OLIVER SACKS

AWAKENINGS

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ACKNOWLEDGEMENTS

My first (and infinite) debt is to the remarkable patients at Mount Carmel Hospital, New York, whose stories I relate in this book, and to whom *Awakenings* was originally dedicated.

It is difficult now, looking back over a quarter of a century, to recall all of those at Mount Carmel who were concerned with our patients, and who directly or indirectly contributed to *Awakenings*; but I have warm memories of the nursing staff – Ellen Costello, Eleanor Gaynor, Janice Grey, and Melanie Epps; of the medical staff – Walter Schwartz, Charles Messeloff, Jack Sobel, and Flora Tabbador; of our speech therapist, and my closest helpmate in the crucial three years when our patients were being awakened, Margie Kohl Inglis; of our EEG technician, who was my collaborator on 'The Electrical Basis of Awakenings,' Chris Carolan; of our pharmacist, Bob Malta, who spent hours encapsulating L-DOPA, surrounded by clouds of dopaminergic dust; and of devoted occupational and physiotherapists. I have to single out our music therapists – Kitty Stiles in the early years of our patients' awakenings, and Connie Tomaino since – with whom I have had the closest relation, for music has been the profoundest non-chemical medication for our patients.

I owe a special debt to my English colleagues at the Highlands Hospital, for enabling me to keep in touch with an extraordinary group of patients, profoundly similar to, yet profoundly different from, ours at Mount Carmel. In particular, I must acknowledge the friendly assistance of Gerald Stern and Donald Calne, who helped 'awaken' these patients in 1969; James Sharkey and Rodwin Jackson, who between them have looked after these patients since 1945; Bernard Thompson, a nurse who was with the patients for many years; and, above all, James Purdon Martin, who had known these (and other) post-encephalitic patients for more than sixty years. He made a special visit to Mount Carmel in

1969, to see our patients in the first flush of their 'awakenings,' and was thereafter something of a father figure and a guide.

Innumerable other colleagues and friends have helped me, or *Awakenings*, along the way: D. P. dePaola, Roger Duvoisin, Stanley Fahn (and the Basal Ganglia Club), Ilan Golani, Elkhonon Goldberg, Mark Homonoff, William Langston, Andrew Lees, Margery Mark, Jonathan Mueller, H. Narabayashi, Isabelle Rapin, Robert Rodman, Israel Rosenfield, Sheldon Ross, Richard Shaw, Bob Wasserman. Among these I should especially mention Jonathan Miller, who preserved a copy of the 1969 manuscript, when I had destroyed the original, and conveyed this to Colin Haycraft, my first editor-publisher (and who, much later, was to make the remarkable BBC film portrait of Ivan Vaughan, *Ivan*); Eric Korn, who helped edit the 1976 edition; Lawrence Weschler, who knew many of the post-encephalitic patients at Mount Carmel, and has discussed aspects of *Awakenings* with me in every way, intensively, for ten years; and Ralph Siegel, who is now working with me on chaos theory and 'awakenings.'

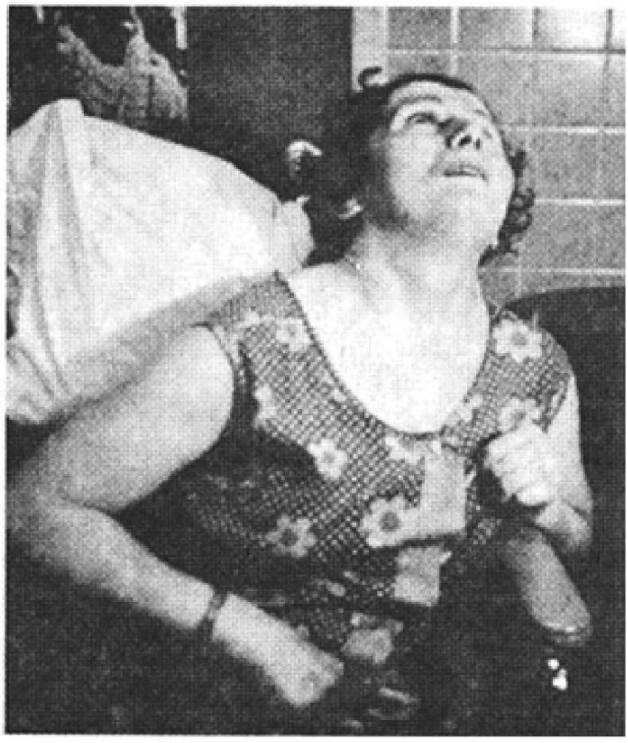
A special place must be reserved for those colleagues who are themselves patients, and who know and can describe the world of the Parkinsonian with an incomparable authority, from the inside. Among these have been Ivan Vaughan, Sidney Dorros, and Cecil Todes (all of whom have written their own accounts of living with Parkinson's); and Ed Weinberger, who has provided powerful insights and images for me in innumerable ways. Many people with Tourette's syndrome have helped me to understand their own condition, a condition with many similarities to that of hyperkinetic encephalitis. Finally, my own post-encephalitic patient, Lillian Tighe, whom I have known now for over twenty years: Lillian was central in the documentary film of *Awakenings*, and was an inspiration during the making of the feature film of it too.

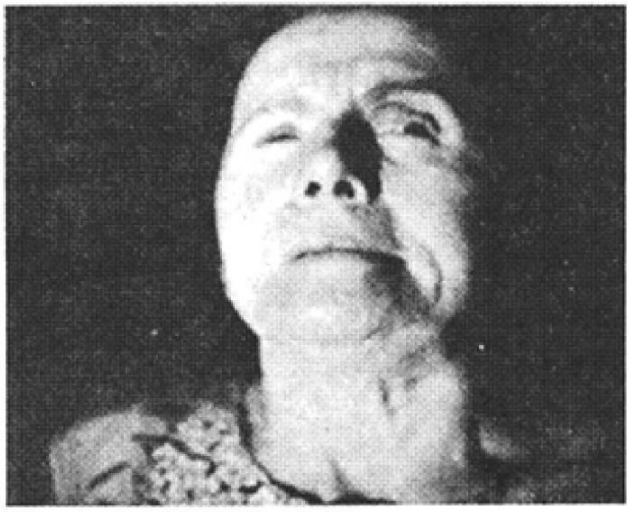
Many people have devoted their creative talents to writing, producing, or performing dramatic versions of *Awakenings*: first and foremost, Duncan Dallas, of Yorkshire Television, who made a beautiful documentary film of *Awakenings* in

1973 – this contains unforgettable images of the patients and events I write of in *Awakenings*, and I wish could be seen by everyone who reads it; Harold Pinter, who in 1982 sent me an extraordinary play *(A Kind of Alaska)* inspired by *Awakenings*, which was first performed in England at the National Theatre in October of that year; John Reeves, who produced a moving radio adaptation of *Awakenings* for the Canadian Broadcasting Corporation in 1987; Arnold Aprill of City Lit, who masterminded a remarkable stage version in Chicago in 1987; Carmel Ross, who produced an audio version of *Awakenings*; and now the cast and crew of the feature film of *Awakenings* – in particular, Walter Parkes and Larry Lasker, its producers; Steve Zaillian, its screenwriter; Penny Marshall, its director; and, of course, its great actors, Robert De Niro and Robin Williams.

Finally, I am grateful to my agent, Suzanne Gluck, and to the many editors of *Awakenings*, who have guided it through its many editions in the last seventeen years: Colin Haycraft, Ken McCormick, Julia Vellacott, Anne Freedgood, Mike Petty, Bill Whitehead, Jim Silberman, Rick Kot, and Kate Edgar. Though it is invidious to single out names, I must single out the first and the last of these: Colin Haycraft of Duckworth, whose faith in me, and *maieuticĕ techně*, allowed the original edition to be brought forth in 1973; and Kate Edgar, who has helped bring to birth this present, greatly enlarged edition.

In the second edition I made two very special acknowledgements – to W. H. Auden and A. R. Luria, who were mentors, friends, and 'awakeners' to me. I now omit these, but dedicate *Awakenings*, in gratitude and love, to the memory of these two men.





Rose R.: entranced, awakened, and blocked.

PREFACE TO THE ORIGINAL EDITION

The theme of this book is the lives and reactions of certain patients in a unique situation – and the implications which these hold out for medicine and science. These patients are among the few survivors of the great sleeping-sickness epidemic *(encephalitis lethargica)* of fifty years ago, and their reactions are those brought about by a remarkable new 'awakening' drug (laevodihydroxyphenylalanine, or L-DOPA). The lives and responses of these patients, which have no real precedent in the entire history of medicine, are presented in the form of extended case-histories or biographies: these form the major part of the book. Preceding these case-histories are introductory remarks on the nature of their illnesses, the sort of lives they have led since first being taken ill, and something about the drug which has transformed their lives. Such a subject might seem to be of very special or limited interest, but this, I believe, is by no means the case. In the latter part of the book, I have tried to indicate some of the far-reaching implications which arise from the subject – implications which extend to the most general questions of health, disease, suffering, care, and the human condition in general.

In a book such as this – about living people – a difficult, perhaps insuperable, problem arises: that of conveying detailed information without betraying professional and personal confidence. I have had to change the names of my patients, the name and location of the hospital where they live, and certain other circumstantial details. I have, however, tried to preserve what is important and essential – the real and full *presence* of the patients themselves, the 'feeling' of their lives, their characters, their illnesses, their responses – the essential qualities of their strange situation.

The general style of this book – with its alternation of narrative and reflection, its proliferation of images and

metaphors, its remarks, repetitions, asides, and footnotes – is one which I have been impelled towards by the very nature of the subject-matter. My aim is not to make a system, or to see patients as systems, but to picture a world, a variety of worlds – the landscapes of being in which these patients reside. And the picturing of worlds requires not a static and systematic formulation, but an active exploration of images and views, a continual jumping-about and imaginative *movement*. The stylistic (and epistemological) problems encountered have been precisely those described by Wittgenstein in the Preface to *Philosophical Investigations* when he spoke of the necessity of depicting landscapes (thoughtscapes) by images and 'remarks':

> … This was, of course, connected with the very nature of the investigation. For this compels us to travel over a wide field of thought criss-cross in every direction. The … remarks in this book are, as it were, a number of sketches of landscapes which were made in the course of these long and involved journeyings. The same or almost the same points were always being approached from different directions, and new sketches made … Thus this book is really only an album.

Running throughout the book is a metaphysical theme – the notion that it is insufficient to consider disease in purely mechanical or chemical terms; that it must be considered equally in biological or metaphysical terms, i.e. in terms of organization and design. In my first book, *Migraine*, I suggested the necessity of such a *double* approach, and in the present work I develop this theme in much greater detail. Such a notion is far from new – it was understood very clearly in classical medicine. In present-day medicine, by contrast, there is an almost exclusively technical or mechanical emphasis, which has led to immense advances, but also to intellectual regression, and a lack of proper attention to the full needs and feelings of patients. This book represents an attempt to regain and restore this metaphysical attention.

I have found the writing unexpectedly difficult, although its ideas and intentions are simple and straightforward. But one cannot go straight forward unless the way is clear, and the way is *allowed.* One struggles to gain the right perspective, focus, and tone – and then, one loses it, all unawares. One must continually fight to regain it, to hold accurate awareness. I cannot better express the problems which have challenged me, and which my readers must challenge, than in the splendid words of Maynard Keynes in the Preface to his *General Theory*:

> The composition of this book has been for the author a long struggle of escape, and so must the reading of it be for most readers if the author's assault upon them is to be successful – a struggle of escape from habitual modes of thought and expression. The ideas which are here expressed so laboriously are extremely simple and should be obvious. The difficulty lies, not in the new ideas, but in escaping from the old ones, which ramify, for those brought up as most of us have been, into every corner of our minds.

Force of habit, and resistance to change – so great in all realms of thought – reaches its maximum in medicine, in the study of our most complex sufferings and disorders of being; for we are here compelled to scrutinize the deepest, darkest, and most fearful parts of ourselves, the parts we all strive to deny or not-see. The thoughts which are most difficult to grasp or express are those which touch on this forbidden region and re-awaken in us our strongest denials and our most profound intuitions.

O.W.S.

New York
February 1973

PREFACE TO THE 1990 EDITION

Awakenings has been through several editions and formats since its original appearance in 1973. There have been, over the years, all sorts of additions, subtractions, revisions, and other changes, which have sometimes been confusing to bibliographers and readers. The brief publishing history which follows may also help to trace the evolution of the present edition.

Awakenings was first published in 1973, by Duckworth, in England. The first U.S. edition was published by Doubleday in 1974. This included a little additional material: a dozen or so extra footnotes, and a short follow-up on Rolando P. (who had died when the U.K. edition was in press).

A paperback edition was brought out in 1976 by Penguin Books in England, and by Random House (Vintage Books) in the United States. This contained a huge additional mass of footnotes, some with the length and format of miniature essays, and amounting *in toto* to a third the length of the book. (These had been written during a period of enforced immobilization, in the autumn of 1974, when I was a patient myself – the period described in *A Leg to Stand On*.)

