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To cite this version:

Niall Mccrae. ‘A violent thunderstorm’: Cardiazol treatment in British mental hospitals. History of Psychiatry, 2006, 17 (1), pp.67-90. 10.1177/0957154X06061723 . hal-00570852

HAL Id: hal-00570852

https://hal.science/hal-00570852v1

Submitted on 1 Mar 2011

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History of Psychiatry, 17(1): 067–090 Copyright © 2006 SAGE Publications (London, Thousand Oaks, CA and New Delhi) www.sagepublications.com [200603] DOI: 10.1177/0957154X06061723

'A violent thunderstorm': Cardiazol treatment in British mental hospitals

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In the annals of psychiatric treatment, the advent of Cardiazol therapy has been afforded merely passing mention as a stepping-stone to the development of electroconvulsive therapy. Yet in the 1930s it was the most widely used of the major somatic treatment innovations in Britain's public mental hospitals, where its relative simplicity and safety gave it preference over the elaborate and hazardous insulin coma procedure. Devised on a dubious hypothesis of biological antagonism, Cardiazol armed psychiatry with an immediately effective weapon in the battle against schizophrenia, an enduring and debilitating condition responsible for over half of the mental hospital population. What made Cardiazol work – or appear to work? This account shows how evaluation of convulsive therapy was skewed by naïve outcome measurement and diagnostic discrepancies, and how its therapeutic indication evolved from schizophrenia to affective disorders. Psychological mechanisms are considered, with the suggestion that the intense fear experienced during treatment – the major reason for abandoning Cardiazol in favour of electroshock – was therapeutically advantageous.

***Keywords**: Britain; Cardiazol treatment; convulsive therapy; electroshock; history; Isabel Wilson; Ladislaus von Meduna; Manfred Sakel; physical treatments; psychiatry*

The birth of Cardiazol treatment

On the heels of Wagner-Jauregg's malarial therapy for neurosyphilis, ground-breaking discoveries in the early 1930s raised the prospect of slaying that great dragon of mental disorder, schizophrenia, by somatic methods. The first revelation arose from Vienna, after Manfred Sakel, who had been using

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insulin in treating morphine addiction, made an intriguing observation in patients who had inadvertently slipped into hypoglycaemic coma. On recovering, previously mentally disturbed patients became tranquil. Sakel considered whether inducing insulin coma might cure schizophrenia, and was permitted to test his hypothesis by Otto Pöetzl, Wagner-Jauregg's successor as Professor of Psychiatry at Vienna's university clinic (James, 1992). The remarkable results, first disseminated in 1934, met scepticism in some quarters, but received an enthusiastic response in Switzerland where another physical procedure, Jakob Klaesi's *Dauerschlaf* (prolonged narcotic therapy) had been devised in the 1920s. The Münsingen public mental institution, under Max Müller, became the world centre of insulin coma therapy, while Sakel fled from anti-Semitic persecution to New York. Meanwhile, further down the Danube, another assault on schizophrenia was underway.

Speculation about the relationship between schizophrenia and epilepsy had been brewing for some years. Gyula Nyïro and Jablonsky observed in 1929 that epileptics with psychotic features became lucid during periods of frequent fitting; in 1930 Max Müller reported two cases of schizophrenia improving after epileptic fits; then Glaus remarked in 1931 on the near absence of epilepsy in 6000 schizophrenics (Kennedy, 1937). Ladislaus von Meduna at the Brain Research Institute in Budapest, on observing neurological differences between epileptics and schizophrenics in autopsy, postulated a theory of incompatibility in these conditions. In 1932 his colleague, Nyïro, transfused schizophrenics with the blood of epileptics, without success, but Meduna (1934) had the idea of inducing fits with an analeptic agent. He chose camphor, which had actually been used before in mental disease. In his *Medizinisches Praktisches Handbuch* of 1798, Weickhardt had recommended its administration in lunacy to the point at which seizures occurred (Kennedy, 1937), but such clinical application was an obscure historical artefact until its theoretically-grounded twentieth-century revival. After trials on guinea pigs, Meduna gave an intramuscular dose to a chronic schizophrenic on 23 January 1934 at the Royal State Mental Hospital in Budapest. Unfortunately the composition and speed of action of camphorated oil were inconsistent. A camphor-like preparation, pentamethylenetetrazol, a cardiac stimulant, produced by Knoll under the trade name Cardiazol (Metrazol in the USA), was procured. This substance was easily soluble in water, making it suitable for intravenous use, and was rapidly excreted, thus producing few toxic effects. Results from Meduna's (1935) first 26 patients showed that 10 had completely recovered. Fellow Hungarians Angyal and Gyárfás (1936) provided further evidence, and soon a flood of accounts of convulsive treatment appeared in the German literature.

Having broadly the same indication, Cardiazol and insulin each had their advocates, but some practitioners strove to maximize therapeutic potential by administering the methods in synergy. Angyal and Gyárfás (1936) found that alternating treatment enhanced remission rates in schizophrenics responding

poorly to Cardiazol alone. Although Sakel recognized the epileptiform convulsions that frequently occurred during insulin coma as potentially beneficial (Wyllie, 1938), he did not pursue this. Others took more interest. After observing that spontaneous seizures improved outcomes of insulin therapy, F. Georgi (1937) developed 'summation treatment'. His method, whereby Cardiazol was administered during hypoglycaemic coma, was one of several presentations at an international congress on Cardiazol, insulin coma therapy and deep sleep treatments at Münsingen in May 1937, organized by Müller (Schweizerischen Gesellschaft für Psychiatrie, 1937). This event, with its report translated for the English-speaking world, was a springboard for a zealous physical onslaught on schizophrenia.

Introduction in British mental hospitals

At the time of these developments British psychiatry remained stifled in the austere setting of vast custodial county mental institutions yet to discard their stigmatized association with the Poor Law. Although the 1930 Mental Treatment Act had facilitated better provisions for treating early cases, the new admission units, while detached from the main block, were immersed in the Victorian asylum estate. The Maudsley Hospital was a step towards the progressive German model of a university-linked psychiatric clinic, but elsewhere a stultifying regime prevailed. The monotonous ardour of mental doctoring carried little prestige, and the profession was lacking in academic prowess (Anonymous, 1936). While the search for pathological agents continued in mental hospital laboratories around the country, genetic determinism remained a prominent theme, and eminent members of the profession were active in the eugenics movement. Not that psychological aspects were neglected: the standard postgraduate textbook by David Henderson and Robert Gillespie (1936) adhered to Adolf Meyer's psychobiological approach, defining mental disorders as 'reaction types'. Occupying a middle ground between the organicist and Freudian paradigms, this was common-sense psychiatry based on observation of the psychophysiological constitution and environmental factors of each case (Gelder, 1991). However, while psychiatric training promoted individualism, opportunities to practise accordingly were severely restricted by miniscule doctor to patient ratios.

