten years no single advance has approached in importance that made through the study of epidemic encephalitis. No individual group of disease-reactions has been . . . so far-reaching in modifying the entire foundations of neuropsychiatry in general . . . *An entirely new orientation has been made imperative.*' (Jelliffe, 1927)

[24] The astonishing variability of such crises, and their openness to suggestion, were well shown in another patient, Lillian W., whose history is not in this book. Lillian W. had at least a hundred clearly different forms of crisis: hiccoughs; panting attacks; oculogyrias; sniffing attacks; sweating attacks; attacks in which her left shoulder would grow flushed and warm; chattering of the teeth; paroxysmal ticcing attacks; ritualized iterative attacks, in which she would tap one foot

ever having exactly the same sort of crises, and they expressed, in various ways, fundamental aspects of the character, personality, history, perception, and fantasies of each patient.[25] These

in three different positions, or dab her forehead in four set places; counting attacks; verbigerative attacks, in which certain set phrases were said a certain number of times; fear attacks; giggling attacks, etc., etc. Any allusion (verbal or otherwise) to any given type of crisis would infallibly call it forth in this patient.

Lillian W. would also have bizarre 'miscellaneous' crises, in which a great variety of phenomena (sniffing, oculogyria, panting, counting, etc., etc.) would be thrown together in unexpected (and seemingly senseless) combinations; indeed new and strange combinations were continually appearing. Although I observed dozens of these complex crises I was almost never able to perceive any physiological or symbolic unity in them, and after a while I ceased to look for any such unity, and accepted them as absurd juxtapositions of physiological oddments, or, on occasion, improvised collages of physiological bric-à-brac. This was also how Mrs W., a talented woman with a sense of humour, regarded her own miscellaneous crises: 'They are just a mess,' she would say, 'like a junk shop, or a jumble-sale, or the sort of stuff you just throw in the attic.' *Sometimes,* however, one could see patterns which were clear-cut but unintelligible, or patterns which seemed to hint, tantalizingly, at some scarcely imaginable unity or significance; and of these crises Mrs W. would say: 'This one's a humdinger, a surrealistic attack – I *think* it's saying something, but I don't know what it is, nor do I know what language it's in.' Some of my students who happened to witness such attacks also received a surrealistic impression: 'That's absolutely wild,' one of them once said. 'It's just like a Salvador Dali!' Another student, fantastically inclined, compared her crises to uncanny, unearthly buildings or music ('Martian churches or Arcturan polyphonies'). Although none of us could agree on the 'interpretation' of Lillian W.'s crises, we all felt them as having a strange fascination – the fascination of dreams, or peculiar art-forms; and, in this sense, if I sometimes thought of Parkinsonism as a relatively simple and coherent dream of the midbrain, I thought of Lillian W.'s crises as surrealistic deliria concocted by the forebrain.

[25] Not infrequently a single, sensational *moment-of-being* is 'caught' by a crisis, and preserved thereafter. Thus Jelliffe (1932) alludes to a man whose first oculogyric crisis came on during a game of cricket, when he had suddenly to fling one hand up to catch a high ball (he had to be carried off the field still entranced, with his right arm still outstretched and clutching the ball). Subsequently, whenever he had an oculogyric crisis, these would be ushered in by a *total replay* of this original, grotesque, and comic moment: he would suddenly feel it was 1919 once again, an unusually hot July afternoon, that the Saturday match was in progress again, that Trevelyan had just hit a probable 'six,' that the ball was approaching him, and that he had to catch it – RIGHT NOW! Similar, dramatic moments-of-being may also be incorporated into epileptic seizures, especially those of psychomotor type; Penfield and Perot, who have provided the most detailed accounts of this, suggest that 'fossilized memories' may be preserved in the cortex – memories which are normally dormant and forgotten, but which can suddenly come to life and be re-activated under special conditions. Such phe-

crises could be greatly influenced, for better or worse, by suggestion, emotional problems, or current circumstances. Crises of all sorts became rare after 1930, but I stress them and their characteristics because they show remarkable affinities to certain states induced by L-DOPA, not merely in post-encephalitic patients, but in the normally much stabler patients with common Parkinson's disease.

One thing, and one alone, was (usually) spared amid the ravages of this otherwise engulfing disease: the 'higher faculties' – intelligence, imagination, judgement, and humour. These were exempted – for better or worse. Thus these patients, some of whom had been thrust into the remotest or strangest extremities of human possibility, experienced their states with unsparing perspicacity, and retained the power to remember, to compare, to dissect, and to testify. Their fate, so to speak, was to become unique witnesses to a unique catastrophe.

THE AFTERMATH OF THE SLEEPING-SICKNESS (1927–67)

Although many patients seemed to make a complete recovery from the sleeping-sickness, and were able to return to their former lives, the majority of them subsequently developed neurological or psychiatric disorders, and, most commonly, Parkinsonism. Why they should have developed such 'post-encephalitic syndromes' – after years or decades of seemingly perfect health – is a mystery, and has never been satisfactorily explained.

nomena endorse the notion that our memories, or beings, are 'a collection of moments' (see n. 133, p. 256).