In the third edition, published in 1982 by Pan Books in England and in the United States the following year by Dutton, I added, in the form of an epilogue, detailed follow-ups on all the patients (by this time, I had seen nearly 200 patients with post-encephalitic syndromes, most of whom had been maintained on L-DOPA for eleven or twelve years), and a sort of meditation on the general nature of health, sickness, music, etc., as well as the specifics of L-DOPA and Parkinsonism. I also added an appendix on some new EEG observations I had been able to make with our patients. Still other observations and thoughts I put in (my favourite format of) footnotes – though I also acceded to a publisher's request

that I remove all footnotes as such, incorporating them in the text wherever possible, and relegating what remained (often much shortened) to endnotes at the end of the book. Some 20,000 words of footnote material were entirely removed. (In 1987, in a new U.S. hardback published by Summit Books, I added a massive new foreword, otherwise keeping the book the same.) This 1982–3 edition was altogether neater, it was felt, than the 1976 one, but (to my mind, and many others) impoverished by the omission of so much material.

The need to correct this impoverishment, and restore the missing footnotes, coupled with the need to add a good deal more new material, has moved me to recast *Awakenings* once again, and rather radically, for this new 1990 edition. I have restored to its original form the most important part of the book – the *text* – relegating all additional and new material to footnotes and appendices. I have not, I should add, restored *all* the footnotes of the 1976 edition; some I felt constrained to shorten or remove. I cannot help feeling a sense of loss here, and a wondering whether (to paraphrase Gibbon) I may not have eradicated some choice flowers, some flowers of fancy, along with the weeds. I have also moved some of the longest 1976 footnotes (on the history of sleeping-sickness and on Parkinsonian space and time) to new appendices. I have not been able to resist adding a few further footnotes (but there are no more than a handful of these) and three newly-written appendices. The new material added has reference to the last surviving post-encephalitic patients (both in the United States and the United Kingdom); the remarkable advances in our understanding and treatment of Parkinsonism in the last six or seven years; some new theoretical formulations which have only emerged for me in the past few months; and finally, the striking dramatic and cinematic adaptations of *Awakenings* which have been created and shown in the last eight years, culminating in the feature film of *Awakenings* this year.

There are special difficulties in updating a book – at least a highly personal book composed largely of observation and reflection, of consciousness – for the subject is always evolving in one's mind. There may be formulations one no

longer adheres to or believes in, formulations which are obsolete, in a way; and yet these formulations – some perhaps extravagant, some seemingly abortive, but others genuinely precursory and embryonic – have formed the path by which one arrived at one's present position. Therefore, although there are formulations in *Awakenings* I no longer agree with, I have left them, out of fidelity to the process by which such a book comes into being. And, by the same token, who knows what visions and revisions the 1990s have in store? I still see Parkinsonian patients with a sense of complete wonder, a sense that I have only touched the surface of an infinite condition, a sense that there may be wholly different ways of viewing it.

It is now 21 years since my patients' awakenings, and 17 years since this book was first published; yet, it seems to me, the subject is inexhaustible – medically, humanly, theoretically, dramatically. It is this which demands new additions and editions, and which keeps the subject for me – and, I trust, my readers – evergreen and alive.

O.W.S.

New York
March 1990

FOREWORD TO THE 1990 EDITION

Twenty-four years ago I entered the wards of Mount Carmel and met the remarkable post-encephalitic patients who had been immured there since the great *encephalitis lethargica* (sleeping-sickness) epidemic just after the First World War. Von Economo, who first described the *encephalitis lethargica* half a century before, had spoken of the most affected patients as 'extinct volcanoes.' In the spring of 1969, in a way which he could not have imagined, which no one could have imagined or foreseen, these 'extinct volcanoes' erupted into life. The placid atmosphere of Mount Carmel was transformed – occurring before us was a cataclysm of almost geological proportions, the explosive 'awakening,' the 'quickening,' of eighty or more patients who had long been regarded, and regarded themselves, as effectively dead. I cannot think back on this time without profound emotion – it was the most significant and extraordinary in my life, no less than in the lives of our patients. All of us at Mount Carmel were caught up with the emotion, the excitement, and with something akin to enchantment, even awe.

It was not a purely 'medical' excitement, any more than these awakenings were a purely medical event. There was a tremendous *human* (even allegorical) excitement at seeing the 'dead' awaken again – it was at this point that I conceived the title *Awakenings*, taken from Ibsen's *When We Dead Awaken* – at seeing lives which one had thought irremediably blighted suddenly bloom into a wonderful renewal, at seeing individuals in all their vitality and richness emerge from the almost cadaveric state where they had been frozen and hidden for decades. We had had inklings of the vivid personalities so long immured – but the full reality of these only emerged, indeed burst upon us, with our patients' awakenings.

I was exceedingly lucky to encounter such patients at such

a time, in such working conditions. But they were not the only post-encephalitic patients in the world – there were, in the late '60s, still many thousands, some in large groups, in institutions all over the world. There was no major country *without* its complement of post-encephalitics. And yet *Awakenings* is the only existing account of such patients – their decades-long 'sleep' and, then, their dramatic 'awakening' in 1969.

I found this exceedingly peculiar at the time: why, I thought, were there not other accounts of what must be happening all over the world? Why, for example, was there not an 'Awakenings' from Philadelphia, where I knew of a group of patients not so dissimilar to my own? Why not from London, where the Highlands Hospital housed the largest post-encephalitic colony in England?[1] Or from Paris or Vienna, where the disease first struck?

There is no single answer to this; there were many things that mitigated against the sort of description, the 'biographic' approach, of *Awakenings*.

One factor that made *Awakenings* possible had to do with the nature of the *situation*. Mount Carmel is a chronic hospital, an asylum; and physicians in general avoid such hospitals, or visit them briefly, and leave as soon as they can. This was not always the case: in the last century, Charcot virtually lived in the Salpětrière, and Hughlings-Jackson at the West Riding Asylum – the founders of neurology realised well that it was only in such hospitals that the depths and details of the profounder disorders could be explored and worked out. As a resident I myself had never been to a chronic hospital, and though I had seen a number of patients with post-encephalitic Parkinsonism and other problems in outpatient clinics, I had no idea how profound and strange the effects of post-encephalitic disease might be. I found coming to Mount Carmel, in 1966, a revelation. It was my first encounter with disease of a depth I had never seen, read of, or heard of, before. The medical literature on the sleeping-sickness had virtually come to a stop in 1935, so that the profounder forms of it, occurring later, had never

been described. I would not have imagined it *possible* for such patients to exist; or, if they existed, to remain undescribed. For physicians do not go, and reports do not emerge, from the 'lower reaches,' these abysses of affliction, which are now (so to speak) beneath the notice of Medicine. Few doctors ever entered the halls and back wards of chronic hospitals and asylums, and few had the patience to listen and look, to penetrate the physiologies and predicaments of these increasingly inaccessible patients.

The 'other' side, the good side, of chronic hospitals is that what staff they have may work and live in them for decades, may become extraordinarily close to their charges, the patients, get to know and love them, recognize, respect them, *as people*. So when I came to Mount Carmel I did not just encounter 'eighty cases of post-encephalitic disease,' but eighty individuals, whose inner lives and total being was (to a considerable extent) known to the staff, known in the vivid, concrete knowing of relationship, not the pallid, abstract knowing of medical knowledge. Coming to this community – a community of patients, but also of patients and staff – I found myself encountering the patients as individuals, whom I could less and less reduce to statistics or lists of symptoms.

And, of course, this was a unique *time* for the patients, and for all of us. It had been established in the late 1950s that the Parkinsonian brain was lacking in the transmitter dopamine, and that it might therefore be 'normalised' if the level of dopamine could be raised. But attempts to do this, by giving L-DOPA (a precursor of dopamine) in milligram quantities, had failed persistently – until Dr George Cotzias, with great audacity, gave a group of patients L-DOPA in doses of a *thousand times* greater than had ever been used. With the publication of Cotzias's results in February 1967, the outlook for Parkinsonian patients was changed at a stroke: a sudden, unbelievable hope appeared – that patients hitherto able to look forward only to miserable and increasing disability might be (if not cured) transformed by the new drug. Life opened out once again, in imagination, for all our patients. For the first time in forty years they could believe in a future.

The atmosphere from this time on was electric with excitement. One of the patients, Leonard L., when he heard of L-DOPA, rapped on his letterboard with mixed enthusiasm and irony, 'Dopamine is Resurrectamine. Cotzias is the Chemical Messiah.'

Yet it was not L-DOPA, or what it offered, which was so exciting for me when I first came as a young doctor, a year out of residency, to Mount Carmel. What excited me then was the spectacle of a disease that was never the same in two patients, a disease that could take any possible form – one rightly called a 'phantasmagoria' by those who first studied it. ('There is nothing in the literature of medicine,' wrote McKenzie in 1927, 'to compare with the phantasmagoria of disorder manifested in the course of this strange malady.') At this level of the fantastic, the phantasmagoric, the encephalitis was enthralling. Much more fundamentally, it was, by virtue of the enormous range of disturbances occurring at every level of the nervous system, a disorder that could show, far better than any other, how the nervous system was organised, how brain and behavior, at their more primitive levels, worked. The biologist, the naturalist, in me was enthralled by all this – and led me to start gathering data at this time for a book on primitive, subcortical behaviours and controls.

But then, over and above the disorder, and its direct effects, were all the responses of the patients to their sickness – so what confronted one, what one studied, was not just disease or physiology, but *people*, struggling to adapt and survive. This too was clearly realised by the early observers, above all Ivy McKenzie: 'The physician is concerned (unlike the naturalist) … with a single organism, the human subject, striving to preserve its identity in adverse circumstances.' In perceiving this, I became something more than a naturalist (without, however, ceasing to be one). There evolved a new concern, a new bond: that of commitment to the patients, the individuals under my care. Through them I would explore what it was like to be human, to *stay* human, in the face of unimaginable adversities and threats. Thus, while continually

monitoring their organic nature – their complex, ever-changing pathophysiologies and biologies – my central study and concern became *identity* – their struggle to maintain identity – to observe this, to assist this, and, finally, to describe this. All this was at the junction of biology and biography.

This sense of the dynamics of illness and life, of the organism or subject striving to survive, sometimes under the strangest and darkest circumstances, was not a viewpoint which had been emphasised when I was a student or resident, nor was it one I found in the current medical literature. But when I saw these post-encephalitic patients, it was clearly and overwhelmingly true – indeed, it was the only way in which I *could* view them. Thus what had been dismissed disparagingly by most of my colleagues ('chronic hospitals – you'll never see anything interesting in *those* places') revealed itself as the complete opposite: an ideal situation in which to observe, to care, to explore. *Awakenings* would have been written, I think, even if there had not been any 'awakening': it would then have been *People of the Abyss* (or *Cinquante Ans de Sommeil*, as the French edition has it), a delineation of the stillness and darkness of these arrested and frozen lives, and of the courage and humour with which patients, nonetheless, faced life.

The intensity of feeling for these patients, and equally of intellectual interest and curiosity about them, bound us together as a community at Mount Carmel; and this intensity rose to a peak in 1969, the actual year of the patients' 'awakenings.' In the spring of that year, I moved to an apartment a hundred yards from the hospital and would sometimes spend twelve or fifteen hours a day with our patients. I was with the patients constantly – I grudged the hours of sleep – observing them, talking with them, getting them to keep notebooks, and keeping voluminous notes myself, thousands of words each day. And if I had a pen in one hand, I had a camera in the other: I was seeing such things as had never, perhaps, been seen before – and which, in all probability, would never be seen again; it was my duty, and my joy, to record and bear witness. Many others also

dedicated themselves, spent countless hours in the hospital. All of us involved with the patients – nurses, social workers, therapists of every sort – were in constant communication: talking to each other excitedly in the passage, phoning each other on weekends and at night, constantly exchanging new experiences and ideas. The excitement, the enthusiasm, of that year was remarkable; *this*, it seems to me, was an essential part of the 'Awakenings' experience.

And yet, at the start, I scarcely knew what to expect. I had read the half-dozen reports on L-DOPA published in 1967 and '68, but felt my own patients to be very different. They did not have ordinary Parkinson's disease (like the other patients reported), but a post-encephalitic disorder of far greater complexity, severity, and strangeness. How would *these* patients, with their so-different disease, react? I felt I had to be cautious – almost exaggeratedly so. When, early in 1969, I embarked on the work which was later to become *Awakenings*, I conceived it in quite limited and narrowly 'scientific' terms – as a 90-day, double-blind trial of L-DOPA in a large group of patients who had become institutionalised after having encephalitis. L-DOPA was considered an experimental drug at this time, and I needed to get (from the Food and Drug Administration) a special investigator's licence to use it. It was a condition of such licences that one use 'orthodox' methods, including a double-blind trial, coupled with presentation of results in quantitative form.

But it became obvious within a month or less that the original format would have to be abandoned. The effects of L-DOPA in these patients was decisive – spectacular; while, as I could infer from the precise fifty percent failure rate, there was no significant placebo effect whatever. I could no longer, in good conscience, continue the placebo but had to try L-DOPA in every patient; and I could no longer think of giving it for 90 days and then stopping – this would have been like stopping the very air that they breathed. Thus what was originally conceived as a limited 90-day experiment was transformed instead into an historical experience: a story, in effect, of life for these patients as it had been before L-DOPA,

and as it was changed, and as it was to become, after starting treatment with L-DOPA.

Thus I was impelled, willy-nilly, to a presentation of case-histories or biographies, for no 'orthodox' presentation, in terms of numbers, series, grading effects, etc., could have conveyed the historical reality of the experience. In August 1969, then, I wrote the first nine case-histories, or 'stories,' of *Awakenings*.

