A very British kind of social psychiatry*

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Social psychiatry, as represented in the work of Michael Shepherd, his pupils, and his colleagues at the Maudsley, all intellectual progeny of Sir Aubrey Lewis, is a very British kind of science. Lest this arouse your ire, implying an insult hurled at his betters in the mother country by a colonial parvenu, I hasten to add that "very British" refers to the best in your long and admirable scientific tradition in medicine: an unswerving commitment to empiricism, one which rarely moves much beyond its database; sober understatement, which makes no claim of possessing an exclusive path to salvation; sound methodology; and epistemological caution. It is aptly described by the adjective Apollonian, defined in my *New Shorter Oxford English Dictionary (NSOED)* as "serene, rational, self-disciplined".

I contrast this with what has been a dominant stream in American social psychiatry, one given to catch-phrases and slogans, innocent of data altogether. Its proponents were sure they knew the truth; what was there to learn? Indeed, the engineers of the movement in the federal government operated in such fashion as to assure that facts would not get in the way; available funds were expended in the doing, not the studying, despite the fact that the Community Mental Health Act had required a 5% set-aside for evaluation. Community psychiatry issued claims about prevention; when these promissory notes could not be redeemed, public disenchantment resulted. If only, they cried plaintively, if only the world be given in to our charge, we will fill it with mental health. It does not seem inappropriate to term this movement Dionysian, defined in the *NSOED* as "ecstatic; inspired by instinct and emotion".

Obviously, this contrast between British and American social psychiatry is overdrawn and oversimplified. You had your Dionysians, we our Apollonians. Jones (1968, p. 80) defined social psychiatry as "an elastic concept, to include all social, biological, educational and philosophical considerations which may come to empower psychiatry in its striving towards a society which functions with greater equilibrium and fewer psychological casualties". Ingleby's (1980) *Critical Psychiatry* dismissed psychiatrists as "servile functionaries" of the state, and derided them for medicalising deviant behaviour in order "to divest such behavior of any political significance". Laing's "apocalyptic message . . . bracketed the mentally ill with the criminal, the sexual deviant, and the political dissident in a coalition of oppressed bearers of an authentic statement about the human condition" (Clare, 1992). At the same time, on our side of the Atlantic, sober voices expressed doubts about the new evangelism: "How are we going to take preventive action if we are still uncertain about causes?" asked Dunham (1965). Kubie (1968) warned of the pitfalls of abandoning patient care in the name of community psychiatry. Good intentions, I pointed out, do not assure good results (Eisenberg, 1968). Without systematic research on the effectiveness of new programmes:

> we will face a succession of psychiatric 'revolutions,' each of which will be based on the rediscovery of moral treatment but none of which will have advanced beyond the starting point of its predecessors.

It is, of course, grossly unfair to dismiss the social critics so cavalierly. They called attention to real problems; they were angry at injustice. They borrowed a sociological hypothesis known as labelling theory (Lemert, 1951) to call into question the very existence of the disorder we call schizophrenia. The original theory emphasised a distinction between 'primary' and 'secondary' deviance. Primary deviance is an attribute of the individual, a trait which may be pathological or no more than a normal variant. Once others label that attribute as a stigmatised defect, the label itself initiates a cascade of transactions in the course of which the patient's self-concept comes to be dominated by the invidious label and he is socialised into a role that becomes a self-fulfilling prophecy. The 'anti-psychiatrists' of the 1960s and 1970s contended that 'diagnosis' is a social process designed for the convenient disposition of people who make trouble.

Two observations lent credence to labelling theory. Psychiatrists in the former Soviet Union acted as agents of the State to diagnose dissidents as schizophrenic by taking their political heresy as *ipso facto* evidence of mental disorder. This phenomenon is neither modern nor limited to totalitarian states. Barker's (1991) remarkable novel, *Regeneration*, recreates the story of how the poet Siegfried Sassoon was labelled 'shell-shocked' by the military after he wrote a letter to the London *Times* attacking the folly of the First World War. His insubordination demanded official response; because he had been awarded a medal for bravery, he could not be peremptorily dismissed as a coward. The credibility of his anti-war beliefs was undermined by attributing them to combat-induced shell shock and returning him to England for psychiatric hospitalisation. The second sort of evidence supporting the validity of labelling theory in contemporary psychiatric practice was the discovery in England (Wing & Brown, 1970) and in America (Gruenberg, 1967) that many of the symptoms displayed by patients who had been hospitalised in impersonal and faceless institutions were superimposed by the institutional environment on top of the disorder which had led to the admission.