Existing clinical treatments were not illness-specific but aimed at ameliorating troublesome behaviour or symptoms. Crude drugs dulled acute disturbance at the cost of toxicity and dependence, while the barely palliative hydrotherapy remained in vogue. Joel Braslow (1997) in his thesis on somatic treatment rejected the notion that psychiatrists prior to shock therapy saw their therapeutic repertoire as meagre, but there was no illusion that baths or bromides cured mental disorder. The launch of the new physical methods promised actually to tackle the underlying illness – with the corollary of a much-needed boost to professional esteem. The drastic nature of these procedures conflicted with

the prevailing caution of British medicine (Kennedy, 1940): insulin treatment risked irreversible coma, while Cardiazol induced major seizures that Andrew Wyllie (1938: 271) described as: 'to the novice greatly alarming'. Yet failure to grasp these opportunities would condemn schizophrenics to a miserable life in the back wards; in the 1930s the census of British mental hospitals was rising by two thousand per year.

Sakel's innovation reached British shores in 1935, Herbert Pullar-Strecker receiving a Medical Research Council grant to commence the treatment at the Royal Edinburgh Hospital. In 1936 the Board of Control, normally confined to the drudgery of its role as mental hospital inspectorate for England and Wales, took the progressive step of sending one of its members, Isobel Wilson, to Austria to report on this novel treatment (Wilson, 1936). In England insulin coma therapy was first used at Moorcroft House, a private hospital in Middlesex, where would-be champion of physical treatments, William Sargant (1967), enlisted the help of Rudolf Freudenberg from Vienna. Moorcroft House also appears to have hosted the first application of Cardiazol treatment, probably in early 1937. Freudenberg and colleagues, noting that incidental seizures during insulin coma therapy boosted prospects of recovery, decided to supplement the regime by administering analeptic shocks on days when insulin was omitted (James, Freudenberg and Cannon, 1937). Two brief reports in *The Lancet*, 17 July 1937, suggest that the first public institution to introduce convulsive therapy was either West Ham Mental Hospital in Essex (Gillies, 1937) or Three Counties Hospital at Arlesey in Bedfordshire, where several months of use were reported by deputy superintendent Leonidas Finiefs (1937). Finiefs had begun using Cardiazol as an adjunct to insulin, after observing the latter treatment in Vienna. In April 1937 the London County Council funded a visit to Budapest by Leslie Cook, deputy medical superintendent at Bexley Hospital (1937), to observe the treatment.[1] A dedicated Cardiazol ward was established on his return, and the authorities initially considered designating Bexley as a treatment centre, although the simplicity of the procedure made such specialization unnecessary. In its Annual Report for 1937, the Board of Control (1938) noted that convulsive therapy was proceeding at several institutions. In autumn 1937 they sent Wilson and W. Rees Thomas to Hungary, although their report was not published until the following summer (Thomas and Wilson, 1938), by which time convulsive treatment was already widespread.

The exciting advances in treatment raised lively discussion at the annual gathering of the Royal Medico-Psychological Association at Ilkley in July 1938 (RMPA, 1938). Sir Laurence Brock, chairman of the Board of Control, described it as one of the most memorable meetings in the history of the association. Some dissent towards Cardiazol was heard, one member fearing an iatrogenic community of epileptics (RMPA, 1938), but the momentum could not be quelled. A Board of Control survey in late 1938 (published 1939) showed that shock treatments had commenced at 92 institutions, of

which 89 were using Cardiazol. Meanwhile, only 31 had introduced insulin therapy. While Cardiazol was readily embraced by British psychiatrists, introduction of insulin coma therapy, despite Wilson's encouraging report, was tentative.

As both treatments targeted the same condition, implementation was heavily influenced by expedience. Commencing convulsive therapy was relatively straightforward, with free trial supplies obtainable. By contrast, the complex insulin coma procedure entailed significant resource implications, placing great demand on understaffed hospitals. Whereas convulsants were given to large numbers of patients three times per week, each administration taking the doctor a few minutes, the insulin method required daily treatments and constant medical attendance for five hours (Pullar-Strecker, 1938). Strict gender segregation demanded special units on both male and female sides. The London County Council limited the treatment to selected hospitals. Edward Mapother, head of the Maudsley Hospital, initially barred insulin treatment on the grounds of its risks, eventually permitting it under supervision of a Swiss expert in December 1938 (Sargant, 1967). Insulin shock therapy was further thwarted by World War II, due to workforce depletion as staff left for military service, space curtailments after entire institutions were commandeered by the War Office, and sugar shortages (James, 1992). Some examples are: at Brentwood in Essex, insulin coma therapy was introduced in 1946, eight years after convulsive therapy (Nightingale, 1969); Haywards Heath Mental Hospital commenced Cardiazol treatment in 1939, and insulin coma therapy in 1945 (Gardner, 1999); and, as stated in the 1946 report of St Andrew's in Norfolk, insulin coma therapy was 'not yet available at this hospital, owing to the shortage of nursing and medical staff' (McCulley, 1946). Indeed leucotomy, a swift singular psychosurgical operation, arrived at many British mental hospitals before Sakel's procedure.

The procedure

The technique of Cardiazol treatment was described in detail for a British audience by Alexander Kennedy in *Journal of Mental Science*, November 1937. As well as presenting methodological guidance from European pioneers, Kennedy referred to his own early experience as assistant medical officer at West Park Hospital. Production of fits – the *raison d'être* of treatment – depended on the substance rapidly reaching the central nervous system, as there was little cumulative action. A wide-bore needle was pushed 2 cm into the vein to avoid leakage resulting from the high pressure at which the solution was injected. Almost immediately, colour drained from the patient's face, which became stiff and motionless. Onset of seizure was signalled by a cough or cry, before tonic contractions began. A peculiarity of Cardiazol convulsion was a sudden yawning spasm, at which an attendant inserted a gag to avert dislocated jaw. As tonus increased, the first clonic

jerks appeared. The pupils would widen and stare. Clonic spasms lasted around 40 seconds, the patient being protected from injury by manual restraint. Incontinence was common. Patients then fell into a comatose sleep for about ten minutes, their recovery monitored by attendants.