The same impulse, the same sense that one had to convey stories and phenomena – the drama of stories, the delight of phenomena – led me to write a number of letters to the editor, which I despatched to the *Lancet* and the *British Medical Journal* early the next year. I enjoyed writing these letters, and as far as I could gather, readers of these journals enjoyed reading them too. There was something about their format and style that allowed me to convey the wonder of the clinical experience, in a way that would have been quite impossible in a medical article.

I now decided to present my overall observations, and my general conclusions, while still adhering to an epistolary format. My earlier letters to the *Lancet* had been anecdotal (and everyone loves anecdotes); I had not yet attempted any general formulations. My first experiences, the patients' first responses, in the summer of '69, had been happy ones; there had been an astonishing, festive 'awakening,' at the time – but then all of my patients ran into trouble and tribulation. I observed, at this time, not only specific 'side-effects' of L-DOPA, but certain *general* patterns of trouble – sudden and unpredictable fluctuations of response, the rapid development of oscillations, the development of extreme sensitivity to L-DOPA, and, finally, the absolute impossibility of matching dose and effect – all of which I found dismaying in the extreme. I tried altering the dose of L-DOPA, but this no longer worked – the 'system' now seemed to have a dynamic of its own.

In the summer of 1970, then, in a letter to the *Journal of the American Medical Association*, I reported these findings, describing the total effects of L-DOPA in 60 patients whom I had

maintained on it for a year. *All* of these, I noted, had done well at first; but all of them, sooner or later, had escaped from control, had entered complex, sometimes bizarre, and unpredictable states. These could not, I indicated, be seen as 'side-effects,' but had to be seen as integral parts of an evolving whole. Ordinary considerations and policies, I stressed, sooner or later ceased to work. There was a need for a deeper, more radical understanding.

My *JAMA* letter caused a furor among some of my colleagues. (See Sacks et al., 1970c and letters appearing in the December 1970 *JAMA*.) I was astonished and shocked by the storm that blew up; and, in particular, by the tone of some of the letters. Some colleagues insisted that such effects 'never' occurred; others that, even if they did, the matter should be kept quiet, lest it disturb 'the atmosphere of therapeutic optimism needed for the maximal efficiency of L-DOPA.' It was even thought, absurdly, that I was 'against' L-DOPA – but it was not L-DOPA but reductionism I was against. I invited my colleagues to come to Mount Carmel, to see for themselves the reality of what I had reported; none of them took up my invitation. I had not properly realised, until this time, the power of *wish* to distort and deny – and its prevalence in this complex situation, where the enthusiasm of doctors, and the distress of patients, might lie in unconscious collusion, equally concerned to wish away an unpalatable truth. The situation had similarities to what had occurred twenty years before, when cortisone was clothed with unlimited promise; and one could only hope that with the passage of time, and the accumulation of undeniable experience, a sense of reality would triumph over wish.

Was my letter too condensed – or simply confusing? Did I need to put things in the form of extended articles? With much labour (because it went against the grain, so to speak), I put everything I could in an orthodox or conventional format – papers full of statistics and figures and tables and graphs – and submitted these to various medical and neurological journals. To my amazement and chagrin, none was accepted – some of them, indeed, elicited vehemently

censorious, even violent, rejections, as if there were something intolerable in what I had written. This confirmed my feeling that a deep nerve had been struck, that I had somehow elicited not just a medical, but a sort of epistemological, anxiety – and rage.[2]

I had not only cast doubt on what had appeared at first to be the extremely simple matter of giving a drug and being in control of its effects; I had cast doubt on predictability itself. I had (perhaps without fully realising it myself) hinted at something bizarre, a contradiction of ordinary ways of thinking, and of the ordinary, accepted picture of the world. A spectre of extreme oddness, of radical contingency, had come up – and all this was disquieting, confounding, in the extreme ('These things are so bizarre that I cannot bear to contemplate them' – Poincaré).

And so, by mid-1970 I was brought to a halt, at least so far as any publication was concerned. The work continued, full of excitement, unabated, and I accumulated (I dared to think) an absolute treasure of observations and of hypotheses and reflections associated with them, but I had no idea what to do with them. I knew that I had been given the rarest of opportunities; I knew that I had something valuable to say; but I saw no way of saying it, of being faithful to my experiences, without forfeiting medical 'publishability' or acceptance among my colleagues. This was a time of great bewilderment and frustration, considerable anger, and sometimes despair.

This impasse was broken in September of 1972, when the editor of *The Listener* invited me to write an article on my experiences. This was going to be my opportunity. Instead of the censorious rejections I was used to, I was actually being invited to write, being offered a chance to publish, fully and freely, what had been accumulating and building up, dammed up, for so long. I wrote 'The Great Awakening' at a single sitting – neither I nor the editor altered a single word – and it was published the following month. Here, with a sense of great liberation from the constraints of 'medicalising' and medical jargon, I described the wonderful panorama of

phenomena I had seen in my patients. I described the raptures of their 'awakenings,' I described the torments that so often followed; but above all, it was *phenomena* which I was concerned to describe, with a neutral and phenomenological (rather than a therapeutic, or 'medical') eye.

But the picture, the theory, implied by the phenomena: this seemed to me to be of a revolutionary sort – 'a new neurophysiology,' as I wrote, 'of a quantum-relativistic sort.' These were bold words indeed; they excited me, and others – although I soon came to think that I had said too much, and too little. That there was *something*, assuredly, very strange going on – not quantality, not relativity, but something much commoner, yet stranger. I could not imagine what this was, in 1972, though it haunted me when I came to complete *Awakenings*, and rippled through it constantly, evasively, as half-tantalising metaphors.

The article in *The Listener* was followed (in contrast to the hateful *JAMA* experience of two years earlier) by a wave of interest, and a great number of letters, an exciting correspondence which lasted several weeks. This response put an end to my long years of frustration and obstruction and gave me a decisive encouragement and affirmation. I picked up my long discarded case-histories of 1969, added eleven more, and in two weeks completed *Awakenings*. The case-histories were the easiest to write; they wrote themselves, they stemmed straight from experience, and I have always regarded them with especial affection as the true and unassailable centre of *Awakenings*. The rest is disputable, the stories are so.

But the 1973 publication of *Awakenings*, while attracting much general attention, met the same cold reception from the profession as my articles had done earlier. There was not a single medical notice or review, only a disapproving or uncomprehending silence. There was one brave editor (of the *British Clinical Journal*) who spoke out on this, making *Awakenings* his 'editor's choice' for 1973, but commenting on 'the strange mutism' of the profession towards it.

I was devastated at this medical 'mutism,' but at the same

time reassured and encouraged by the reaction of A. R. Luria. Luria himself, after a lifetime of minute neuropsychological observations, had himself published two extraordinary, almost novelistic case-histories – *The Mind of a Mnemonist* (in 1968) and *The Man with a Shattered World* (1972). To my intense pleasure, in the strange medical silence which attended the publication of *Awakenings*, I received a letter, two letters, from him; in the first, he spoke of his own 'biographic' books and approaches:

> Frankly said, I myself like very much the type of 'biographical' study, such as Sherashevsky [the Mnemonist] and Zazetski [the man with the 'shattered world'] … firstly because it is a kind of 'Romantic Science' which I wanted to introduce, partly because I am strongly *against* a formal statistical approach and *for* a qualitative study of personality, *for* every attempt to find *factors* underlying the structure of personality. [Letter of July 19, 1973, emphasis in original]

And in the second, he spoke of *Awakenings*:

> I received *Awakenings* and have read it at once with great delight. I was ever conscious and sure that a good clinical description of cases plays a leading role in medicine, especially in Neurology and Psychiatry. Unfortunately, the ability to describe which was so common to the great Neurologists and Psychiatrists of the 19th century [is] lost now, perhaps because of the basic mistake that mechanical and electrical devices can replace the study of personality … Your excellent book shows, that the important tradition of clinical case studies can be revived and with a great success. [Letter of July 25, 1973]

He then went on to ask me some specific questions, above all expressing his fascination that L-DOPA should be so various and unstable in effect.[3]

* * *

I had admired Luria infinitely since my medical school days, and before. When I heard him lecture in London in 1959, I was overwhelmed by his combination of intellectual power and human warmth – I had often encountered these separately, but I had not too often encountered them *together* – and it was exactly this combination which so pleased me in his work, and which made it such an antidote to certain trends in medical writing, which attempted to delete both subjectivity and reflection. Luria's early works had been, sometimes, a little stilted in character, but they grew in intellectual warmth, in wholeness, as he grew older, culminating in his two late works, *The Mind of a Mnemonist* and *The Man with a Shattered World*. I do not know how much either of these works influenced me, but they certainly emboldened me, and made it easier to write and publish *Awakenings*.

Luria often said that he had to write two sorts of books, wholly different but wholly complementary: 'classical,' analytic texts (like *Higher Cortical Functions in Man*) and 'romantic,' biographical books (like *The Mind of a Mnemonist* and *The Man with a Shattered World*). I was also conscious of this double need, and found there were always *two* books, potentially, demanded by every clinical experience: one more purely 'medical' or 'classical' – an objective description of disorders, mechanisms, syndromes; the other more existential and personal – an empathic entering into patients' experiences and worlds. Two such books dawned in me when I first saw our post-encephalitic patients: *Compulsion and Constraint* (a study of subcortical disorders and mechanisms) and *People of the Abyss* (a novelish, Jack Londonish book). They only came together, finally, in 1969 – to a book which tried to be *both* classical and romantic; to place itself at the intersection of biology and biography; to combine, as best it could, the modes of paradigm and art.

But *no* model, finally, seemed to suit my requirements – for what I was seeing, and what I needed to convey, was neither purely classical nor purely romantic, but seemed to move

into the profound realm of allegory or myth. Even my title, *Awakenings*, had a double meaning, partly literal, partly in the mode of metaphor or myth.

The elaborate case-history, the 'romantic' style, with its endeavour to present a whole life, the repercussions of a disease, in all its richness, had fallen very much out of favour by the middle of the century – and this, perhaps, was one reason for the 'strange mutism' of the profession when *Awakenings* was first published in 1973. But as the seventies progressed, this antipathy to case-history diminished – it even became possible (though difficult) to publish case-histories in the medical literature. With this thawing of atmosphere, there was a renewed sense that complex neural and psychic functions (and their disorders) *required* detailed and non-reductive narratives for their explication and understanding.[4]

At the same time, the unpredictable responses to L-DOPA I saw with my patients in 1969 – their sudden fluctuations and oscillations, their extraordinary 'sensitization' to L-DOPA, to *everything* – were now being seen, increasingly, by everyone. Post-encephalitic patients, it became clear, might show these bizarre reactions within weeks, sometimes days – whereas 'ordinary' Parkinsonian patients, with their more stable nervous systems, might not show them for several years. Yet, sooner or later, *all* patients maintained on L-DOPA started to show these strange, unstable states – and with the FDA approval of L-DOPA in 1970, their numbers mounted, finally to millions. And now, everybody found the same: the central promise of L-DOPA was confirmed, a million-fold – but so too was the central threat, the certainty of 'side-effects' or 'tribulations,' sooner or later.

Thus what had been surprising or intolerable when I first published *Awakenings* was – by the time the third edition was published in 1982 – confirmed for all my colleagues by their own, undeniable experience. The optimistic and irrational mood of the early days of L-DOPA had changed to something

more sober and realistic. This mood, well established by 1982, made the new edition of *Awakenings* acceptable, and even a classic, to my medical colleagues, where the original had been unacceptable nine years before.

It is the imagination of other people's worlds – worlds almost inconceivably strange, yet inhabited by people just like ourselves, people, indeed, who might *be* ourselves – that forms the centre of *Awakenings*. Other worlds, other lives, even though so different from our own, have the power of arousing the sympathetic imagination, of awakening an intense and often creative resonance in others. We may never have seen a Rose R., but once we have read of her we see the world differently – we can imagine her world, with a sort of awe, and with this our world is suddenly enlarged. A wonderful example of such a creative response was given by Harold Pinter in his play, *A Kind of Alaska*; this is Pinter's world, the landscape of his unique gifts and sensibility, but it is also Rose R.'s world, and the world of *Awakenings*. Pinter's play has been followed by several adaptations of *Awakenings* for stage and screen; each of these has drawn on different aspects of the book. Every reader will bring to *Awakenings* his own imagination and sensibilities, and will find, if he lets himself, his world strangely deepened, imbued with a new depth of tenderness and perhaps horror. For these patients, while seemingly so extraordinary, so 'special,' have in them something of the universal, and can call to everyone, awaken everyone, as they called to and awakened me.

I hesitated very greatly in regard to the original publication of our patients' 'story' and their lives. But they themselves encouraged me, and said to me from the first, 'Tell our story – or it will never be known.'

A few of the patients are still alive – we have known each other for twenty-four years now. But those who have died are in some sense not dead – their unclosed charts, their letters, still face me as I write. They still live, for me, in some very personal way. They were not only patients but teachers and friends, and the years I spent with them were the most

significant of my life. I want something of their lives, their presence, to be preserved and live for others, as exemplars of human predicament and survival. This is the testimony, the only testimony, of a unique event – but one which may become an allegory for us all.

O.W.S.