The hypothesis that labelling can initiate a process of secondary deviance is important and correct. But the radical critics ignored the role of the primary deviance in the sequence and failed to consider its determinants. Would ignoring the primary deviance allow it to fade away? A longitudinal study of mental patients and their families by Clausen (1981) led him to conclude that "their [patients'] feelings of stigmatisation are not so much a consequence of the response of others to their having been hospitalised for mental illness or labeled mentally ill as of self-doubts and chronic manifestations of mental illness".

As to the social activism so prominent in the community movement, let us assume, for the sake of discussion, that unemployment leads to increased rates of depression.

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What are the implications for social policy? Inveighing against unemployment on health grounds rather than economic grounds does little to rally supporters. After all, few contend that unemployment is good for health; most agree that high levels of unemployment are undesirable. The issue is how to decrease unemployment, a topic on which we have no claim as experts. As citizens, health professionals do have a moral responsibility to fight against the social conditions that impair health. In the words of Ryle (1943):

> Many of the social evils, so widely manifest by disease . . . call not for medical action but for drastic social and economic reform. For these the electorate through their representatives, and not the doctors (as doctors), must become responsible. But who unearths and exposes the evils? . . . We have reached a time in which 'the physician (to quote Professor Sigerist) must assume leadership in the struggle for the improvement of conditions' . . .

Sigerist, Professor of the History of Medicine at the Johns Hopkins University, was one of my medical heroes. Michael Shepherd (1990) traced the origin of his own interests in social psychiatry to John Ryle, the founding Professor of Social Medicine at Oxford in 1943.

Why was it necessary 50 years ago to invent social medicine – more accurately, to rediscover what Virchow, Neumann and others had discovered a century earlier (Rosen, 1947) – and to create a department with that name?

The importance of social context for health status had to be re-learned because the spectacular triumphs of bacteriology in the last decades of the 19th century completely pre-empted medical vision. The identification of specific micro-organisms as the cause of one after another of the infectious diseases that had plagued mankind led to the conception of disease as an entity which invaded the body from without. Although public health initiatives for pure water and sanitary waste disposal had begun in the 18th and 19th centuries, well before pathogenic bacteria were discovered, the rationale for such measures shifted from eliminating unsightly wastes and noxious fumes to control measures for particular disease-causing agents. By the turn of the century, Paul Ehrlich was writing about "selective affinities" between certain cells and certain dyes. The search for "magic bullets" began: lethal chemicals with a high tropism for invasive bacteria. By the middle of the 20th century, effective chemotherapy seemed to guarantee the conquest of disease. Medical vision limited its focus to disease pathogenesis; it became blind to the social context in which disease became manifest.

Yet, there had been a marked reduction in morbidity and mortality well before the new basic sciences yielded effective treatments. Mortality from tuberculosis in the UK had fallen by half between 1840 and 1880, two years before Koch discovered the tubercle bacillus; it fell by more than half again by 1940, a decade before the introduction of effective chemotherapy (McKeown, 1976). Similar trends occurred with other infectious diseases well before biomedicine appeared on the scene (Kass, 1971). The observed improvement in health had resulted from reduced exposure to pathogenic bacteria by the provision of pure water and sewage disposal, better personal hygiene, less-crowded housing conditions, and the isolation of infectious cases, on the one hand, and from improved host resistance secondary to better nutrition and general health, on the other. These gains were made possible by higher living standards accompanying the industrial revolution and by the political struggles for human rights which had accompanied it.

The very changes in the mode of life of ordinary citizens that led to the observed decline in mortality also resulted in massive shifts in disease prevalence. Diabetes provides a telling example. It appears to have originated from relative affluence; the very same 'thrifty genes' that protect against intermittent starvation become pathogenic on a rich diet (Neel, 1969). Further, the clinical course of diabetes, once a relatively acute and rapidly fatal disease, has been transformed into a chronic disease by the discovery of insulin in the 1920s, the development of renal dialysis in the 1950s, and the availability of renal transplantation in the 1960s.