Optimum dose for each patient was found by trial and error. The needle remained *in situ* so that if no fit occurred, a back-up dose could be delivered. Concentrations were limited to a 10% aqueous solution to prevent sclerosis, but repeated injections tended to obliterate veins. Intramuscular administration was advised where veins were unsuitable or in resistive patients. Technical difficulties and the unpleasantness of Cardiazol therapy led to the use of an alternative substance, cyclohexylethyltriazol, branded by Boehringer as Triazol. Tested at Cane Hill Hospital in 1938 by Willi Mayer-Gross and Alexander Walk (1938), Triazol proved less awkward, without noticeable difference in the nature of fits, and several practitioners switched to this compound (e.g., Atkin, 1939).

Problems in evaluation

'That man is different', Rees Thomas exclaimed to Isobel Wilson as a patient was carried in for his second treatment, obviously more alert than on the previous day (Wilson, 1963). Meduna, bathing in international publicity as he performed for his British guests, had achieved startling results in a stubborn condition that for so long had defied psychiatric intervention. Impressed though Thomas and Wilson (1938) were, they issued caveats about the problems in critically evaluating Cardiazol treatment. Wary of excessive optimism, they attempted a balanced appraisal, beginning by reviewing recovery rates from schizophrenia with conventional hospital treatment. It had been relatively easy for Wagner-Jauregg to attribute remissions to his malarial therapy, because untreated general paretics rarely departed from a terminal course, but for Cardiazol the matter was less clear-cut. It was known that catatonic syndromes, marked by episodes of excitement, depression and stupor, sometimes occurred as a single attack followed by full recovery. Published spontaneous remission rates ranged from 5% to 60%, averaging somewhere in between.

Meduna informed Thomas and Wilson that with Cardiazol he had achieved 45% remissions overall, rising to 75% in cases of less than six months' duration. Time of onset was a crucial variable, with an inverse relationship emerging between duration of illness and positive outcomes. Meduna urged treatment as soon as symptoms appeared, rather than wasting time in confirming a diagnosis. Equally important was that results differed considerably between schizophrenic syndromes: a consensus emerged at the Münsingen conference that catatonics fared best with Cardiazol, whereas paranoid types benefited most from insulin coma. As some practitioners were more selective than others, not surprisingly results varied from spectacular to

mediocre. Unhelpfully, classification by duration and nature of schizophrenic illness was often absent in published reports. Inconsistent outcome measurement exacerbated the difficulty in deciphering the results. A plethora of categories were devised, detracting from comparative analysis. While many writers adhered to Müller's classification (complete, partial and incomplete remission, and unimproved), some practitioners based verdicts on whether patients had been discharged; others identified remission on the merest sign of social improvement. Reports typically related to a small series of cases, with tabular presentation lacking indications of statistical significance. Input of statisticians was not always welcomed: at the RMPA meeting in 1938 some members (e.g., Hunter Gillies) resented such interference in clinical matters. Yet the most serious shortcoming in evaluation was the degree of subjectivity exercised by doctors themselves: as Thomas and Wilson acknowledged, keen exponents were liable to be swayed by their own enthusiasm.

Impact on schizophrenia

The first British papers specifically relating to Cardiazol therapy were early findings in *The Lancet* in July 1937 by Finiefs at Arlesey, who reported 'fairly good results' (1937: 131), and in the same issue by Gillies, deputy superintendent at West Ham, who described improvement in a delusional woman who had failed to respond to insulin treatment (1937: 132). Kennedy (1937), reporting on three cases treated at West Park and reviewing results from abroad, suggested that it compared favourably with insulin shock therapy, while acknowledging that permanence of remissions was yet to be proven. At the time of writing their monograph Thomas and Wilson (1938) thought it too early to discuss findings from British hospitals, although they included preliminary results from West Ham, Bexley, Arlesey and West Park. Soon an abundance of reports on Cardiazol use were published in Britain, not only in specialist journals but also in the general medical bulletins; *The Lancet* featured eleven papers on Cardiazol and Triazol from 1937 to 1939.

Several British writers doubted Meduna's theoretical basis for Cardiazol treatment, but what mattered were its results. Having given over 1450 shocks, Cook opened his account of Cardiazol to the Royal Society of Medicine in January 1938 with the assertion that 'its value in schizophrenia is no longer in doubt' (Cook, 1938*a*: 567). He told of initially sceptical nurses being won over by the results. He described a 19-year-old girl who for nine months had displayed schizophrenic disorder manifested by detachment from reality, vivid hallucinations, bizarre delusions, impulsive aggression, repeatedly throwing herself on the floor, and dirtiness. After 21 fits she was discharged completely sane, after expressing gratitude for her treatment. Geoffrey Nightingale, who commenced convulsive therapy in September 1937 at Brentwood, reported outcomes in 44 schizophrenics in May 1938. While confirming that best results were obtained in recent admissions of

catatonic presentation, he noted some amelioration in advanced cases. Early findings from 28 patients, including 20 chronic schizophrenics, were presented by Wyllie (1938) at Crichton Royal. Finding physical and mental improvement in the majority of cases, he opined that Cardiazol would supersede insulin shock treatment, mainly because of its convenience and safety.