New York
March 1990

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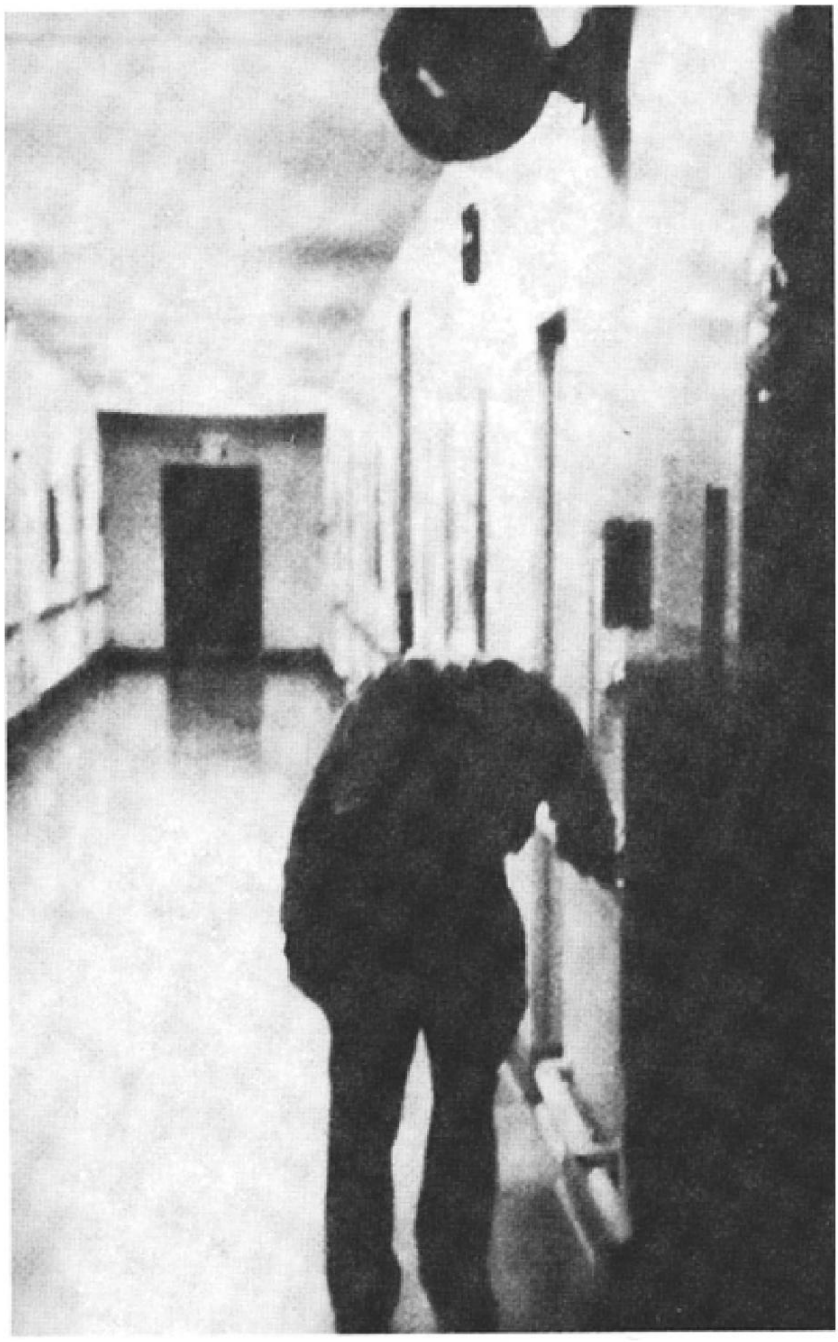
[1] There was a short, statistical paper by Calne et al. (1969), describing a six-week trial of L-DOPA in some of the Highlands patients, but there were no *biographical* accounts of 'awakenings' in these, or any other, patients.

[2] Five years later, it happened that one of the neurologists who had taken such exception to my letter in *JAMA* – he had said that my observations were beyond credibility – found himself chairing a meeting at which the documentary film of *Awakenings* was being shown. There is a particular point in the film at which various bizarre 'side-effects' and instabilities of drug reaction are shown in dizzying array, and I was fascinated to observe my colleague's reactions here. First, he stared amazed, and his mouth dropped open; it was as if he were seeing such things for the first time, and his reaction was one of innocent and almost childlike wonder. Then he flushed a dark and angry crimson – whether with embarrassment or mortification, I could not tell; these were the very things he had dismissed as 'beyond credibility,' and now he was being forced to see them for himself. Then he developed a curious *tic*, a convulsive movement of the head which kept turning it away from the screen he could no longer bear to see. Then, finally, with great abruptness and violence, and muttering to himself, he burst out of his seat, in mid-film, and rushed out of the room. I found this behaviour extraordinary and instructive, for it showed how profound, how utterly overwhelming, reactions to the 'incredible' and 'intolerable' might be.

[3] He returned to this topic the following month, when he said that he had been fascinated by the case of Martha N., and the fact that she had responded to L-DOPA in six different ways: '*Why* was it different each time?' he asked, '*Why* could one not replay things again and again?' – questions I could not answer in 1973. It seemed to me typical of the genius of Luria

that he had at once homed in on one of the central mysteries and challenges of *Awakenings* – the various and unrepeatable and unpredictable character of patients' responses – and been fascinated by this; whereas my neurological colleagues, by and large, had been frightened and dismayed by this, had tended to asseverate, 'It's not so, it's not so.'

[4] There has been a parallel movement in anthropology since 1970 – this had also been becoming meagre and mechanical – with a new, or renewed, insistence on what Clifford Geertz has dubbed 'thick' description.



Seymour L. would often be frozen for hours like this in the corridor.

Prologue

PARKINSON'S DISEASE AND PARKINSONISM

In 1817, Dr James Parkinson – a London physician – published his famous *Essay on the Shaking Palsy*, in which he portrayed, with a vividness and insight that have never been surpassed, the common, important, and singular condition we now know as Parkinson's disease.

Isolated symptoms and features of Parkinson's disease – the characteristic shaking or tremor, and the characteristic hurrying or festination of gait and speech – had been described by physicians back to the time of Galen. Detailed descriptions had also appeared in the non-medical literature – as in Aubrey's description of Hobbes's 'Shaking Palsy.' But it was Parkinson who first saw every feature and aspect of the illness as a whole, and who presented it as a distinctive human condition or *form of behaviour*.[1]

Between 1860 and 1890, working amid the large population of chronically ill patients at the Salpětrière in Paris, Charcot filled in the outline which Parkinson had drawn. In addition to his rich and detailed characterizations of the illness, Charcot perceived the important relations and affinities which existed between the symptoms of Parkinson's disease and those of depression, catatonia, and hysteria: indeed, it was partly in view of these striking relationships that Charcot called Parkinsonism 'a neurosis.'

In the nineteenth century, Parkinsonism was almost never seen before the age of fifty, and was usually considered to be a reflection of a degenerative process or defect of nutrition in certain 'weak' or vulnerable cells; since this degeneration could not actually be demonstrated at the time, and since its cause was unknown, Parkinson's disease was termed an idiosyncrasy or 'idiopathy.' In the first quarter of this century, with the advent of the great sleeping-sickness epidemic *(encephalitis lethargica)*, a 'new' sort of

Parkinsonism appeared, which had a clear and specific cause: this encephalitic or post-encephalitic Parkinsonism,[2] unlike the idiopathic illness, could affect people of any age, and could assume a form and a severity much graver and more dramatic than ever occurred in the idiopathic illness. A third great cause of Parkinsonism has been seen only in the last twenty years, and is an unintended (and usually transient) consequence or 'side-effect' of the use of phenothiazide and butyrophenone drugs – the so-called 'major tranquillizers.' It is said that in the United States alone there are two million people with Parkinsonism: a million with idiopathic Parkinsonism or Parkinson's disease; a million with drug-induced Parkinsonism; and a few hundred or thousand patients with post-encephalitic Parkinsonism – the last survivors of the great epidemic. Other causes of Parkinsonism – coal-gas poisoning, manganese poisoning, syphilis, tumours, etc. – are excessively rare, and are scarcely likely to be seen in a lifetime of practice by the ordinary physician.

Parkinson's disease has been called the 'shaking palsy' (or its Latin equivalent – *paralysis agitans*) for some centuries. It is necessary to say at the outset that the shaking or tremor is by no means a constant symptom in Parkinsonism, is never an isolated symptom, and is often the least problem which faces the Parkinsonian patient. If tremor is present, it tends to occur at rest and to disappear with movement or the intention to move;[3] sometimes it is confined to the hand, and has a characteristic 'pill-rolling' quality or (in Gowers's words) a quality 'similar to that by which Orientals beat their small drums'; in other, and especially in post-encephalitic patients, tremor may be extremely violent, may affect any or every part of the body, and tends to be increased by effort, nervousness, or fatigue. The second commonly mentioned symptom of Parkinsonism, besides tremor, is stiffness or rigidity; this has a curious plastic quality – often compared to the bending of a lead pipe – and may be of intense severity.[4] It must be stressed, however, that neither tremor nor rigidity is an essential feature of Parkinsonism; they may both be completely absent, especially in the post-encephalitic forms

of disease with which we shall especially be concerned in this book. The essential features of Parkinsonism, which occur in every patient, and which reach their extremest intensity in post-encephalitic forms of disease, relate to disorders of movement and 'push.'

The first qualities of Parkinsonism which were ever described were those of *festination* (hurry) and *pulsion* (push). Festination consists of an acceleration (and with this, an abbreviation) of steps, movements, words, or even thoughts – it conveys a sense of impatience, impetuosity, and alacrity, as if the patient were very pressed for time; and in some patients it goes along with a *feeling* of urgency and impatience, although others, as it were, find themselves hurried against their will.[5] The character of movements associated with festination or pulsion are those of quickness, abruptness, and brevity. These symptoms, and the peculiar 'motor impatience' (akathisia) which often goes along with them, were given full weight by the older authors: thus Charcot speaks of the 'cruel restlessness' suffered by many of his patients, and Gowers of the 'extreme restlessness … which necessitates … every few minutes some slight change of posture.' I stress these aspects – the alacrity and pressure and precipitation of movement – because they represent, so to speak, the less familiar 'other side' of Parkinsonism, Parkinsonism-on-the-boil, Parkinsonism in its expansile and explosive aspect, and as such have peculiar relevance to many of the 'side-effects' of L-DOPA which patients exhibit.

The opposite of these effects – a peculiar slowing and difficulty of movement – are more commonly stressed, and go by the general and rather uninformative name of 'akinesia.' There are many different forms of akinesia, but the form which is exactly antithetical to hurry or pulsion is one of active *retardation* or *resistance* which impedes movement, speech, and even thought, and may arrest it completely. Patients so affected find that as soon as they 'will' or intend or attempt a movement, a 'counter-will' or 'resistance' rises up to meet them. They find themselves embattled, and even

immobilized, in a form of physiological conflict – force against counter-force, will against counter-will, command against countermand. For such embattled patients, Charcot writes: 'There is no truce' – and Charcot sees the tremor, rigidity, and akinesia of such patients as the final, futile outcome of such states of inner struggle, and the tension and tiredness of which Parkinsonian patients so often complain as due to the pre-emption of their energies in such senseless inner battles. It is these states of push and constraint which one patient of mine (Leonard L.) would always call 'the goad and halter.'[6] The appearance of passivity or inertia is deceiving: an obstructive akinesia of this sort is in no sense an idle or restful state, but (to paraphrase de Quincey) '… no product of inertia, but … resulting from mighty and equal antagonisms, infinite activities, infinite repose.'[7]

In some patients, there is a different form of akinesia, which is not associated with a feeling of effort and struggle, but with one of continual repetition or perseveration: thus Gowers records the case of one patient whose limbs '… when raised remained so for several minutes, and then slowly fell' – a form of akinesia which he correctly compares to catalepsy; this is generally far more common and far more severe in patients with post-encephalitic forms of Parkinsonism.[8]

These characteristics – of impulsion, of resistance, and of perseveration – represent the active or positive characteristics of Parkinsonism. We will later have occasion to see that they are to some extent interchangeable, and thus that they represent different phases or forms or transformations of Parkinsonism. Parkinsonian patients also have 'negative' characteristics – if this is not a contradiction in terms. Thus some of them, Charcot particularly noted, would sit for hours not only motionless, but apparently without any impulse to move: they were, seemingly, content to do nothing, and they lacked the 'will' to enter upon or continue any course of activity, although they might move quite well if the stimulus or command or request to move came from another person – *from the outside*. Such patients

were said to have an absence of the will – or 'aboulia.'

Other aspects of such 'negative' disorder or deficiency in Parkinsonian patients relate to feelings of tiredness and lack of energy, and of certain 'dullness' – an impoverishment of feeling, libido, motive, and attention. To a greater or less degree, all Parkinsonian patients show alteration of 'go,' impetus, initiative, vitality, etc., closely akin to what may be experienced by patients in the throes of depression.[9]

Thus Parkinsonian patients suffer simultaneously (though in varying proportions) from a pathological absence and a pathological presence. The former cuts them off from the fluent and appropriate flow of normal movement (and – in severe cases – the flow of normal perception and thought), and is experienced as a 'weakness,' a tiredness, a deprivation, a destitution; the latter constitutes a preoccupation, an abnormal activity, a pathological organization, which, so to speak, distends or inflates their behaviour in a senseless, distressing, and disabling fashion. Patients can be thought of as *engorged* with Parkinsonism – with pathological excitement ('erethism') – as one may be engorged with pain or pleasure or rage or neurosis. The notion of Parkinsonism as exerting a pressure on the patient seems to be supported, above all, by the phenomenon of *kinesia paradoxa* which consists of a sudden and total (though transient) disappearance or deflation of Parkinsonism – a phenomenon seen most frequently and most dramatically in the most intensely Parkinsonian patients.[10]

It is scarcely imaginable that a profound deficiency can suddenly be made good, but it is easy to conceive that an intense pressure might suddenly be relieved, or an intense charge discharged. Such conceptions are always implicit, and sometimes explicit, in the thinking of Charcot, who goes on, indeed, to stress the close analogies which could exist between the different forms or 'phases' of Parkinsonism and those of neurosis: in particular Charcot clearly saw the formal similarity or analogy between the three clearly distinct yet interchangeable phases of Parkinsonism – the compliant-perseverative, the obstructive-resistive, and the

explosive-precipitate phases – with the plastic, rigid, and frenzied forms of catatonia and hysteria. These insights were reinforced during the 1920s, by observation of the extraordinary amalgamations of Parkinsonism with other disorders seen in the encephalitis epidemic. They were then completely 'forgotten,' or thrust out of the neurological consciousness. The effects of L-DOPA – as we shall see – compel us to reinstate and elaborate the forgotten analyses and analogies of Charcot and his contemporaries.