The response to medical treatment is in itself a profoundly psychosocial phenomenon. Consider the findings from a randomised double-blind clinical trial of clofibrate, a drug administerred to lower cholesterol levels and, thus, to reduce mortality from coronary heart disease (Coronary Drug Project Research Group, 1980). The investigators measured compliance by checking the number of pills left in the container each time they issued a new supply. Among the 1100 men in the clofibrate arm of the study, those who took their pills more than 80% of the time had a significantly lower five-year mortality (15%) than those who took them less often (24.6%). These data suggested that clofibrate is a highly effective drug when taken as directed. However, among the 2800 men in the placebo arm of the study, those who took their placebo religiously also experienced a significantly lower five-year mortality (15.1%) than did the poor compliers (28.3%). Clofibrate, as such, had no effect on mortality, but compliance did, at a P value of 10^{-16}! The researchers limited themsleves to the lament that "these findings . . . show the serious difficulty . . . of evaluating efficacy in sub-groups determined by patient responses . . . ". They did not pursue the reduction in mortality associated with compliance, an effect so significant that it would have caused the stock of a pharmaceutical company to soar had the difference between attributable to the medication. What were the behavioural correlates of compliance? Changes in cigarette smoking, alcohol consumption, diet, exercise, other health-related behaviours? We will never know – none was measured.

The diseases that afflict men and women are determined by how they live, where they live, with whom they live, what they do and the resources they command. Virologists may insist: "Social context be damned! There can be no measles without exposure to the measles virus!" Yet, whether the virus succeeds in producing an infection in an individual depends on whether he/she is immune to the disease through vaccination or prior infection. That, in turn, may depend, in the USA, on the religious affiliation of the child's parents and on their economic circumstances. Religion is relevant in so far as fundamentalist groups refuse routine immunisations so that their children remain suceptible; socio-economic status applies in so far as rates of immunisation are lower among the poor because public programmes fail to reach them. The course of the disease in a given patient depends upon host resistance. The case fatality rate for measles in the USA had fallen to less than 1 in 100 000 well before immunisation was introduced (Langmuir, 1962); yet, it can be as high as 10–12 per 100 in infants in tropical Africa because of crowding, which results in massive exposure, malnutrition, and comorbidity, all of which increase vulnerability (*Lancet*, 1976; Walsh, 1983).

If we move in time instead of space, human culture created the conditions necessary for infectious diseases to exert selective evolutionary pressure on the biology of man (Haldane, 1956/7). Diseases that are infectious only in the acute phase, such as

measles or poliomyelitis, *could not become* endemic in neolithic populations (Black, 1975). The penetration of such a virus into a small hunter–gatherer community of several hundred behaves today as it did 50 or 100 thousand years ago; it rapidly kills or immunises so high a proportion of the population that the virus is no longer able to propagate itself and disappears until the next encounter with strangers. Contact between European and Amerindian populations during the colonial expansion led to severe mortality among the indigenous population because of infectious agents Europeans brought with them. In turn, part of the reason for the greater mortality was the restricted gene pool among the inbred aborigines. A virus which grows in one host is 'pre-adapted' to a genetically similar host and thus gains in virulence. Because of limited polymorphism at many loci among previously isolated peoples, exposure to mutable pathogens wreaks havoc (Black, 1992). Only when the agrarian revolution generated resources sufficient to permit the large-scale aggregation of human groups did the infectious agents have a host reservoir large enough to maintain the chain of transmission.

This long, I hope not too long, excursion into population medicine is intended to persuade you, first, that all medicine is social and, second, that this is not an imperialistic claim. I want my biomedical colleagues to know that they have been doing social medicine all along, much as Molière's middle class gentleman spoke prose without knowing it! I hope they will be as delighted at the discovery as he was.

If all of medicine is social, the argument applies to psychiatry *a fortiori*. Yet, when my friend, Sam Guze (1989), entitled his Royal College Lecture: 'Biological psychiatry: is there any other kind?" he meant the answer to be: "Of course not" and he was absolutely right. When I ask: "Social psychiatry: is there any other kind?" the only tenable answer is: "Of course not". To ignore either term in the equation is to short-change patients. Let me pursue this argument through the British kind of social psychiatry as exemplified in the work of Michael Shepherd, his students and his colleagues at the Maudsley.