Early enthusiasm was tempered by the disappointing tendency for relapse. Nightingale (1938) reported a girl who received 33 fits, all with good effect, only for her mute state to resume a few days after each course completion. Assessing results at Bexley, Arthur Harris believed that while convulsive therapy arrested progress of the illness and hastened recovery, it had no significant impact on the course of schizophrenia. He supported its use on the grounds that 'our therapeutic resources for the combating of schizophrenia are so poor that we cannot afford to neglect any palliative' (Harris, 1938: 766). Pullar-Strecker (1938), reviewing European results, found that Cardiazol produced partial or complete remissions in 37% of schizophrenic cases, compared with 40% with insulin treatment. He warned against relying on fits or comas to cure mental disorder, and that as the difficulty was in maintaining improvement, other aspects of hospital treatment should not be neglected. In the Board of Control's nationwide survey of physical treatments, data collated on 2875 completed Cardiazol cases showed that 31% had been discharged, compared with 44% of 476 insulin cases. Although 46% of Cardiazol recipients were unimproved, the Board (1939: 38) remarked that 'until better methods are available there is justification for continuing to use those which we have'. To boost remission rates, Georgi's summation therapy was applied at some British hospitals, Leonard Russell (1938) at St Bernard's Hospital extolling its virtues over Cardiazol or insulin alone

Francis Reitmann (1939) at the Maudsley, who had worked under Meduna, collated worldwide results of shock therapy in schizophrenic cases of less than 18 months' duration. Claimed recovery rates with Cardiazol averaged 52%, higher than achieved with insulin coma therapy, but varied from 39% in Germany to 79% in Italy. English results were at the lower end of the international scale, at 41%. However, the extent to which positive outcomes could be attributed to Cardiazol was debatable. A comparative study by Harry Stalker and colleagues (1939) of insulin, Cardiazol and standard care at the Royal Edinburgh Hospital revealed little difference in outcomes. After emigrating to the USA, Meduna accepted that Cardiazol was no panacea for schizophrenic illness, but maintained that it stimulated remission in patients with good prognosis (Meduna and Rohny, 1939). Pollock (1939), however, concluded from a survey of New York institutions that Metrazol therapy for schizophrenia in US state hospitals was not justifiable.

With disappointing results in schizophrenia overall, indications for Cardiazol increasingly narrowed. Attributing wide variation in results to conceptual problems in diagnosis, Ian Skottowe at Bucks Mental Hospital devised a

symptomatic typology of schizophrenia for better targeting of shock therapy. While 'dyskinetic' and 'simple paranoid' cases responded well, for the 'dys-symbolic' type, which equated to the primary dementia of earlier nomenclature, such treatment was 'a waste of time, money and skill' and 'without exception futile' (Skottowe, 1939: 851). This was confirmed by Sawle Thomas (1940) at Littlemore, who found that of 32 dys-symbole cases treated with either Cardiazol or insulin, none recovered. Meduna had recommended at least 25 fits before giving up a case as hopeless (Thomas and Wilson, 1938), but Donald Blair at Cane Hill advised that a course should never exceed ten injections, terminating after two convulsions if improvement was achieved. With only four of 104 cases of over six months' duration showing any benefit, Blair (1940: 473) argued that 'its indiscriminate use is ... deeply to be deplored'. Archibald Bain (1940), after observing deterioration in nine of 16 chronic schizophrenics treated at Leicester Mental Hospital, suspected that adverse outcomes were more common than the literature suggested. He explained that for the schizophrenic, phantasy was preferable to reality, and that a disturbing stimulus was likely to do more harm than good.

Some clinicians persevered with the treatment in advanced cases, despite little prospect of remission. Despite only three of 68 chronic schizophrenics recovering, Wyllie (1940) believed that convulsive therapy raised social performance in half of these cases. According to Isaac Atkin (1939) at Park Prewett, the treatment made wards more manageable for nursing staff, while Campbell Young (1938) at Hull Mental Hospital reported marked improvement in the habits of the wet and dirty class of patient. However, Bain (1940) warned against judging outcomes merely on the basis of conduct. He described a patient, considered to have improved, who had previously pestered doctors for his discharge. On closer examination he had simply lost interest in his future, having been crushed by the volley of repeated convulsion treatments. Such material implies a departure from original curative goals to what Braslow (1997) termed 'therapeutic discipline', whereby illness was redefined in terms of behaviours altered by treatment, and supports sociological interpretations of somatic therapies as devices of control (Scull, 1994).

While doubts grew over the merits of Cardiazol, insulin coma gradually gained ascendancy in the treatment of schizophrenia. Analysing results at Moorcroft House, Freudenberg (1941) found no factors about schizophrenic cases that reliably predicted an outcome of convulsive or hypoglycaemic treatment, but he conferred with Müller's view that the latter produced a more lasting effect. Insulin was thought to influence the underlying thought disorder, and courses could be repeated as maintenance therapy. Cardiazol, however, was a useful first line of attack (Cook, 1944). Freudenberg (1941) used it to produce rapid improvement in acute paranoid states, excitement or catatonic stupor, followed if necessary by insulin. Moreover, with many

British mental hospitals unable to establish insulin units, convulsive therapy remained the somatic treatment of convenience, if not efficacy, for schizophrenia.

The true value revealed

In the absence of an established therapeutic mechanism, the basis for a specific action on schizophrenia was weakened. The first report of an alternative application came from Verstraeten (1937) in Belgium, who declared that convulsive therapy produced most benefit not in schizophrenia but in affective psychoses, particularly depressive states. In fact, practitioners everywhere had been experimenting beyond the original target condition. Widening the net to treat cases of mania, depression and hysteria, Cook (1938*b*) concluded that convulsive therapy removed morbid reaction states, whatever the diagnosis. Harris and Birnie (1938) at West Park found positive outcomes in stuporous cases whether schizophrenic or depressive. Convulsive therapy appeared particularly valuable in involutional melancholia, as, unlike the self-remitting tendency of other depressive disorders, sufferers commonly slid into perpetual gloom (Cook, 1944). Agitated suicidal cases could be promptly and effectively treated, thus reducing the burden of close surveillance. Just five seizures sufficed to eradicate depressive illness and, in contrast to schizophrenia, recoveries were sustained. The treatment could correct swings to either extreme of affect. In April 1938 Low, Sonenthal, Blaurock, Kaplan and Sherman in Illinois reported good recoveries at either pole of manic-depressive psychosis regardless of duration of illness, findings replicated in Britain by Wyllie (1940).

As the principal value of convulsive therapy emerged, some commentators attributed discrepancy between initial and later results to anomalous case definition. Good (1940) suspected that manic-depressives had been erroneously identified as schizophrenics, while the depressions most amenable to Cardiazol were those with psychomotor retardation descending into stupor, a condition shared with catatonic schizophrenia. Although Meduna had conceptualized schizophrenia as a concrete disease entity, in reality this was a diagnostic construct with arbitrary boundaries, liable to variations in diagnostic practice between countries and cultures. In the 1930s, British psychiatrists continued to use the labels 'primary dementia' or 'dementia praecox'. While some patients obviously fitted the simple or 'true' form of the condition, in catatonic cases doctors may have waited until a series of episodes had occurred before confirming a diagnosis that usually led to lifetime incarceration. Unlike the fixed degenerative process described by Kraepelin, Bleuler's (1911) psychodynamic reformulation was based on his belief that the minds of sufferers were in a state of disorganization, and that certain types (particularly catatonic) were amenable to full recovery. Bleuler's term had a more elastic circumference, and it is probable that Meduna's early successes resulted from painting

schizophrenia with a broad brush in his haste to treat acute cases. Aubrey Lewis, in his recently unearthed report of a tour of European psychiatry in 1937, observed that Meduna displayed 'the common weakness of calling every anergic patient catatonic, so that some retarded depressions are likely to be diagnosed schizophrenic' (Lewis, 2003: 107).