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[1] It is true, in a sense, that Parkinson had many 'predecessors' (Gaubius, Sauvages, de la Noë, and others) who had observed and classified various 'signs' of Parkinsonism. But there was a radical difference between Parkinson and these men – perhaps more radical than Parkinson himself allowed or admitted. Observers of Parkinsonism, before Parkinson himself, had been content to 'spot' various characteristics (in much the same way as one 'spots' trains or planes), and then to arrange these characteristics in classificatory schemes (somewhat as a butterfly-spotter, or would-be entomologist, might arrange his specimens according to colour and shape). Thus Parkinson's predecessors were entirely concerned with 'diagnosis' and 'nosology' – an arbitrary, pre-scientific diagnosis and nosology, based entirely on superficial characteristics and relationships: the Zodiacal charts of Sauvages and others represent a sort of pseudo-astronomy, first attempts to come to grips with the unknown. Parkinson's own initial observations were also made 'from the outside,' so to speak, from seeing Parkinsonians in the streets of London, inspecting their peculiarities of motion from a distance. But his observations were deeper than those of his predecessors, deeper-rooted and more deeply related. Parkinson resembles a *genuine* astronomer, and London the field of his astronomical observations, and at this stage, through his eyes, we see Parkinsonians as bodies-in-transit, moving like comets or stars. Soon, moreover, he came to recognize that certain stars form a *constellation*, that many seemingly unrelated phenomena form a definite and constant 'assemblage of symptoms.' He was the first to recognize this 'assemblage' as such, this constellation or syndrome we now call 'Parkinsonism.'

This was a clinical achievement of the first magnitude, and Parkinsonism was one of the first neurological syndromes to be recognized and defined. But Parkinson was not merely talented – he was a man of genius. He

perceived that the curious 'assemblage' he had noted was something more than a diagnostic syndrome – that it seemed to have a coherent inner logic and order of its own, that the constellation was a sort of *cosmos* … Sensing this, he now realized that inspection-at-a-distance, however acute, was insufficient if he wished to understand its nature; he realized it was necessary to meet actual patients, to engage them in clinical and dialogic encounter. With this he adopted an entirely different stance and concurrently with this a quite different language. He ceased to see Parkinsonians as remote objects in orbit, and saw them as patients and fellow human beings; he ceased to use diagnostic jargon, and used words indicative of *intention* and *action*; he ceased to see Parkinsonism as 'an assemblage of symptoms' and now thought of being-Parkinsonian as a strange form of *behaviour*, a peculiar and characteristic mode of Being-in-the-World. Thus Parkinson, compared to his predecessors, was a radical, a revolutionary, in two different ways: first in establishing a genuine empiricism – a science of 'facts' and their interrelations; second, in making a still more radical move in intellectual mid-course, by moving from an empirical to an existential position.

[2] The term 'post-encephalitic' is used to denote symptoms which have come on *following* an attack of *encephalitis lethargica*, and as a direct or indirect consequence of this. The onset of such symptoms may be delayed until many years after the original attack.

[3] There are many actors, surgeons, mechanics, and skilled manual workers who show severe Parkinsonian tremor at rest, but not a trace of this when they concentrate on their work or move into action.

[4] It was observed by Charcot, and is observed by many Parkinsonian patients themselves, that rigidity can be loosened to a remarkable degree if the patient is suspended in water or swimming (see below the cases of Hester Y., Rolando P., Cecil M., etc.). The same is also true, to some extent, of other forms of stiffness and 'clench' – spasticity, athetosis, torticollis, etc.

[5] Thus festination *('scelotyrbe festinans')* is portrayed by Gaubius in the eighteenth century: 'Cases occur in which the muscles, duly excited by the impulses of the will, do then, with an unbidden agility, and with an impetus not to be repressed, run before the unwilling mind.'

[6] Analogous concepts are used by William James, in his discussion of 'perversions' of will (*Principles*, 2, xxvi). The two basic perversions delineated by James are the 'obstructive' will and the 'explosive' will; when the former holds sway, the performance of normal actions is rendered

difficult or impossible; if the latter is dominant, abnormal actions are irrepressible. Although James uses these terms with reference to neurotic perversions of the will, they are equally applicable to what we must term Parkinsonian perversions of the will: Parkinsonism, like neurosis, is a *conative* disorder, and exhibits a formal analogy of conative structure.

[7] At this point we must introduce a fundamental theme which will re-appear and re-echo, in various guises, throughout this book. We have seen Parkinsonism as sudden starts and stops, as odd speedings and slowings. Our approach, our concepts, our terms have so far been of a purely mechanical or empirical type: we have seen Parkinsonians as bodies, but not yet as *beings* … if we are to achieve any understanding of *what it is like to be Parkinsonian*, of the actual nature of Parkinsonian existence (as opposed to the parameters of Parkinsonian motion), we must adopt a different and complementary approach and language.

We must come down from our position as 'objective observers,' and meet our patients face-to-face; we must meet them in a sympathetic and imaginative encounter: L-DOPA, often speeding too far, into a veritable tachyphrenia, with thoughts and associations almost too fast to follow. Again, there is not merely motor, but a perceptual inertia in Parkinsonism: a perspective drawing of a cube or a staircase, for example, which the normal mind perceives first this way and then that, in alternating perceptual configurations or hypotheses, may be absolutely frozen in one configuration for the Parkinsonian; it will unfreeze as he 'awakens' and may then be thrust, with the continuing stimulation of L-DOPA, in the opposite direction, with a near-delirium of perceptual hypotheses alternating many times a second.

[8] Arrest (akinesia) or profound slowing (bradykinesia) are equally evident in other spheres – they affect *every* aspect of life's stream, including the stream of consciousness. Thus, Parkinsonism itself is not 'purely' motor – there is, for example, in many akinetic patients, a corresponding 'stickiness' of mind or bradyphrenia, the thought stream as slow and sluggish as the motor stream. The thought stream, the stream of consciousness, speeds up in these patients with for it is only in the context of such a collaboration, a participation, a relation, that we can hope to learn anything about *how they are*. They can tell us, and show us, what it is like being Parkinsonian – they can tell us, but nobody else can.

Indeed we must go further, for if – as we have reason to suspect – our patients may be subject to experiences as strange as the motions they show,

they may need much help, a delicate and patient and imaginative collaboration, in order to formulate the almost-unformulable, in order to communicate the almost-incommunicable. We must be co-explorers in the uncanny realm of being-Parkinsonian, this land beyond the boundaries of common experience; but our quarry in this strange country will not be 'specimens,' data, or 'facts,' but images, similitudes, analogies, metaphors – whatever may assist to make the strange familiar, and to bring into the thinkable the previously unthinkable. What we are told, what we discover, will be couched in the mode of 'likeness' or 'as if,' for we are asking the patient to make *comparisons* – to compare being-Parkinsonian with that mode-of-being which we agree to call 'normal.'

All experience is hypothetical or conjectural, but its intensity and form vary a great deal: thus patients able to achieve some detachment, or patients only partially or intermittently affected, will describe their experiences in metaphorical terms; whereas patients who are continually and completely engulfed by their experience will tend to describe it in hallucinatory terms.... Thus, images such as 'Saturnian gravity' are used with great frequency by patients. One patient (Helen K.) was asked how it felt to be Parkinsonian: 'Like being stuck on an enormous planet,' she replied. 'I seemed to weigh tons, I was crushed, I couldn't move.' A little later she was asked how she had felt on L-DOPA (she had become very flighty, volatile, mercurial): 'Like being on a dotty little planet,' she said. 'Like Mercury – no, that's too big, like an asteroid! I couldn't stay put, I weighed nothing, I was all over the place. It's all a matter of gravity, in a way – first there's too much, then there's too little. Parkinsonism is gravity, L-DOPA is levity, and it's difficult to find any mean in between.' Such comparisons are also used, in reverse, by patients with Tourette's (Sacks, 1981).

[9] A special form of negative disorder, not described in the classical literature, is depicted with Hester Y. (see this page–this page).

[10] Thus one may see such patients, rigid, motionless, seemingly lifeless as statutes, abruptly called into normal life and action by some sudden exigency which catches their attention (in one famous case, a drowning man was saved by a Parkinsonian patient who leapt from his wheelchair into the breakers). The return of Parkinsonism, in circumstances like these, is often as sudden and dramatic as its vanishing: the suddenly 'normal' and awakened patient, once the call-to-action is past, may fall back like a dummy into the arms of his attendants.

Dr Gerald Stern tells me of one such patient at the Highlands Hospital in London who was nicknamed 'Puskas' after the famous footballer of the 1950s. Puskas would often sit frozen and motionless *unless* he were thrown a ball; this would instantly call him to life, and he would leap to his feet, swerving, running, dribbling the ball, with a truly Puskas-like acrobatic genius. If thrown a matchbox he would catch it on the tip of one foot, kick it up, catch it, kick it up again, and in this fashion, juggling the matchbox on one foot, hop the entire length of the ward. He scarcely showed any 'normal' activity; only this bizarre and spasmodic super-activity, which ended, as it started, suddenly and completely.

There is another story of the post-encephalitic patients at Highlands. Two of the men had shared a room for twenty years, but without any contact or, apparently, any feeling for each other; both were totally motionless and mute. One evening, while doing rounds, Dr Stern heard a terrific noise coming from this room of perpetual silence. Rushing to it with a couple of nurses, he found its inmates in the midst of a violent fight, throwing each other around and shouting obscenities. The scene, in Dr Stern's words, was 'not far short of incredible – none of us ever *imagined* these men could move.' With some difficulty the men were separated and the fight was stopped. The moment they were separated, they became motionless and mute again – and have remained so for the last fifteen years. In the thirty-five years they have shared a room, this is the *only* time they 'came alive.'

This mixture of akinesia and a sort of motor genius is very characteristic of post-encephalitic patients; I think of one such, not at Mount Carmel, who sits motionless until she is thrown three oranges (or more). Instantly she starts juggling them – she can juggle up to seven, in a manner incredible to see – and can continue doing so for half an hour on end. But if she drops one, or is interrupted for a moment, she suddenly becomes motionless again. With another such patient (Maurice P.), who came to Mount Carmel in 1971, I had no idea that he was *able* to move, and had long regarded him as 'hopelessly akinetic,' until, one day, as I was writing up my notes, he suddenly took my ophthalmoscope, a most intricate one, unscrewed it, examined it, put it together again, and gave a stunning imitation of me examining an eye. The entire 'performance,' which was flawless and brilliant, occupied no more than a few seconds.

Less abrupt and complete, but of more therapeutic relevance, is the *partial* lifting of Parkinsonism, for long periods of time, in response to interesting and activating situations, which *invite* participation in a non-Parkinsonian mode. Different forms of such therapeutic activation are

exemplified throughout the biographies in this book, and explicitly discussed on this page, and in an Appendix: Parkinsonian Space and Time, this page.

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[1] 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.[2]

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.[3] 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.[4] 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.[5]

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 always a defensive manoeuvre undertaken by schizophrenic patients at periods of unendurable stress and desperation.[6]

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 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*.[7]

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.'[8] Sexual and destructive outbursts were rarely outspoken in adults, being 'converted' (presumably) to other, more 'allowable,' reactions and expressions. Jelliffe,[9] 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.[10] They were highly individual, no two patients 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.[11] These 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.

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[1] 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.'

[2] 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.'

[3] 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.'

[4] 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 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*.

[5] 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 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).

[6] 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.

[7] 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.

[8] 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 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.

[9] 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)

[10] 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 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.

[11] 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 phenomena endorse the notion that our memories, or beings, are 'a collection of moments' (see n. 15).

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.

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.

LIFE AT MOUNT CARMEL

Mount Carmel was opened, shortly after the First World War, for war-veterans with injuries of the nervous system, and for the expected victims of the sleeping-sickness. It was a cottage hospital, in these early days, with no more than forty beds, large grounds, and a pleasant prospect of surrounding countryside. It lay close to the village of Bexley-on-Hudson, and there was a free and friendly exchange between the hospital and the village: patients often went to the village for shopping or meals, or silent movies, and the villagers, in turn, frequently visited the hospital; there were dates, and dances, and occasional marriages; and there were friendly rivalries in bowls and football, in which the measured deliberation of the villagers would be met by the abnormal suddenness and speed of movement characteristic of so many encephalitic patients, fifty years ago.[1]

All this has changed, with the passage of years. Bexley-on-Hudson is no longer a village, but a crowded and squalid suburb of New York; the leisurely life of the village has gone, to be replaced by the hectic and harried anti-life of New York; Bexleyites no longer have any time, and rarely spare a thought for the hospital among them; and Mount Carmel itself has grown sick from hypertrophy, for it is now a 1,000-bed institution which has swallowed its grounds; its windows no longer open on pleasant gardens or country, but on ant-nest suburbia, or nothing at all.