I first met Michael 40 years ago in Baltimore when he was a Travelling Fellow of the British Post-Graduate Medical Federation (Shepherd, 1957*a*). I was immediately and immensely taken with his scholarship, his intellectual acumen and his bemusement at the American obsession with psychoanalysis. I did not find the words to describe him until many years later (Russell, 1989). A former patient portrayed him as:

> . . . a tall dark pale man, with a chillingly superior glance and quellingly English voice . . . Attracting his attention and observing his serious face had reduced my store of confidence. I knew, however, that if anyone could discover the 'truth' it would be he . . .

I next met him at an Anglo-American Meeting on Mental Disorders Classification held at Somerset House in London in September 1962. Sir Aubrey Lewis chaired the British delegation. Michael had been among the first to call to attention the surprising difference in mental hospital admissions by age and diagnosis between the USA and the UK. Rates for schizophrenia were lower, and for depression higher, in the UK (Kramer, 1963). That meeting led to a joint USA–UK study led by John Cooper and Bob Kendell (Cooper *et al*, 1972). The principal finding? Diagnostic practices, not disease rates, suffered from transatlantic travel. When the World Health Organization Office of Mental Health convened a seminar in London on the standardisation of psychiatric diagnosis, classification and mental health statistics, Michael Shepherd (Shepherd *et al*, 1968) was the leading intellectual force in organising a series of annual meetings which proceeded systematically through the major categories of mental illness. That work led to new psychiatric diagnostic guidelines for the eighth and ninth editions of the International Classification of Diseases (Sartorius *et al*, 1993). The specification of criteria for the diagnosis of schizophrenia was what made the International Pilot Study of Schizophrenia (WHO, 1975) possible and led to the uncovering of surprising differences in patient outcome, with patients in developing countries doing better than those in industrialised countries (WHO, 1979; Jablensky *et al*, 1992). Michael Shepherd had been instrumental in building a firm foundation for several decades of cross-national research. These studies were led by one of Michael's pupils, Norman Sartorius (Sartorius *et al*, 1986), himself by now an honorary Englishman in taste and judgement. There is much more to be said, but time demands I move on to the epidemiology of psychotropic drug use (Tognoni *et al*, 1981).

Michael Shepherd's (1957*b*) doctoral thesis was based on a study of psychotic patients hospitalised in Buckinghamshire County during two epochs, 1931–33 and 1945–47. The availability of that carefully scrutinised database proved to be particularly important at the time psychotropic drugs were received with great acclaim. The new medications were credited for the greatly shortened lengths of hospital stay and the successful ambulatory management of psychotic patients. Amid these hosannahs, Michael and his colleagues returned to look at patient movement in the Buckinghamshire catchment area from just before to just after extensive drug use. They demonstrated (Shepherd *et al*, 1961) that shorter stays and higher discharge rates were already evident a decade before the drugs because of the introduction of a progressive health service; under those circumstances, the drugs added relatively little. Odegaard (1964) reported a similar pattern from Norway; psychotropic drugs brought little change in hospitals where discharge rates were high before drugs but led to considerable improvements where pre-drug rates had been low. Sir Aubrey Lewis's (1959) trenchant comment puts matters in perspective:

> If we had to choose between abandoning the new industrial resettlement units and social facilities . . . there would be no hesitation about the choice: the drugs would go.

John Wing's group in the MRC Social Psychiatry Unit (Wing *et al*, 1964; Wing & Freudenberg, 1971) went on to demonstrate the underside of community care: some chronic schizophrenic patients, who had lived quietly on an understimulating ward, developed florid symptoms if the social pressures became too great. They (Hewett *et al*, 1975; Ryan & Wing, 1979) highlighted the aggregation of 'new long-stay' patients who required accommodation in special hostels if they were to be successfully discharged. Abolishing hospitals, without building up suitable accommodation in the community, treatment programmes, and rehabilitation services, could only add to the misery of the most severely disabled patients and their families (Wing, 1980).

