In My Own Time

Depression

ALAN NORTON

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Within a year of qualifying in 1937 I was offered a house job at Bethlem Royal Hospital, a venerable hospital in a new building on the Kent/Surrey border in south London. It housed about 250 patients. At that time Bethlem was one of a small group of "registered hospitals" providing psychiatric care for modest fees—about £5 a week, but often reduced—and concentrating on early and treatable cases. Patients with chronic illnesses were not admitted.

The chief diagnoses were, firstly, schizophrenia in all its forms, including Kraepelin's category of paraphrenia; and, secondly, psychotic depression in its several forms—simple endogenous, recurrent, manic-depressive, and involutional. At that time, clinical interest was focused on schizophrenia because Sakel's insulin treatment, fresh from Vienna, had been introduced to Britain in 1935 in Edinburgh. By 1938 every self-respecting go-ahead hospital had its insulin unit.

Depressive illnesses, on the other hand, were in the shadow. Depressed patients went into hospital only if their illnesses were severe, and quite often they were under compulsion. Patients with milder depressions were looked after as outpatients or remained in the care of their own doctors, often with vague and less opprobrious diagnostic labels. Admission was sought not for any specific treatment—there was none—but to prevent suicide, for skilled nursing, and for the relief of symptoms.

Precautions as deterrents

The ever-present risk of suicide preoccupied nurses and doctors; if the event occurred it was regarded, much more then than now, as an occasion for inquiry, criticism, and self-searching. Windows were "blocked" and would open only a few inches; stair wells were fenced in; doors were locked and double-locked; and, in the gardens, innocent-looking hedges concealed stout chicken wire. Patients were not allowed matches, scissors, nail files, or even, sometimes, spectacles; pyjama cords and bootlaces were viewed with suspicion; bath taps had keys; guards on open fires were locked. Actual suicide attempts or the discovery of a plan for one could lead to doubling restrictions and to issuing a caution card, printed in red ink, which had to be signed by every nurse on the ward. This was intended to ensure that the patient was not left alone for a moment, even in the lavatory where, in any event, the chain was boxed in.

A few young psychiatrists, myself included, were inclined to think some of these precautions excessive—at least until some frightful event taught us a lesson. In my own case, this was a 50-ish agitated woman who smeared herself and her clothes with furniture polish and somehow set herself alight. She died later that evening, and I am still liable to instant recall by the smell of scraps of clothing, hair, or fur on a bonfire. Though it may well be true that a really determined person will succeed in killing himself in spite of all precautions, it is important still, as it was then, not to be fatalistic about an illness with a good chance of complete remission. Even quite modest precautions do deter people.

Many depressives, especially those who were verging on depressive stupor, failed to eat enough. Hesitancy, slowness, and agitation made each meal a battle and called for exemplary patience from the nurses. Regular weighing was therefore of great importance to help us to decide whether tube-feeding should be resorted to. In depressive illnesses at that time, this was uncommon; in each case, a few tube-feeds were usually enough to get the patient eating again. Coercive? Perhaps; but not punitive, and likely to prevent inhalation pneumonia from over-enthusiastic spoon-feeding. In my time, it was a catatonic schizophrenic, not a depressive, who had to be tube-fed twice a day for over a year.

Some symptoms could be dealt with fairly successfully: sleeplessness, for example, with barbiturates, chloral, or, preferably, the safer paraldehyde. The smell of this last drug contributed—with that of the rubber chamber pots and the rubber lining of the padded room—to the characteristic odour of acute psychiatric wards all over Britain. Agitation, too, or rather its severer manifestations—head-banging, rubbing, and pulling out of hair, relentless picking away at the skin causing sepsis—could be somewhat relieved by sedative drugs. In extreme form these symptoms, as well as excitement and aggression, might be relieved for the time being by Hyoscine Co A, a potent compound of hyoscine, morphine, and atropine.

But of anything more than symptomatic treatment and good nursing, or anything nearer the core of the illness, there was no sign. To preserve life and physical health and await remission was all that anyone could do for an illness widely recognised (in the words of a *Lancet* editorial)[1] as "perhaps the most unpleasant illness that can fall to the lot of man."

Social measures and talk treatment might help in milder cases, but in those ill enough to enter hospital the condition seemed impervious to argument, persuasion, or reassurance. Delusions abounded: wickedest woman in the world; committed the unpardonable sin; brain rotted away; bowels permanently blocked; irretrievable ruin faces me; despaired of by God; throat closing up; riddled with cancer. These and other delusions were voiced commonly enough in our wards, though not all at the same time, and less extreme symptoms were more frequent. Many patients just felt endlessly miserable and hopeless, couldn't be bothered to do anything, felt that they were failures and that nothing was worth while. Some were drearily repetitive and importunate, plucking at one's clothing as one went by. Most compared rather unfavourably with the schizophrenic patients for interest, oddness, and as teaching material.

Dartmouth Row, Greenwich, London SE10
ALAN NORTON, DM, FRCPSYCH, honorary consultant psychiatrist

Continuous narcosis with somnifaine, a barbiturate mixture, had been introduced by Kläsi in 1922. It aimed at keeping the patient asleep for 20-22 hours a day; but, though it seemed so reasonable, it had proved a disappointment. It had an appreciable mortality rate. Moreover, those who did make some improvement usually relapsed. Nevertheless, so far as I can remember, it was still occasionally used at Bethlem in 1938.