Still sadder, and more serious, has been the change in its character, the insidious deterioration in atmosphere and *care*. In its earlier days – indeed, before 1960 – the hospital was both easy-going and secure; there were devoted nurses and others who had been there for years, and most of the medical positions were honorary and voluntary, calling forth the best side, the kindness, of visiting doctors; and though its patients had grown older and frailer, they could look forward to

excursions, day-trips, and summer-camps. In the past ten years, and especially the last three years, almost all this has changed. The hospital has assumed somewhat the aspect of a fortress or prison, in its physical appearance and the way it is run. A strict administration has come into being, rigidly committed to 'efficiency' and rules; 'familiarity' with patients is strongly discouraged. Law and order have been ousting fellow-feeling and kinship; hierarchy separates the inmates from staff; and patients tend to feel they are 'inside,' unreachably distant from the real world outside. There are, of course, gaps in this totalitarian structure, where *real* care and affection still maintain a foothold; many of the 'lower' staff – nurses, aides, orderlies, physiotherapists, occupational therapists, speech therapists, etc. – give themselves unstintingly, and with love, to the patients; volunteers from the neighbourhood provide non-professional care; and, of course, *some* patients are visited by relatives and friends. The hospital, in short, is a singular mixture, where freedom and bondage, warmth and coldness, human and mechanical, life and death, are locked together in perpetual combat.[2]

In 1966, when I first went to Mount Carmel, there were still some eighty post-encephalitic patients there, the largest, and perhaps the only, such group remaining in the United States, and one of the very few such groups remaining in the world. Almost half of these patients were immersed in states of pathological 'sleep,' virtually speechless and motionless, and requiring total nursing-care; the remainder were less disabled, less dependent, less isolated, and less depressed, could look after many of their own basic needs, and maintain a modicum of personal and social life. Sexuality, of course, was forbidden in Mount Carmel.

Between 1966 and 1969, we brought the majority of our post-encephalitic patients (many of whom had been immured in remote, unnoticed bays of the hospital) into a single, organic, and self-governing community; we did what we could to give them the sense of being *people*, and not condemned prisoners in a vast institution; we instituted a search for missing relatives and friends, hoping that some

relationships – broken by time and indolence, rather than hostility and guilt – might thus be reforged; and I myself formed with them such relationships as I could.

These years, then, saw a certain establishment of sympathies and kinships, and a certain melting-away of the rigid staff/inmate dichotomy; and with these, and all other forms of treatment, a certain – but pitifully limited – improvement in their overall condition, neurological and otherwise. Opposing all forms of therapeutic endeavour, and setting a low ceiling on what could be achieved, was the crushing weight of their illness, the Saturnian gravity of their Parkinsonism, etc; and behind this, and mingling with it, all the dilapidations, impoverishments, and perversions of long isolation and immurement.[3]

Some of these patients had achieved a state of icy hopelessness akin to serenity: a realistic hopelessness, in those pre-DOPA days:[4] they *knew* they were doomed, and they accepted this with all the courage and equanimity they could muster. Other patients (and, perhaps, to some extent, all of these patients, whatever their surface serenity) had a fierce and impotent sense of outrage: they had been *swindled* out of the best years of life; they were consumed by the sense of time lost, time *wasted*; and they yearned incessantly for a twofold miracle – not only a cure for their sickness, but an indemnification for the loss of their lives. They wanted to be given back the time they had lost, to be magically replaced in their youth and their prime.

These were their expectations before the coming of L-DOPA.

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[1] This abnormal suddenness and speed of movement, often allied to an odd and unexpected, and sometimes very playful, quality may be of distinct advantage in certain sports. Thus one of my patients, Wilbur F., had been a very successful amateur boxer in his post-encephalitic youth. He showed me some fascinating old newspaper clippings from this time which attributed his success less to strength and skill than to the extraordinary speed and *strangeness* of his movements – movements which, without being illegal, were so odd as to be completely unanswerable. A similar tendency

to sudden, 'prankish' moves, allied with great speed and inventiveness, a bizarre sort of 'motor genius,' is sometimes characteristic of Tourette's syndrome (see Sacks, 1981).

[2] We have seen that Parkinsonism and neurosis are innately coercive, and share a similar *coercive structure*. Rigorous institutions are also coercive, being, in effect, *external neuroses*. The coercions of institutions call forth and aggravate the coercions of their inmates: thus one may observe, with exemplary clarity, how the coerciveness of Mount Carmel aggravated neurotic and Parkinsonian tendencies in post-encephalitic patients; one may also observe, with equal clarity, how the 'good' aspects of Mount Carmel – its sympathy and humanity – reduced neurotic and Parkinsonian symptoms.

[3] It is of the greatest interest to compare the state of these patients at Mount Carmel with that of the only remaining post-encephalitic community in England (at the Highlands Hospital). Conditions at Highlands – where there are large grounds, free access to and from a neighbouring community, devoted attention, and a much freer and easier atmosphere – are akin to those which obtained at Mount Carmel in its early days. The patients at Highlands (most of whom have been there since the 1920s), although they have severe post-encephalitic syndromes, convey an altogether different appearance from the patients at Mount Carmel. They tend, by and large, to be mercurial, sprightly, impetuous, and hyper-active – with vivid and ardent emotional reactions. This is in the greatest contrast to the deeply Parkinsonian, entranced, grave, or withdrawn appearance of so many patients at Mount Carmel. It is clear that both groups of patients have the same disease, and it is equally clear that the *form* and evolution of illness have been quite different in the two groups.

It has never been clear to me whether these different forms of illness are due to different pathophysiological 'fates,' or the effects of differing environment and atmosphere: a rather open and cheery atmosphere at Highlands, a rather gloomy and withdrawn atmosphere at Mount Carmel. I favoured the latter interpretation in previous editions, but without clear supporting evidence. I should say that we also have a number of sprightly, impish, witty-ticcy patients at Mount Carmel, strongly reminiscent of their brothers in pathology at Highlands. So perhaps it is 'fate,' not environment. Most likely it is both in interaction. The peculiar *antic* character of such post-encephalitics is extremely characteristic, and often endearing, and earned them the affectionate nickname of 'enkies' in England. The qualities of 'enkieness' were not too striking at Mount Carmel, at first, because so

many of the patients were wrapped in deep Parkinsonism when I saw them. They have become much more striking with the lifting of Parkinsonism – the continued stimulation of L-DOPA and (in some cases) a return to the effervescence of their earlier days.

[4] Anticholinergic drugs (hyoscyamine was the first) for the treatment of Parkinsonism had been introduced by Charcot, who used extracts of black henbane *(hyoscyamus niger)*, as long ago as 1869 – but they were useful only for treating rigidity and tremor, not for the profound akinesia which post-encephalitic patients tended to have. The same was true of surgical treatments: chemo-pallidectomies and later thalamotomies, were introduced in the 1930s, and found invaluable in treating rigidity and tremor – but were of no help for akinesia. Apomorphine was found in the 1950s to reduce akinesia, but it required injection, and was too brief and too emetic in action, to be of much use. Amphetamines too could reduce akinesia a little, but had prohibitive 'side effects' at the large doses required. Thus akinesia – the single most overwhelming feature of post-encephalitic Parkinsonism – remained untreatable until the advent of L-DOPA.

notion that 'health,' 'well-being,' 'happiness,' etc. can be reduced to certain 'factors' or 'elements' – principles, fluids, humours, commodities – *things* which can be measured and weighed, bought and sold. Health, thus conceived, is reduced to a *level*, something to be titrated or topped-up in a mechanical way. Metaphysics in itself makes no such reductions: its terms are those of organization or design. The fraudulent reduction comes from alchemists, witch-doctors, and their modern equivalents, and from patients who long *at all costs* to be well.

It is from this debased metaphysics that there arises the notion of a mystical substance, a miraculous drug, something which will assuage all our hungers and ills, and deliver us instantly from our miserable state: metaphorical equivalents of the Elixir of Life.[2] Such notions and hopes fully retain today their ancient, magical, mythical force, and – however we may disavow them – show themselves in the very words we use: 'vitamins' (vital amines), and the vitamin-cult; or 'biogenic amines' (life-giving amines) – of which dopamine (the biologically active substance into which L-DOPA is converted) is itself an example.

The notion of such mystical, life-giving, sacramental remedies gives rise to innumerable cults and fads, and to enthusiasms of a particularly extravagant and intransigent type. One sees this in Freud's espousal of the drug cocaine;[3] in the first wild reactions to the appearance of cortisone, when some medical conferences, in the words of a contemporary observer, 'more closely resembled revivalist meetings'; in the present world-wide 'drug-scene';[4] and, not least, in our present enthusiasm for the drug L-DOPA. It is impossible to avoid the feeling that here, over and above all legitimate enthusiasms, there is this special enthusiasm, this mysticism, of a magical sort.

We may now pass on to the 'straight' story of L-DOPA, remembering the mystical thread which always winds through it. Parkinson himself looked in vain for the 'seat' or substrate of Parkinsonism, although he tentatively located it in the 'pith' of the lower or medullary parts of the brain. Nor

was there any real success in defining the location and nature of the pathological process until a century after the publication of Parkinson's 'Essay.'[5] In 1919 von Economo, and separately Trétiakoff, described the findings of severe damage to the *substantia nigra* (a nucleus in the midbrain, consisting of large pigmented cells) in a number of patients with *encephalitis lethargica* who had shown severe Parkinsonian symptoms. The following year Greenfield, in England, and pathologists elsewhere, were able to define similar, but milder, changes in these cells in patients who had had ordinary Parkinson's disease. These findings, in company with other pathological and physiological work, suggested the existence of a clearly defined *system*, linking the *substantia nigra* to other parts of the brain: a system whose malfunctioning or destruction might give rise to Parkinsonian symptoms. In Greenfield's words:

> … A general survey has shown *paralysis agitans* in its classical form to be a systemic degeneration of a special type affecting a neuronal system whose nodal point is the *substantia nigra*.

In 1920 the Vogts, with remarkable insight, suggested that this anatomically and functionally distinct system might correspond with a *chemically distinct* system, and that a specific treatment for Parkinsonism, and related disorders, might become possible if this hypothetical chemical substance could be identified and administered.

> Studies should answer the question [they wrote], whether the striatal system or parts of it do or do not possess a special disposition towards certain injuring agents … Such a positive or negative tendency to react can be assumed to be ultimately due to the specific chemistry of the corresponding centre. The disclosure of the existence of such specific chemistry represents, in turn, at least the first step towards elucidation of its true nature, thereby initiating the development of a biochemical approach to treatment …

Thus in the 1920s, there was not merely a vague notion of 'something missing' in Parkinsonism patients (such as Charcot had entertained), but a clear path of research stretching out, pointing towards a prospect of ultimate success.

The most astute clinical neurologists, however, had reservations about this: was there not *structural* damage in the *substantia nigra*, and perhaps elsewhere, damage to nerve-cells and their connections? Could *this* be reversed? Would the administration of the missing chemical substrate be sufficient, or safe, given a marked degree of structural disorganization? Might there not be some danger of over-stimulating or over-loading such cells as were left? These reservations were expressed, with great pungency, by Kinnier Wilson:

> *Paralysis agitans* seems at present an incurable malady *par excellence*; the antidote to the 'local death' of cell-fibre systems would be the equally elusive 'elixir of life' … It is worse than useless to administer to the Parkinsonian any kind of nerve tonic to 'whip up' his decaying cells; rather must some form of readily assimilable pabulum be sought, in the hope of supplying from without what the cell itself cannot obtain from within.

Neurochemistry, as a science, scarcely existed in the 1920s, and the project envisaged by the Vogts had to await its slow development. The intermediate stages of this research form a fascinating story in themselves, but will be omitted from consideration here. Suffice it that in 1960 Hornykiewicz, in Vienna, and Barbeau, in Montreal, using different approaches, but almost simultaneously, provided clear evidence that the affected parts of the brain in Parkinsonian patients were defective in the nerve-transmitter *dopamine*, and that the transfer and metabolism of dopamine in these areas was also disturbed. Immediate efforts were made to replenish the brain-dopamine in Parkinsonian patients by giving them the natural precursor of dopamine –

laevodihydroxyphenylalanine, or L-DOPA (dopamine itself could not pass into the brain).[6] The results of these early therapeutic efforts were encouraging but inconclusive, and seven more years of arduous research had to be undertaken. Early in 1967, Dr Cotzias and his colleagues, in their now-classic paper, were able to report a resounding therapeutic success in the treatment of Parkinsonism, giving massive doses of L-DOPA by mouth.[7]

The impact of Cotzias's work was immediate and astounding in the neurological world. The good news spread quickly. By March 1967, the post-encephalitic and Parkinsonian patients at Mount Carmel had already heard of L-DOPA: some of them were eager to try it at once; some had reservations and doubts, and wished to see its effects on others before they tried it themselves; some expressed total indifference: and some of course were unable to signal any reaction.

The cost of L-DOPA in 1967 and 1968 was exceedingly high (more than $5,000 a pound), and it was impossible for Mount Carmel – a charity hospital, impoverished, unknown, unattached to any university or foundation, beneath the notice of drug-firms, industrial, or government sponsors – to buy L-DOPA at this time. Towards the end of 1968, the cost of L-DOPA started a sharp decline, and in March 1969 it was first used at Mount Carmel.