These post-encephalitic syndromes were very variable in course: sometimes they proceeded rapidly, leading to profound disability or death; sometimes very slowly; sometimes they progressed to a certain point and then stayed at this point for years or decades; and sometimes, following their initial onslaught, they remitted and disappeared. This great variation of pattern is also a mystery, and seems to admit of no single or simple explanation.

Certainly it could not be explained in terms of microscopically visible disease-processes, as was considered at one time. Nor was it true to say that post-encephalitic patients were suffering from a 'chronic encephalitis,' for they showed no signs of active infection or inflammatory reaction. There was, moreover, a rather poor correlation between the severity of the clinical picture and that of the pathological picture, insofar as the latter could be judged by microscopic or chemical means: one saw profoundly disabled patients with remarkably few signs of disease in the brain, and one saw evidences of widespread tissue-destruction in patients who were scarcely disabled at all. What *was* clear, from these discrepancies, was that there were many other determinants of clinical state and behaviour besides localized changes in the brain; it was clear that the susceptibility or propensity to Parkinsonism, for example, was not a fixed expression of lesions in the 'Parkinsonism-centre' of the brain, but dependent on innumerable other 'factors' in addition.

It seemed, as Jelliffe and a few others repeatedly stressed, as if the *'quality'* of the individual – his 'strengths' and 'weaknesses,' resistances and pliancies, motives and experiences, etc. – played a large part in determining the severity, course, and form of his illness. Thus, in the 1930s, at a time of almost exclusive emphasis on specific mechanisms in physiology and pathology, the strange evolutions of illness in these post-encephalitic patients recalled Claude Bernard's concepts of the *terrain* and the *milieu interne,* and the immemorial ideas of 'constitution,' 'diathesis,' 'idiosyncrasy,' 'predisposition,' etc., which had become so unfashionable in the twentieth century. Equally clear, and beautifully analysed by Jelliffe, were the effects of the external environment, the circumstances and vicissitudes of each patient's life. Thus, post-encephalitic illness could by no means be considered a simple

disease, but needed to be seen as an individual creation of the greatest complexity, determined not simply by a primary disease-process, but by a vast host of personal traits and social circumstances: an illness, in short, like neurosis or psychosis, a coming-to-terms of the sensitized individual with his total environment. Such considerations, of course, are of crucial importance in understanding the total reactions of such patients to L-DOPA.

There remain today a few survivors of the encephalitis who, despite Parkinsonism, tics, or other problems, still lead active and independent lives (see for instance the case of Cecil M.). These are the fortunate minority, who for one reason or another have managed to keep afloat, and have not been engulfed by illness, disability, dependence, demoralization, etc. – Parkinson's 'train of harassing evils.'

But for the majority of post-encephalitic patients – in consequence of the basic severity of their illness, their 'weaknesses,' their propensities, or their misfortunes – a much darker future was in store. We have already stressed the inseparability of a patient's illness, his self, and his world, and how any or all of these, in their manifold interactions, through an infinity of vicious circles, can bring him to his nadir of being. How much is contributed by this, and that, and that, and that, can perhaps be unravelled by the most prolonged, intimate contact with individual patients, but cannot be put in any general, universally applicable form. One can only say that most of the survivors went down and down, through circle after circle of deepening illness, hopelessness, and unimaginable solitude, their solitude, perhaps, the least bearable of all.

> As *Sicknes* is the greatest misery, so the greatest misery of sicknes, is *solitude* . . . *Solitude* is a torment which is not threatened in *hell* itselfe.
>
> DONNE

The character of their illness changed. The early days of the epidemic had been a time of ebullition or ebullience, pathologically speaking, full of movements and tics, impulsions and impetuosities, manias and crises, ardencies and appetencies. By the late twenties, the acute phase was over, and the encephalitic syndrome started to cool or congeal. States of immobility and arrest had been distinctly uncommon in the early 1920s, but from 1930 onwards started to roll in a great sluggish, torpid tide over many of the survivors, enveloping them in metaphorical (if not physiological) equivalents of sleep or death. Parkinsonism, catatonia, melancholia, trance, passivity, immobility, frigidity, apathy: this was the quality of the decades-long 'sleep' which closed over their heads in the 1930s and thereafter. Some patients, indeed, passed into a timeless state, an eventless stasis, which deprived them of all sense of history and happening. Isolated circumstances – fire alarms, dinner-gongs, the unexpected arrival of friends or news – might set them suddenly and startlingly alive for a minute, wonderfully active and agog with excitement. But these were rare flashes in the depths of their darkness. For the most part, they lay motionless and speechless, and in some cases almost will-less and thoughtless, or with their thoughts and feelings unchangingly fixed at the point where the long 'sleep' had closed in upon them. Their minds remained perfectly clear and unclouded, but their whole beings, so to speak, were encysted or cocooned.

Unable to work or to see to their needs, difficult to look after, helpless, hopeless, so bound up in their illnesses that they could neither react nor relate, frequently abandoned by their friends and their families, without specific treatment of any use to them – these patients were put away in chronic hospitals, nursing homes, lunatic asylums, or special colonies; and there, for the most part, they were totally forgotten – the lepers of the present century; there they died in their hundreds of thousands.

And yet some lived on, in diminishing numbers, getting older and frailer (though usually looking younger than their age), inmates of institutions, profoundly isolated, deprived of experience, half-forgetting, half-dreaming of the world they once lived in.