The high natural remission rate was the only good thing about these depressions. The great majority could expect to recover more or less completely in six to eight months, though there were reservations about the risk of further attacks and about the prognosis of those patients with special features such as depersonalisation, hypochondriasis, and arterial disease. Involutional depression, depressive illness coming on for the first time over the age of 50, took rather longer to remit. These comfortable beliefs persisted despite the jolt that Sir Aubrey Lewis's classic papers had administered in 1934[2 3] and 1936.[4]

Good effects and side effects

Gloom about the futility of efforts at treatment, tempered by the fairly favourable prognosis, was not relieved by rumours and then reports of progress with another form of treatment—besides insulin—for schizophrenia. Convulsion treatment with camphor had been begun by von Meduna in Budapest in 1934. With chronic patients, the results were at first disappointing, but by the time Professor Alexander Kennedy's paper[5] appeared in 1937 a new drug, leptazol (Cardiazol), was being used, and the results on first illnesses were more encouraging. For depressives, the outlook began to brighten only in 1938 when Dr Leslie Cook[6] reported dramatically promising results in five cases of psychotic depression treated with Cardiazol.

There were all sorts of difficulties: the liability of Cardiazol to sclerose veins; the need for very rapid injection to produce a fit; the risk of the convulsion dislocating the shoulder and the jaw, and causing fractures there and elsewhere. But, worst of all was the experience of patients during the 5-10 seconds between the injection of the chemical and the onset of the fit, made much worse if the fit aborted. Many patients refused to complete the course, some saying that the experience of Cardiazol was worse than death. I recall having some ethical doubts myself about giving this most unpleasant treatment to patients who in all probability would get well in a few months without it—a few months of hell, of course, and perhaps of continuing suicidal risk. A colleague of mine at Bethlem, of Quaker persuasion, felt that he could not justify continuing to use this method unless he experienced it himself. Full of doubt and misgivings, three of us assembled one morning in his bedroom. All went well, happily, and he said that the experience had not been too bad. Nevertheless, he had only one treatment and did not have an abortive fit; I wouldn't do that again.

After Drs Alexander Walk and W Mayer-Gross[7] published their article in 1938 on cycloethyltriazol (Triazol, Azoman), many hospitals turned to this as a convulsive agent. It had many advantages over Cardiazol and did away with some of the horrors. Refusal became much less common. Psychiatry, after standing still for so long or, so it seemed to me, at most exploring blind alleys, was undoubtedly on the march. Preceded by two research papers[8 9] describing Drs U Cerletti's and L Bini's method of provoking convulsions by electricity, the next step forward was described in a paper by Drs W H Shepley and J S McGregor[10] in 1940. As compared with injection methods, they said, electric convulsion therapy (ECT) produced instant unconsciousness, no dread, no physical upset after the convulsion, no vomiting, no venous thrombosis, no repetition of a fit, and fewer fractures and dislocations. But crush fractures of vertebrae still occurred, and the exertion of a major fit limited the treatment to those in fair physical shape. The early papers, and indeed one's early experience with ECT, did not draw anyone's attention to memory disorder persisting beyond 24 hours after the fit. This is a modern side effect, and the reasons for it are obscure. I well recall our Solus machine arriving; it was the size of a small cinema organ, its top a rather bewildering array of dials and switches. Although the machine was fearsome to us nurses and doctors to begin with, the fears of patients about convulsion treatment became unusual and outright refusal rare.

The war now intervened, and during it and its aftermath I myself treated no seriously depressed people. By 1947, when I returned to work with depressives, progress, which had slowed down but not stopped in wartime, had accelerated. Psychiatrists were still worried by the many (but symptomless) fractures of the dorsal spine sustained by patients having convulsion therapy. These had been noticed by Dr Harold Palmer[11] as far back as 1939, and to prevent them he had used a derivative of curare. Subsequently, other muscle relaxants were developed, each briefer in action than the one before but all needing a simultaneous short general anaesthetic to avoid the patient feeling as if his breathing was being paralysed. This modification of ECT became popular, and it was soon clear that the help of an anaesthetist was necessary for every session of treatment. By the early 1950s unmodified ECT had become outdated.

In the next 25 years the treatment of depression became as varied as were the places where it was carried out. First and foremost was the development of two families of effective antidepressant drugs—effective, that is, against all but the most severe attacks accompanied by retardation and delusions; and lithium began to be used as a means of preventing relapse. Secondly, and related to the availability of drugs, there was a shift in treatment towards milder cases, for which "psychosocial" methods were more appropriate than they had been for the severer ones. And whatever the treatment it may be carried out in an outpatient department, a day hospital, a psychiatric unit of a general hospital, as well as an admission ward of a psychiatric hospital. But, despite the choice now available, ECT has by no means been displaced; it has retained its popularity over the past 40 years, especially for severe cases that other forms of treatment do not seem to touch. Whether the depression is mild or severe, treatment has shortened its duration from several months to a few weeks.

Even today, the successful treatment of a case of depression must be rewarding, although the method has become almost routine. To have taken part in the successful treatment of this very unpleasant illness while it was being developed was exhilarating. I count myself lucky to have been in the right place at the right time.

References

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Should a patient who has had a cholecystectomy for gall bladder stones be advised to diet? Are the stones likely to recur?

There is no necessity for any special diet after cholecystectomy. After cholecystectomy stones are sometimes found in the common bile duct, but it is thought unlikely that they form here unless there is a stricture or other bile duct abnormality; most were probably present at the time of operation but not discovered. Such stones are not amenable to dietary or drug treatment and require removal by reoperation or by endoscopic papillotomy.