I could, perhaps, despite its cost, have started a few of our patients on L-DOPA after reading Cotzias's paper. But I hesitated – and hesitated for two years. For the patients under my care were not 'ordinary' patients with Parkinson's disease: they had far more complex pathophysiological syndromes, and their situations were more complex, indeed without precedent – for they had been institutionalised, and out of the world, for decades – in some cases since the time of the great epidemic. Thus even before I started, I was faced by scientific and human complexities, complexities and perplexities of a sort which had not arisen in previous trials of levodopa, or indeed of any treatment in the past. Thus

there was an element of the extraordinary, the unprecedented, the unpredictable. I was setting out, with my patients, on an uncharted sea …

I did not know what might happen, what might be released – the more so as some of my patients had been violently impulsive and hyperkinetic *before* being enclosed in a straightjacket of Parkinsonism. But as illness and death claimed some of my patients – especially in the fierce summer of 1968 – the need to do something became ever clearer and stronger, finally moving me to start L-DOPA, though with great caution, in March 1969.

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[1] One of the great surprises (or should one say providences?) of nature is that the plant world contains so many substances which have a profound effect upon animals – and yet, apparently, are of no obvious 'use' to the plant. Thus the foxglove *(Digitalis)* contains digitalis glycosides, which are invaluable in the treatment of heart-failure; the autumn crocus *(Colchicum)* contains colchicine, invaluable in the treatment of gout, etc., etc. It is again characteristic that many such 'natural remedies' are discovered at a very early stage of human history, and may form part-and-parcel of a folk-medicine long before their efficacy is allowed by conventional or established medical science. It has recently been established, by chemical analysis, that several species of bean (especially the fava bean) contain large amounts of L-DOPA (of the order of 25 gm. L-DOPA in a pound of beans). There is also a suggestion (which requires careful examination) that such L-DOPA-rich beans may have constituted a 'folk-remedy' for Parkinsonians for many centuries, if not longer. Thus although we ascribe 'The Coming of L-DOPA' to A.D. 1967, it may well have 'come' by 1967 B.C.

[2] The notion of 'mystical substances' arises from a *reductio ad absurdum* of two world-views which, legitimately employed, have great elegance and power: one is the mosaic or topist view, associated with the philosophies of empiricism and positivism, and the other is a holist or monist view. These derive, respectively, from Aristotelian and Platonic metaphysics. Used with mastery, and a full understanding of their powers and limits, these two world-views have provided a groundwork for fundamental discoveries in physiology and psychology during the past two hundred years.

Mysticism arises by taking analogy for identity – turning similes and

Awakenings

FRANCES D.

Miss D. was born in New York in 1904, the youngest and brightest of four children. She was a brilliant student at high school until her life was cut across, in her fifteenth year, by a severe attack of *encephalitis lethargica* of the relatively rare hyperkinetic form. During the six months of her acute illness she suffered intense insomnia (she would remain very wakeful until four in the morning, and then secure at most two or three hours' sleep), marked restlessness (fidgeting, distractible and hyperkinetic throughout her waking hours, tossing-and-turning throughout her sleeping hours), and impulsiveness (sudden urges to perform actions which seemed to her senseless, which for the most part she could restrain by conscious effort). This acute syndrome was considered to be 'neurotic,' despite clear evidence of her previously well-integrated personality and harmonious family life.

By the end of 1919, restlessness and sleep-disorder had subsided sufficiently to allow resumption and finishing of high school, although they continued to affect Miss D. more mildly for a further two years. Shortly after the end of her acute illness, Miss D. started to have 'panting attacks,' at first coming on two or three times a week, apparently spontaneously, and lasting many hours; subsequently becoming rarer, briefer, milder, and more clearly periodic (they would usually occur on Fridays) or circumstantial (they were especially prone to occur in circumstances of anger and frustration). These respiratory crises (as they clearly were, although they also were termed 'neurotic' at the time) became rarer and rarer, and ceased to occur entirely after 1924. Miss D., indeed, made no spontaneous mention of these attacks when first seen by me, and it was only later, when being questioned in greater detail before the administration of L-DOPA, that she recollected these attacks of

half a century previously.

Following the last of her respiratory crises, Miss D. had the first of her oculogyric crises, and these indeed continued to be her sole post-encephalitic symptom for twenty-five years (1924–49), during which time Miss D. followed a varied and successful career as a legal secretary, as an active committee-woman in social and civic affairs, etc. She led a full life, with many friends, and frequent entertaining; she was fond of theatre, an avid reader, a collector of old china, etc. Talented, popular, energetic, well-integrated emotionally, Miss D. thus showed no sign of the 'deterioration' said to be so common after severe encephalitis of the hyperkinetic type.

In the early 1950s, Miss D. started to develop a more sinister set of symptoms; in particular a tendency to freeze in her movements and speech, and a contrary tendency to hurry in her walking, speech, and handwriting. When in 1969 I first asked Miss D. about her symptoms she gave me the following answer: 'I have various banal symptoms which you can see for yourself. But my *essential* symptom is that I cannot start and I cannot stop. Either I am held still, or I am forced to accelerate. I no longer seem to have any in-between states.' This statement sums up the paradoxical symptoms of Parkinsonism with perfect precision. It is instructive, therefore, that in the absence of 'banal' symptoms (e.g. rigidity, tremor, etc., which only became evident in 1963), the diagnosis of Parkinsonism failed to be made, but that a large variety of other diagnoses (such as 'catatonia,' 'hysteria') were offered. Miss D. was finally labeled Parkinsonian in 1964.

Her oculogyric crises, to return to this cardinal symptom, were originally of great severity, coming many times a month and lasting up to fifteen hours each. Within a few months of their onset they had settled down to a fairly strict periodicity, coming 'like clockwork' every fifth day, so much so that Miss D. could plan a calendar for months in advance, knowing that she would inevitably have a crisis every five days, and only very occasionally at other times. The rare departures from this schedule which occurred were usually associated with circumstances of great annoyance or distress. The crises

would occur abruptly, without warning, her gaze being forced first downwards or to either side for several minutes, and then suddenly upwards, where it would stay for the remainder of the attack. Miss D. stated that her face would assume 'a fixed angry or scared expression' during these attacks, although she experienced neither rage nor fear while they lasted. Movement would be difficult during a crisis, her voice would be abnormally soft, and her thoughts seemed to 'stick'; she would always experience a 'feeling of resistance,' a force which opposed movement, speech, and thought, during the attack. She would also feel intensely wakeful in each attack, and find it impossible to sleep; as the crises neared their termination, she would start to yawn and become intensely drowsy; the attack would finally end quite suddenly, with restoration of normal movement, speech, and thought (this sudden restoration of normal consciousness Miss D. – a crossword addict – would call 'resipiscence'). In addition to these classical oculogyric crises, Miss D. started to experience a number of variant crises after 1955: forced deviation of gaze became exceptional, being replaced by a fixed and stony stare; some of these staring attacks were of overwhelming severity, completely depriving her of movement and speech, and lasting up to three days. She was admitted to a municipal hospital on several occasions during the 1960s when neighbours had discovered her in these attacks, and she was displayed at staff meetings as a striking case of 'periodic catatonia.' Since 1962, Miss D. has also had brief staring attacks, lasting only a few minutes, in which she is arrested and feels 'entranced.' Yet another paroxysmal symptom has been attacks of flushing and sweating, coming on at irregular intervals, and lasting fifteen to thirty minutes. (Miss D. had completed her menopause in the mid 1940s.) Since 1965, staring and oculogyric crises had become mild and infrequent, and when admitted to Mount Carmel Hospital at the start of 1969, Miss D. had been free of them for more than a year, and continued to be exempt from them until given L-DOPA in June 1969.

Although, as mentioned, rigidity and tremor had appeared

in 1963, the most disabling of Miss D.'s symptoms, and the ones which finally necessitated her admission to a chronic disease hospital, were threefold: a progressive flexion-dystonia of the neck and trunk, uncontrollable festination and forced running, backwards or forwards, and uncontrollable 'freezing' which would sometimes arrest her in awkward positions for hours on end. A further symptom of relatively recent onset, for which no local infective aetiology could be found, was urinary frequency and urge; sometimes this urge would coexist with or call forth a 'block' or 'reluctance' of micturition – an intolerable coupling of opposing symptoms.

On admission to Mount Carmel Hospital, in January 1969, Miss D. was able to walk freely using two sticks, or for short distances alone; by June 1969, she had become virtually unable to walk by herself alone. Her posture, which was bent on admission, had become almost doubled-up over the course of the following six months. Transferring from bed to chair had become impossible, as had turning over in bed, or cutting up food. In view of this rather rapid deterioration, and the uselessness of all conventional anti-Parkinsonian drugs, the advent of L-DOPA came at a critical time for Miss D., who seemed about to slip into an accelerating and irrevocable decline.

Before L-DOPA

Miss D. was a tiny, bent woman, so kyphotic that, on standing, her face was forced to gaze at the ground. She was able to raise her head briefly, but it would return within seconds to its habitual position of extreme emprosthotonos, with the chin wedged down on the sternum. This habitual posture could not be accounted for by rigidity of the cervical muscles: rigidity was not more than slightly increased in the neck, and in oculogyric crises her head would be forced backwards to an equally extreme degree.

There was quite severe masking of the face, alertness and emotional expression being conveyed almost exclusively by

institutionalized, a healthy self-respect, many interests, and a close attention to her environment, providing a focus of stability and humour and compassion on a large ward of disabled and sometimes very disturbed post-encephalitic patients.

She was started on L-DOPA on 25 June 1969.

Course on L-DOPA

30 June. Although this was only five days after the start of treatment, and Miss D. was receiving no more than 0.5 gm. of L-DOPA daily, she exhibited some general restlessness, increased fidgeting of the right hand, and masticatory movements. The puckering of circumoral muscles had become more pronounced and now showed itself to be a form of compulsive grimace, or tic. There was already an obvious increase of general activity: Miss D. was now always, but always, doing something – crocheting (which had been slow and difficult before administration of the drug), washing clothes, writing letters, etc. She seemed somewhat *driven*, and unable to tolerate inactivity. Miss D. also complained even at this very early stage of 'difficulty in catching the breath,' and showed a tachypnoea of forty breaths to the minute, without variation in the force or rhythm of breathing.

6 July. On the eleventh day of drug-trial, and receiving 2 gm. L-DOPA daily, Miss D. now exhibited a complex mixture of desirable and adverse effects. Among the good effects she showed a sense of well-being and abounding energy, a much stronger voice, less freezing, less postural flexion, and stabler walking with longer strides. Among the adverse effects she showed aggravation of her former mild chewing and biting movements, so that she incessantly chewed on her gums, which had become very sore; increased fidgeting of her right hand, to which was now added a tic-like flexion and extension of the forefinger; finally, and most distressing to her, a disintegration of the normal automatic controls of breathing. Her breathing had now become rapid, shallow, and irregular, and was broken up by sudden violent

inspirations two or three times a minute, each of which would follow a sudden, powerful, and fully conscious though uncontrollable *urge* to breathe. Miss D. remarked at this time: 'My breathing is no longer automatic. I have to think about each breath, and every so often I am *forced* to gasp.'

In view of these adverse symptoms, the dosage was reduced on this day. Over the ensuing ten days, on a dose of 1.5 gm. L-DOPA daily, Miss D. maintained the desirable effects of the drug and showed less restlessness, chewing, and pressure of activity. Her respiratory symptoms, however, persisted, growing more pronounced daily, finally differentiating, around 10 July, into clear-cut respiratory crises.[2] These attacks would start, without any warning whatever, with a sudden inspiratory gasp, followed by forced breath-holding for ten to fifteen seconds, then a violent expiration, and finally an apnoeic pause for ten to fifteen seconds. In these early and relatively mild attacks there were no associated symptoms or autonomic disturbances (e.g. tachycardia, hypertension, sweating, trembling, apprehension, etc.). This strange and distorted form of breathing could be interrupted for a minute or two by a strong effort of will, but would then resume its bizarre and imperative character. Her crises would last between one and three hours, finally subsiding over a period of about five minutes, with resumption of normal, automatic, unconscious breathing of even rate, rhythm, and force. The timing of these attacks was of interest, for it bore no constant relationship to the times at which L-DOPA was administered. Thus, for the first five days of respiratory crises, attacks occurred invariably in the evening and at no other times. On 15 July, for the first time, an attack occurred in the afternoon (at 1 p.m., an hour after L-DOPA had been given): on 16 July, for the first time, an attack occurred very early in the morning, before the first daily dose of L-DOPA had been taken. Subsequently, two or three attacks would occur every day, although the evening attacks continued to be the longest and severest.