The spectre of spinal fractures

Compared with insulin treatment, with its diabolical risk of irreversible coma, the Cardiazol procedure was relatively safe. Ten deaths from 3531 cases (0.3%) were reported in the Board of Control's 1938 survey (published 1939), considerably safer than the 1.3% mortality with insulin. Blair (1940) reported two deaths in 120 Cardiazol recipients at Cane Hill. Deaths were attributed to various causes, including embolism and cardiovascular accidents (Thomas and Wilson, 1938). An unanticipated problem was reactivation of tuberculosis, then highly prevalent in mental institutions. Harris and Binnie (1938) recommended radiography to exclude those with latent tubercular disease. An immediate peril was status epilepticus. The convulsant could reach dangerous levels, particularly when additional amounts were administered in response to lack of seizure, and by the intra-muscular route due to unpredictable rates of absorption. As Triazol was slowly eliminated, the risk of status epilepticus increased. Wyllie (1940) reported four deaths in 85 patients using this substance.

Most adverse events were directly related to the severity of seizure, which caused great stress on the musculoskeletal system. Joint dislocations and long bone fractures sometimes resulted from the extreme contractions. Larkin (RMPA, 1938) commented that fractures were more likely in long-stay patients, due to vitamin D deficiency in the hospital diet. However, it was only after a large number of patients had been treated that the problem of hairline fractures to the spinal vertebrae was discovered. The treatment came under fire at the American Psychiatric Association conference in 1939, with alarming evidence that 40% of patients had crush vertebral fractures (Sargant, 1967). A paper on spinal column damage by Dutch psychiatrists Kraus and Viersma in the *Journal of Mental Science* (1940) added to the disquiet. In defence, it was argued that most radiologically identified fractures were symptomless, while Cook and Sands (1941) believed that discomfort was of muscular origin, causing no permanent disability. However, the treatment's reputation was seriously tarnished, and it was discontinued in some British hospitals as a result.

A solution to the problem materialized in the USA, where Metrazol treatment was in danger of abolition. Walter Freeman, a prominent advocate of physical treatments, met Ohio psychiatrist Abram Bennett, who was using Metrazol to treat depression, but also using small doses of curare to relax the limbs of children with spastic paralysis. Freeman suggested that Bennett try

curare in greater strength to tame the seizure in convulsive therapy, and secured a bulk supplier of the substance in Ecuador (Sneader, 1985). Bennett (1940) found curare a sound premedication and, although higher doses of analeptic were necessary, the treatment was no less effective. However, curare, originally used by Indians as arrow poison, posed the risk of respiratory failure, its clinical and fatal dose being very close. In practice it proved more dangerous than the treatment itself: a disproportionately high mortality occurred in patients receiving curare injections (Kalinowsky, Hoch and Lewis, 1946).

There were also neurological sequelae. Practitioners generally saw amnesia as a merely temporary complication, and neither post-mortem examination nor studies in monkeys revealed significant destruction of nerve tissue (Cook, 1944). However, Dynes (1939) in Edinburgh observed lasting cognitive impairment correlating with treatment duration, and he opposed courses of over 20 fits. After finding amnesia in some cases six months after treatment, Tooth and Blackburn (1939) advised against Cardiazol use in patients who relied on intellectual function in their work. Wyllie (1940: 253) wrote: 'Damage to cerebral structure is of slight degree, and in persons of humble intellectual development is scarcely discernible. In highly educated persons, on the other hand, it is more apparent and might be serious.' Kennedy (1940), however, argued that the risk of permanent damage from prolonged treatment was outweighed by the benefits in combating severe mental disorder.

Fear before fit

A particularly objectionable feature of Cardiazol therapy was an intense dread experienced by patients between injection and seizure. Although patients had no recollection of the fit itself, they often recalled overwhelming alarm during the preceding aura. Reactions to the treatment, described by Thomas and Wilson (1938: 11) as 'very unpleasant', were candidly portrayed in the psychiatric literature, with several papers directly quoting from patients. 'The very thought of it makes me shrink with horror' uttered one of Gillespie's patients at Holloway Sanatorium (1939: 391). Kennedy (1937) advised that patients be deterred from discussing their treatment afterwards, although Good (1940) found repression of the trauma common. From accounts he gathered, Good described the process as a feeling of bewilderment on injection, with steadily building apprehension as the drug circulated, causing sensations of heat, flashing lights and burning matches; some patients felt that they were being electrocuted. Petrie at Banstead heard patients describe the convulsant spreading through the body as an experience 'worse than death' (RMPA, 1938: 687). Some practitioners downplayed the matter: Meduna observed no more than mild apprehension, although he recommended periodically interviewing patients for reassurance (Thomas and Wilson, 1938). Screens prevented patients from witnessing the treatment being given

to another, although to contain any fracas a single room was used for particularly resistive patients (Nightingale, 1938).

Gillespie (1939) subjected himself to a Cardiazol fit. Unlike most of his patients he did not recall the period before fading into unconsciousness as particularly disturbing, but the rest of the day he described as 'distinctly unpleasant', enduring headache, nausea and malaise (p. 391). Patients usually slept after coming round, were woken for lunch, then allowed to spend the afternoon in bed. Kennedy (1937) found that on the following morning patients often became excitable and irrational, running around the ward before reveille, shouting and quarrelling, removing their clothes, a few even attempting suicide; as patients recovered from this thought-disordered phase they grew resentful towards the treatment. Hostility was marked after injections had not culminated in seizure – as happened in around a third of treatments. Subconvulsive doses left patients in a confusional twilight state for several hours.