Had we Americans more fully understood this British research in social psychiatry, might we have spared our patients instant deinstitutionalisation and consequent homelessness? Obviously not; the UK itself followed in our track. Data would not have derailed the juggernaut because data were largely irrelevant to its creation. Emptying out hospitals was driven by a window of opportunity for cost savings, not by concern for the mentally ill. The federal legislation that created Medicaid and Medicare in 1965 and improved benefits for the permanently disabled provided a bonanza

for state governments. By turning patients out of state hospitals, costs were shifted from state to federal budgets. The resident state hospital population decreased by 75% between 1965 and 1986; concomitantly, the nursing home bed count doubled (Grob, 1994). Two-thirds of those beds were occupied by patients carrying a psychiatric diagnosis. Medicaid paid their bills. Other patients were discharged to 'communities' which had existed once-upon-a-time but had disappeared during the years the patients were hospitalised. Their care cost less because there was no care. Misery is not tabulated on the fiscal balance.

Homelessness, it does not take research to tell, is awful. But how good is home? Brown *et al* (1962, 1972) have shown that the likelihood of relapse among schizophrenic patients discharged from hospital is significantly greater if the patients return to families characterised by high 'expressed emotion' (EE). Vaughn & Leff (1976) went on to demonstrate that phenothiazines protect against the relapse of patients in high-EE families but offer no apparent benefit when EE is low, much as Shepherd had shown drug benefits to be fewer in well-run hospitals. Both provide rather splendid proof that psychiatry is as social as it is biological. The power of the family environment is indicated by their observation that the number of hours the patient is in face-to-face contact with high-EE relatives is decisive; that is, if contact is kept below a threshold (35 hours a week), the likelihood of relapse is half as great.

And thereby hangs a tale of whether research, like fine wine, travels well. When Jim Birley gave grand rounds at the Massachusetts General Hospital some years ago when I was Chief, an enthusiastic young clinician on our staff expressed puzzlement at Jim's willingness to make practical use of this finding in patient care rather than to insist on family therapy to correct it. My colleague had complete confidence that 'psychodynamic' family treatment would fix the broken wagon. This contrast between British pragmatism and American enthusiasm for doing something, when just standing there might be better, extends to many fields of medicine. Members of the Social Psychiatry Unit later did develop psychoeducational methods, not psychodynamic treatment, to help high-EE families to cope more effectively and reduce the need for re-hospitalisation (Falloon *et al*, 1985; Leff *et al*, 1985; Tarrier *et al*, 1994), but that is another story for another time.

A third major British contribution, initiated by Michael Shepherd, is the recognition of the amount and type of psychiatric morbidity found in general practice. Studies at the General Practice Research Unit established two major points: first, that the prevalence of depression and anxiety, often masquerading in somatised symptoms, is extensive in general medical practice; second, that what we psychiatrists see in our clinics and hospitals is a very skewed and unrepresentative sample of psychopathology in the general population. It was to take more than a decade before the USA acknowledged that primary care is the *de facto* mental health system (Regier *et al*, 1978). Psychiatrists in every country studied (Ustun & Sartorius, 1995) have been slow to understand that primary care is where the action is. No country, not even one so abundantly supplied with mental health professionals as the USA, can, let alone should, respond to mental health needs through specialist services. As Shepherd *et al* (1966) pointed out 30 years ago:

> The cardinal requirement for the improvement of mental health services . . . is not a . . . proliferation of . . . psychiatric agencies, but rather a strengthening of the family doctor in his therapeutic role.

I hope I have persuaded you that there *is* a "very British kind of social psychiatry" and that Michael Shepherd has been its prime exemplar. The happy news on the otherwise sad occasion of this memorial symposium is that the 30 professors and chief health officers who blossomed under his tutelage are carrying his work forward without losing a step. He will be missed but his work will continue. What characterises that work? Its spirit is captured in the words of Sir Aubrey Lewis, the man Michael revered as his teacher. Sir Aubrey (1962), in an essay on ebb and flow in social psychiatry, wrote:

> The philosophers thought it proper to put not one but two mottoes on the Temple at Delphi: one, the better remembered, was 'Know thyself': but the second, equally imperative, enjoined 'Nothing in Excess'. It might be worth inscribing that over the Temple of Psychiatry.

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