On 16 July, I observed that the attacks were now assuming

a most frightening intensity. A violent and protracted gasp (which looked and sounded as desperate as that of a nearly drowned man finally coming to the surface for a lungful of air) would be followed by forced breath-holding for up to fifty seconds, during which time Miss D. would struggle to expel breath through a closed glottis, in so doing becoming purple and congested from the futile effort; finally the breath would be expelled with tremendous violence, making a noise like the boom of a gun. No voluntary control whatever was possible at this time; in Miss D.'s words: 'I can no more control it than I could control a spring tide. I just ride it out, and wait for the storm to clear.' During this crisis speech was, of course, quite impossible, and there was a clear increase of rigidity throughout the body. The pulse-rate was raised to 120, and the blood-pressure rose from its normal 130/75 to 170/100. Twenty mg. of Benadryl, given intravenously, failed to alter the course of this attack. Despite what I would have imagined was a terrifying experience, and an expression of terror on her face, Miss D. denied that any alteration of thinking or special apprehension had been experienced during the crisis. Greatly concerned about the possible effects of so violent an attack in an elderly patient, I was disposed at this time to discontinue the L-DOPA. But, at Miss D.'s insistence, in view of the real benefits she was obtaining from the drug, and in the hope that her respiratory instability might decrease, I contented myself with reducing its dosage to 1.0 gm. daily.

Despite this small dosage, Miss D. continued to have respiratory crises of varying severity, two or more commonly three times a day. Within two or three days, these had established a routine – a crisis at 9 a.m., a crisis at noon, and a crisis at 7.30 p.m. – which remained fixed despite chance and systematic alterations of the times at which she would receive L-DOPA. We had also come to suspect, by 21 July, that her respiratory crises were readily conditionable: on this day our speech-therapist stopped to talk to Miss D. at five in the afternoon (normally a crisis-free time), and inquired whether she had had any crises recently; before Miss D. could begin to

frame an answer, she was impelled to gasp violently and launch into an unexpected crisis which seemed suspiciously like an answer to the question.

By now a therapeutic dilemma was becoming clear. There was no doubt of the enormous benefit derived from L-DOPA: Miss D. was looking, feeling, and moving far better than she had done in twenty years; but she had also become overexcitable and odd in her behaviour, and in particular seemed to be experiencing a revival or revocation of an idiosyncratic respiratory sensitivity (or behaviour) which had lain dormant in her for forty-five years. There was also, even in her first month of treatment, a number of minor 'side-effects' (a term which I found it increasingly difficult to give any meaning to), with the promise (or threat) of others lurking *in posse* – as I imagined it – in an as-yet unactualized state. Could we find a happy medium, an in-between state and dosage which would greatly assist Miss D. *without* calling forth her respiratory symptoms and other 'side-effects'?

Once more (on 19 July) the dosage was reduced – to a mere 0.9 gm. of L-DOPA daily. This reduction was promptly followed, that very day, by the occurrence of an oculogyric crisis – Miss D.'s first such in almost three years. This was disconcerting, because we had already observed, in several other post-encephalitic patients, a situation in which any given therapeutic dose of L-DOPA evoked respiratory crises, and any lessening of this dose oculogyric crises, and we feared that Miss D., too, might have to walk a tightrope between these two disagreeable alternatives.

Although the reported experience of others encouraged us to suppose that one could 'balance' or 'titrate' patients by finding exactly the right dose of L-DOPA, our experience with Miss D. – at this time – suggested that she could no more be 'balanced' than a pin on its point. Her oculogyric crisis, which was severe, was at once followed by a second and third oculogyric crises; with increase of the L-DOPA to 0.95 gm. a day, *these* crises ceased, but respiratory crises returned; with diminution of L-DOPA to 0.925 gm. a day (we were forced, at this stage, to encapsulate L-DOPA ourselves, in order to allow

these infinitesimal increments and decrements of dose), the reverse switch occurred; and at a dose of 0.9375 gm. a day, she experienced *both* forms of crisis, in alternation, or simultaneously.

It became clear, at this time, that Miss D.'s crises, which were now occurring several times a day, showed a close association not only with overall psycho-physiological state, mood, and circumstance, but with certain specific dynamics, and in this way acted like migraines, and even like hysterical symptoms. If Miss D. had had a poor night and was tired, crises were more likely; if she was in pain (an ingrown toenail was troubling her at this time), she tended to have a crisis; if she became excited, she was especially prone to have a crisis, whether the excitement was fearful, angry, or hilarious in character; when she became frustrated, she exhibited crises; and when she desired attention from the nursing staff, she developed a crisis. I was slow to realize, while noting the causes of Miss D.'s crises, that the most potent 'trigger' of all was me, myself: I had indeed observed that as soon as I entered her room, or as soon as she caught sight of me, she usually had a crisis, but assumed that this was due to some other cause I had failed to notice, and it was only when an observant nurse giggled and remarked to me, 'Dr Sacks, *you* are the object of Miss D.'s crises!', that I belatedly tumbled to the truth. When I asked Miss D. if this could be the case, she indignantly denied the very possibility, but blushed an affirmative crimson. There was, finally, one other psychic cause of her crises which I could not have known of had Miss D. not mentioned it to me: 'As soon as *I think of getting a crisis*,' she confessed, 'I am apt to get one. And if I try to think of not getting a crisis, I get one. And if I try to think about not thinking about my crises, I get one. Do you suppose they are becoming an obsession?'

In the final week of July, Miss D.'s well-being was compromised not only by these crises, but by a number of other symptoms and signs, which increased in number and variety from day to day, and almost from hour to hour – a pathological blossoming, or ebullience, which could not be stopped, and which could scarcely be modified, however we

constraint was accompanied by a most intense, and almost frenzied, urge to move, so that Miss D., though motionless, was locked in a violent struggle with herself. She could not tolerate the idea of bed, and screamed incessantly unless left in her chair. Every so often she would burst loose from her 'jammed' state, and catapult forwards for a few steps only to 'jam' once more, as if she had suddenly run into an invisible wall. She exhibited extreme pressure of speech, and now showed, for the first time, an uncontrollable tendency to repeat words and phrases again and again (palilalia). Her voice, normally low-pitched and soft, rose to a shrill and piercing scream. When she was jammed in awkward positions she would scream: 'My arms, my arms, my arms, my arms, please move my arms, my arms, move my arms ...' Her excitement seemed to come in waves, each wave rising higher and higher towards some limitless climax, and with these waves a mixture of anguish and terror and shame overwhelmed her, to which she gave voice in palilalic screamings: 'Oh, oh, oh, oh! ... please don't ... I'm not myself, not myself ... It's not me, not me, not me at all.'

This crescendo of excitement responded only to massive doses of parenteral barbiturates, and these would allow only a few minutes of exhausted sleep, with resumption of all symptoms immediately on waking. Her L-DOPA, of course, had been stopped with the inauguration of this monstrous crisis.

Finally, on 31 July, Miss D. sank naturally into a deep and almost comatose sleep, from which she awoke after twenty-four hours. She had no crises on 2 and 3 August, but was intensely Parkinsonian (far more than she had ever been before the administration of L-DOPA), and painfully depressed, although she still showed a ghost of her old pluck and humour: 'That L-DOPA,' she whispered (for she was now almost voiceless), 'that stuff should be given its proper name – *Hell-*DOPA!'

1969–72

During August 1969 Miss D. remained in a subterranean state: 'She looks almost dazed at times,' our speech-pathologist, Miss Kohl, wrote to me, 'like someone who has come back from the front line, like a soldier with shell-shock.' During this shock-like period, which lasted about ten days, Miss D. continued to show an exacerbation of her Parkinsonism so extreme that she could perform none of the elementary activity of daily life without help from the nursing staff. For the remainder of the month, she was less Parkinsonian (though still far more so than she had been before the administration of L-DOPA), but quite deeply and painfully depressed. She had little appetite ('She seems to have no appetite for *anything*,' wrote Miss Kohl; 'really no appetite for living. She was like a blow-torch before, and now she's like a candle guttering out. You would never believe the difference'), and lost twenty pounds, and when I returned to New York in September – having been away for a month – I did in fact momentarily fail to recognize the pale, shrunken, and somehow caved-in figure of Miss D.[3]

Before the summer, Miss D., despite her half-century of illness, had always been active and perky, and had seemed considerably younger than her sixty-five years; now she was not only wasted and far more Parkinsonian than I had ever seen her, but frighteningly *aged*, as if she had fallen through another half-century in the month I was away. She looked like an escapee from Shangri-La.

In the months following my return Miss D. spoke to me at length about this month; her candour, courage, and insight provided a convincing analysis of how and *why* she felt as she did; and since her state (I believe) shares essential qualities and determinants with the 'post-DOPA' states experienced by many other Parkinsonian patients (though, of course, it was notably more severe than the majority of patients experience or can expect to experience) I shall interrupt her 'story' for her analysis of the situation.

Miss D. stressed, first, the extreme feeling of 'let-down' produced by the sudden withdrawal of the drugs: 'I'd done a vertical take-off,' she said. 'I had gone higher and higher on L-

DOPA – to an impossible height. I felt I was on a pinnacle a million miles high … And then, with the boost taken away, I crashed, and I didn't just crash to the ground, I shot way in the other direction, until I was buried a million miles deep in the ground.'

Secondly, Miss D. spoke (as has every patient of mine who has been through a comparable experience) of the bewilderment, uncertainty, anxiety, anger, and disappointment which assailed her when the L-DOPA 'started to go wrong'; when it produced more and more 'side-effects' which I – *we, her doctors* – seemed powerless to prevent, despite all our reassurances, and all our fiddlings and manipulations with the dosage; and finally, the extremity of her hopelessness when the L-DOPA was stopped, an act which she saw as a final verdict or decree: something which said in effect, 'This patient has had her chance and lost it. We gave her the magic and it failed. We now wash our hands of her, and consign her to her fate.'

A third aspect of the L-DOPA 'situation' was alluded to again and again by Miss D. (especially in a remarkable diary she kept at this time and of which she showed me portions). This was an acute, an almost intolerable exacerbation of certain feelings which had haunted her at intervals throughout her illness, and which rose to a climax during the final days of L-DOPA administration and the period immediately following withdrawal of the drug. These were feelings of astonishment, rage, and terror that *such things could happen to her*, and feelings of impotent outrage that she, Miss D., could do nothing about these things.[4] But deeper and still more threatening feelings were involved: some of the 'things' which gripped her under the influence of L-DOPA – in particular, her gnawing and biting compulsions,[5] certain violent appetites and passions, and certain obsessive ideas and images – could not be dismissed by her as 'purely physical' or completely 'alien' to her 'real self,' but, on the contrary, were felt to be in some sense *releases* or *exposures* or *disclosures* or *confessions* of very deep and ancient parts of

herself, monstrous creatures from her unconscious and from unimaginable physiological depths below the unconscious, pre-historic and perhaps prehuman landscapes whose features were at once utterly strange to her, yet mysteriously familiar, in the manner of certain dreams.[6] And she could not look upon these suddenly exposed parts of herself with detachment; they called to her with siren voices, they enticed her, they thrilled her, they terrified her, they filled her with feelings of guilt and punishment, they possessed her with the consuming, ravishing power of nightmare.

Connected with all of these feelings and reactions were her feelings towards me – the equivocal figure who had offered her a drug so wonderful and so terrible in its effects; the devious and Janus-faced physician who had prescribed for her a revivifying, life-enhancing drug, on the one hand, and a horror-producing, life-destroying drug, on the other hand. I had first seemed a Redeemer, promising health and life with my sacramental medicine; and then a Devil, confiscating health and life, or forcing on her something worse than death. In my first role – as the 'good' doctor – she necessarily loved me; in my second role – as the 'wicked' doctor – she necessarily hated and feared me. And yet she dared not express the hate and fear; she locked it within herself, where it coiled and recoiled upon itself, coagulating into the thickness and darkness of guilt and depression. L-DOPA, by virtue of its amazing effects, invested me – its giver, the physician held 'responsible' for these effects – with all too much power over her life and well-being. Invested with these holy and unholy powers, I assumed, in Miss D.'s eyes, an absolute, and absolutely contradictory, sovereignty; the sovereignty of parents, authorities, God. Thus Miss D. found herself entangled in the labyrinth of a torturing transference-neurosis, a labyrinth from which there seemed to be no exit, no imaginable exit, whatsoever.

My own disappearance from the scene (on 3 August) at the height of her anguish was experienced both as an enormous relief and as an irretrievable loss. I had placed her in the labyrinth in the first place; yet was I not the thread to lead

her from it?

This, then, was Miss D.'s situation when I returned in September.[7] I *felt* what was happening with her, in a very fragmentary and inchoate way, the moment I laid eyes on her again, but it was, of course, months and even years before my own intuitions, and hers, reached the more conscious and explicit formulations which I have sketched above.

Summer 1972

Three years have passed since these events. Miss D. is still alive and well, and living – living a sort of life. The dramatic quality of summer 1969 is a thing of the past; the violent vicissitudes of that time have never been repeated with her, and in retrospect have some of the unreality and nostalgia of a dream, or of a unique, never-repeated, unrepeatable, and now almost unimaginable historical event. Despite her ambivalences, Miss D. greeted my return with pleasure, and with a gentle, qualified request that the use of L-DOPA should be considered again. The insistence and intransigence had gone out of her manner; I felt that her seemingly subterranean month without L-DOPA had also been a month of deep reflection, and of inner changes and accommodations of great complexity. It had been, I was subsequently to realize, a sort of Purgatory, a period in which Miss D. struggled with her divided and manifold impulses, using all her recently acquired knowledge of herself (and her propensities of response to L-DOPA), and all her strength of mind and character, to achieve a new unification and stability, deeper and stronger than anything preceding it. She had, so to speak, been forged and tempered by the extremities she had passed through, not broken by them (as were so many of my patients). Miss D. was a superior individual; she had lived and fought with herself and for herself through half a century of illness, and had (against innumerable odds) been able to maintain a life of her own, outside an institution, until her sixty-sixth year. Her disease and her pathological potentials I