At the time shock therapies emerged, few ethical safeguards existed to protect patients from such brutal and basically experimental interventions (Musto, 1981). Doctors unilaterally decided what was best for patients, of whom most were legally certified as insane. Coercion for this fearsome procedure was barely disguised by writers. Meduna (1937) urged practitioners to complete the course, disregarding any protests en route. Bain (1940) described treatment of violently resistant patients being curtailed only after several injections, when lack of co-operation made perseverance impossible. Petrie told of patients doing 'dramatic things on the morning of the injection in order to evade it' (RMPA, 1938: 690). Good (1940) heard all kinds of excuses from patients, some putting up a physical struggle when their pleas were ignored; extreme efforts to escape the injection included jumping through the ward window, climbing on to the hospital roof, and suicidal acts. In his memoirs, Henry Rollin remembered reluctantly implementing the treatment at Narborough Mental Hospital, and the 'unseemly and tragic farce of an unwilling patient being pursued by a posse of nurses with me, a fully charged syringe in my hand, bringing up the rear' (Rollin, 1990: 69).

Nightingale (1938) found some patients so violent that injection was impossible without premedication. However, conventional stalwarts hyoscine or paraldehyde increased the likelihood of a negative injection (Sands, 1939), and some practitioners withheld tranquillizers even on the previous night, when perhaps most needed by apprehensive patients. Cook (1938*b*) administered morphine. At Claybury, Sands (1939) used insulin to induce drowsiness in patients displaying marked anxiety and resistance, while Lindesay Neustatter and Harry Freeman (1939) averted terror and subsequent hostility through general anaesthesia. Despite the slightly longer wait between injection and fit, patients found Triazol less unpleasant (Mayer-Gross and Walk, 1938). Horsley (1940) reported least distress with picrotoxin, a useful finding given his bold step of performing convulsive treatment on outpatients

at a Dorset general hospital, whose voluntary status might impede compliance. Cunningham Dax (1940) tried ammonium chloride at Netherne, but although well tolerated the fits were weak. Further search for a satisfactory alternative to Cardiazol were obviated by the arrival of the electrical method.

Psychotherapeutic openings

The rationale of shock therapy was firmly based within the biological paradigm, but few psychiatrists believed that bodily manipulation alone could resolve functional psychoses. By the 1930s the work of Freud and his followers had had considerable influence on psychiatric thought, and analytically-orientated practitioners regarded somatic treatment not as an end in itself but as a useful adjunct to psychotherapeutic work; narco-analysis was practised in a few British hospitals at the time. Pioneers of Cardiazol treatment noted that it made hitherto withdrawn patients accessible to talking therapy (Thomas and Wilson, 1938). Yet the target condition for Cardiazol was not generally regarded as fertile soil for such endeavour. Schizophrenia had a 'Cinderella' status in psychoanalysis, the Freudian school believing that transference, the essential medium for therapy, was unfeasible. Harry Stack Sullivan's school of interpersonal psychiatry and Frieda Fromm-Reichmann had pushed the analytic boat out to the uncharted seas of the schizophrenic mind – focusing on childhood experiences and underlying anxiety they presumed at the root of the disorder (Bellak, 1948) – but psychotherapy was generally regarded as futile or even harmful for such cases (Henderson and Gillespie, 1936). However, such was the dramatic impact of convulsive treatment that Bain (1940) wondered: could shock on the disorganized fantasy world of the schizophrenic have an effect similar to that of the alarm clock on the dreamer?

Cardiazol therapy hardly presented an ideal setting for psychodynamic practice, but the induced trauma and resulting emotional catharsis facilitated analytic insights in this seemingly impenetrable condition. Good (1940) at Glasgow Royal Mental Hospital considered post-convulsive states in terms of Freudian stages of emotional development: oral behaviour was exhibited by desperate efforts by patients to retain the mouth gag between their teeth; anal activity by faecal smearing; and various behaviours involving the genitalia were an expression of phallic impulses. He believed that convulsive treatment presented transference opportunities. The doctor enacted the role of both good and bad mother; Good found patients frantically clutching at his arm on receiving the injection. David Abse (1940), medical officer at Monmouthshire Mental Hospital, followed Freud's theory of anxiety in explaining responses to Cardiazol: repeated exposure to danger aided the process of repression, thus restoring the patient's ability to behave normally.

Emigrant Viennese analyst Isidor Silbermann performed analytic work with

patients undergoing shock therapy at Hatton Mental Hospital in Warwickshire. He observed that insulin and convulsive treatments had similar psychical actions, but while insulin had a gentle impact, he described Cardiazol as 'a violent thunderstorm bursting suddenly', giving 'a far more vehement shock' (Silbermann, 1940: 179). Cardiazol delivered the necessary force to disrupt the psychotic ego, each successive treatment continuing this process until it could not be reconstituted, thus explaining the correlation of recoveries with number of shocks. Silbermann observed the verbal content expressed in the pre-convulsive stage as regression. The sensation of impending death forced patients from their narcissistic state, as they begged for mercy. The post-shock stage he regarded as restitution. On awakening patients became euphoric, one saying 'Now I feel I am a new person, entirely changed, as though I had been born again' (p. 187). Patients emerged from their rigid defences to reappraise reality and adapt to their surroundings positively. In Silbermann's view, the repeated contradictory experiences of death and resurrection necessitated psychotherapeutic intervention between shocks.

Some commentators saw fear as the therapeutic agent, thus likening shock treatments to the surprise baths and swinging chairs of earlier eras (McCowan, 1938). Louis Cohen (1939) in Massachussetts tested this hypothesis by administering Metrazol slowly in order to produce fear but not fit. Most patients showed no improvement, but positive results were achieved when the same cases received the standard delivery. Cohen concluded that seizure was a necessary culmination of the therapeutic process. However, as a psychological mechanism his study failed to consider the different intensity of sensation from the conventional rapid injection. Studying 275 cases at Bexley, Cook (1940) claimed that apprehensiveness correlated inversely with remissions but, interestingly, he noted most dread in excited cases, while stuporous patients (for whom Cardiazol had greatest efficacy) were least troubled. Georgi (1937) had observed that it was only as patients surfaced from catatonic stupor that their increased awareness stimulated aversion to the treatment. Gillespie (1939) described a mute individual who, after apathy towards his first 20 injections, violently revolted, yelling for the police to end his terrifying ordeal. At Carstairs War Hospital, Good (1941) noted how military casualties, many of whom had performed bravely in armed conflict, exhibited pronounced fear towards Cardiazol therapy. He attributed recoveries to a displacement of primal psychopathological anxieties, whereby a generalized fear triggered by battle became focused on the specific threat of the treatment. An alternative psychological mechanism was fulfilment of desire for punishment, the treatment allowing patients to start afresh after being cleansed of their guilt. Silbermann (1940) suggested that Cardiazol shocks relieved the super-ego of its need to oppress the ego, although lasting benefit was unlikely in patients with engrained masochistic attitudes.

The attentions of a few practitioners aside, it seems unlikely that psychotherapeutic opportunities of Cardiazol treatment were pursued in the public

mental hospital setting. Patients may have received reassurance aimed at ensuring course completion, but elaborate therapeutic activity was precluded by practical and ideological constraints. Unlike central Europe and USA where psychiatry had embraced psychoanalysis, Britain remained a psychotherapeutic void, offering few treatment facilities beyond its backward system of understaffed, overcrowded county institutions. A schism existed between orthodox clinical practice and the analytic approach. Leading psychiatrists like Mapother dismissed Freudian formulations as conjectures lacking in clinical utility, while psychoanalysts rejected the division of mental disorder into distinct entities, a key foundation of modern scientific psychiatry (Pines, 1991). Few opportunities arose for medical officers to undertake the lengthy psychoanalytic training. Joshua Bierer (1940), psychotherapist at Runwell Hospital, argued that in terms of psychological input, therapeutic nihilism was rife. Despite underlining the importance of the psyche in a specialty that had tended to prioritize pathological and hereditary factors, the psychoanalytic revolution, by the time of Freud's death in 1939, had barely touched the inhabitants of Britain's mental hospitals.

While psychoanalysis produced little evidence to convince its sceptics, shock treatments allowed doctors to 'do things' to patients with proven effect. As an Australian writer asserted: 'at last there seems to be definite proof that insanity is a physical disease, cured by a physical agent', haughtily observing that 'psychoanalysts are left swirling in their own mephitic vapours, for how can they explain the fact that the hairy horrid complex which has proved impervious to their windy armaments is easily dissolved by a few drops of a chemical solution' (Barry, 1940: 437). Shock therapy bolstered the organic approach in psychiatry, the balance swinging from the 'art' of psychoanalysis towards the 'science' of physical treatment. Disillusioned by this trend, Sullivan (1940: 73) argued: 'These sundry procedures produce "beneficial" results by reducing the patient's capacity for being human. The philosophy is something to the effect that it is better to be a contented imbecile than a schizophrenic.'

Electroshock

For several years Ugo Cerletti, Professor of Neurology and Psychiatry in Rome, had been experimenting with electrically induced convulsions in studying animal models of epilepsy, when he and assistant Lucio Bini considered applying this process in convulsive therapy for schizophrenia. Testing the procedure on dogs, placing the electrodes in the mouth and anus of their canine subjects frequently caused death by electrocution, but administering the shock to the temple proved safe. They took their proposal to the Münsingen conference (Bini, 1937). Although electricity had previously been deployed in psychiatry (Beveridge and Renvoise, 1988), the idea of passing electric shocks through the brain seemed hazardous, especially considering the

strength of current necessary to produce seizure. Cerletti and Bini thus approached their first human subject with trepidation. A psychotic man found wandering in the railway station by police became the 'electroshock' debutant on 18 April 1938. The current was gradually increased until a classic epileptiform seizure was produced. Eleven shocks later, his hallucinations disappeared, and the treatment was pronounced a success (Valenstein, 1973).

The new technique, which became universally known as ECT (electroconvulsive therapy), offered several advantages over the chemical method. It was free of problems associated with venous administration, and as patients immediately lost consciousness when the shock button was pressed, terror was avoided. In England it was initiated at the Burden Neurological Institute, a newly opened clinic for investigation of neuropsychiatric disorders in Bristol, where director Frederic Golla, who had run the pathological laboratories at the Maudsley prior to its closure on the cusp of war, instructed W. Grey Walter to assemble the apparatus. After producing perfect fits in sheep, clinical use began in the Institute in 1939 and then in nearby mental hospitals (Cooper & Bird, 1989), where its technical convenience and better co-operation from patients (Fleming, Golla and Grey Walter, 1939) were confirmed. A key figure in establishing ECT was German psychiatrist Lother Kalinowsky, who had worked at Cerletti's clinic. Facing Fascist controls on Jewish doctors, he fled to Paris and introduced the treatment there. In July 1939 he took refuge in England, assisting Shipley and McGregor (1940) in commencing ECT at Netherne, before moving on to propagate the treatment in the USA.

Amid the controversy about spinal fractures, hospital authorities were understandably wary of this apparently more extreme mode of convulsive therapy. Sargant (1967) found the London County Council obstructive in his efforts to procure a 'shock box'. Teething problems arose with early machines, which often failed to produce a fit, instead leaving the patient with a severe headache (Rollin, 1990). Nonetheless, ECT transpired as a safer method. Hemphill and Walter (1941) reported just one fracture in over 200 patients. The seizures were less rigorous, and absence of pre-convulsive fear meant that patients' posture was less tense, leading to considerably fewer vertebral fractures (Kalinowsky *et al.*, 1946). Use of ECT in outpatient therapy, regarded as unfeasible with the chemical method, began at St Batholomew's general hospital in London (Strauss & MacPhail, 1940). Finding the best results in melancholic and manic patients, Hemphill and Walter (1941) acknowledged that the original conception of convulsive therapy as a treatment for schizophrenia was now abandoned, but commented that 'the possibility of improving the large numbers of old schizophrenics that fill the chronic wards of every large hospital is of special interest' (p. 273). Shipley and McGregor (1940) noted that ECT, unlike Cardiazol, could be used as maintenance therapy for relapsing catatonic schizophrenics.

Despite its rapid uptake, initially ECT did not immediately replace

Cardiazol treatment in British mental hospitals. Journal advertisements in the early 1940s primarily aimed ECT at affective disorder, while the chemical method was marketed towards schizophrenia. In their manual on physical treatments published in 1944, William Sargant and Eliot Slater endorsed Cardiazol in catatonic stupor and schizophrenics with depressive features. Joshua Carse, superintendent at Graylingwell, remained an enthusiastic proponent of Cardiazol throughout the 1940s. In an annual report he documented its use in 'states of acute confusion, agitation, or excitement accompanied by violent motor restlessness' (Carse, 1946: 19), particularly in severely disturbed patients admitted under Urgency Orders, deemed at risk of collapse from exhaustion. Apparently such patients failed to respond to ECT, but they were transformed by Cardiazol into a calm, co-operative state. At Runwell, Ström-Olsen (1945) continued to favour chemical convulsants in certain cases. American psychiatrist Dallas Pratt (1948), touring sixteen British mental hospitals after World War II, heard that, during 1946, 81 patients were treated with chemical convulsants at Graylingwell, and a few manic patients at Runwell, but elsewhere the treatment had been supplanted by ECT. Dax (1951) reported to a Paris meeting on shock treatments in 1950 that Cardiazol continued to be used at Netherne in cases of stupor and food refusal. In the mid-1950s a psychiatrist at Lancaster Moor promoted Triazol for treatment-resistant schizophrenia in *The Lancet*, lamenting disuse of this therapy elsewhere (Pakenham-Walsh, 1956). As a last remnant, in the final edition of their classic textbook, Sargant and Slater (1972) retained Cardiazol as an intervention for stuporous states, advising its injection during insulin sopor to avoid its notorious unpleasantness.

Use of convulsive therapy in schizophrenia declined after the arrival of neuroleptic drugs, but with rapid symptomatic action ECT maintained a place in the therapeutic arsenal. Sargant and Slater (1972) recommended ECT for acute schizophrenics with pronounced affective disturbance, advising lengthy courses of at least 20 shocks. Concerns have persisted about suspected long-term neurological damage, and allegedly indiscriminate use in mental disorder. Dramatized as a punishment tool in Ken Kesey's *One Flew Over the Cuckoo's Nest*, the perceived violence of ECT has aroused polemics from the anti-psychiatry movement. Despite surviving the rigour of randomized controlled trials, ECT remains shrouded in controversy.

Conclusion

The rapid and widespread implementation of Cardiazol therapy in British mental institutions was spurred by convincing claims that the great burden of schizophrenia might relent to a remarkably straightforward physical procedure. In the absence of independent controlled trials with sufficient follow-up, naïve optimism prevailed, until initial promise gave way to sobering reality. Methodological shortcomings in psychiatric evaluation were

underlined by the shock therapy debacle, which shook the profession from its reliance on empiricism and clinical intuition, to embrace the scientific method. Crucial to the evolution of convulsive therapy was case selection. Entailing constructs rather than concrete entities, psychiatric diagnosis is heavily influenced by variations in clinical outlook. Braslow (1997) explained how advocates of somatic therapy reframed illness: whatever symptoms a chosen intervention relieved became the principal diagnostic criteria, thus creating favourable circumstances for therapeutic success. Wagner-Jauregg informed Lewis on his visit to Vienna in 1937 that diagnostic criteria for schizophrenia had considerably loosened in recent years (Lewis, 2003) – just when shock therapy was transforming the psychiatric scene. In their rush for early treatment Meduna and others probably classified many affective states as manifestations of the alternating stupor and excitement of catatonic schizophrenia. Encouraged by positive outcomes in such dubious cases, they unwittingly pushed a schizophrenic salient into affective disorder. Skottowe's proposal (1939) to rebuild the subgroups of schizophrenia based on susceptibility to shock treatment further exemplified the conceptualization of disease around therapeutic action.

Yet Cardiazol treatment, and later ECT, clearly made some impact on schizophrenic illness. Valenstein (1973) explained that basically shock therapy produced an extreme stress stimulating a defensive physiological reaction associated with beneficial changes in mental state. Its *modus operandi* remains unclear, but Cardiazol effected a crude jolt that removed, albeit temporarily, the ravages of catatonic psychosis. Although its theoretical basis was discredited, the relationship between schizophrenia and epilepsy was never settled, and Meduna has been supported by renewed evidence of biological incompatibility (Anonymous, 1968). A plethora of theories emerged as to the physiological action of Cardiazol, typically involving oxygen, sugar metabolism or circulatory mechanisms (Skottowe, 1939), but none convinced. Observing hitherto withdrawn patients reaching out to those around them, practitioners of psychoanalytic perspective attributed recovery to ego reintegration. As postulated for insulin treatment, perhaps 'social remissions' resulted from enthusiasm displayed towards previously neglected patients. Just as Cardiazol was being abandoned, studies by Bianchi and Chiarello, and by Cheney, showed superior results in catatonic states compared with ECT (Kalinowsky *et al.*, 1946), suggesting that the undesirable aspects of Cardiazol – the fear and severity of fit – had therapeutic impact. As it was sometimes used to eradicate the 'faulty habits' of refractory cases (Cook, 1944), some patients may have been able to modify their behaviour to avoid further exposure. Henry Rollin reflected on Cardiazol as 'mediaeval torture' (Valentine, 1996: 80). Ethical stringencies would surely have prevented such a terrifying procedure today.

It would be mistaken to dismiss shock methods as a false dawn in the treatment of schizophrenic illness. The 'wonder drugs' of the 1950s, while

revolutionizing mental health care, transpired as no more curative: despite the 'insulin myth' controversy, Fink's robust trial showed that insulin coma matched chlorpromazine as a symptomatic intervention (James, 1992). It was the inconvenience and risks that consigned insulin treatment to history. Convenience has remained fundamental in shaping psychiatric treatment, as evidenced by the decline in psychotherapy and the growth of pharmacological interventions. The major somatic treatments of the 1930s, while following similar pathways from heroic cures to more selective use and eventually to abandonment as routine interventions for schizophrenia, laid the foundations for the ascendancy of the biomedical model. Meanwhile convulsive therapy has survived, albeit through serendipity, as a frontline tool of clinical psychiatry. That its true potential lay in affective disorder was no detraction from its significance: before shock treatment, the chief healer of depression was time (Lewis, 1934), and for severe forms of this disorder it delivers immediate effects unobtainable with modern medication. Undoubtedly psychiatry is indebted to Meduna for setting out on a path culminating in an empirically proven intervention that has surely rescued thousands from the depths of despair.

Acknowledgement

The author is grateful to Edgar Jones at the Institute of Psychiatry for valuable guidance.

Note

1. Several writers erroneously credited Cook with introducing the treatment to Britain. Although he played a prominent role, he did not begin Cardiazol therapy at Bexley until June 1937 (Board of Control, 1938).

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