Abstract  M.R. Bury has radically questioned the value of social constructionism for medical sociology (1986). The present authors have already responded to this wide-ranging critique (Nicolson and McLaughlin 1987). This article aims to complement our theoretical discussion by embodying its principles in empirical case study. A recent debate surrounding the pathogenesis of multiple sclerosis is analysed in terms of the skills, interests and backgrounds of the medical personnel involved. It is noted that the proponents of the vascular theory possess developed expertises in interpreting disease in structural, vascular terms, whereas their opponents' skills lie in immunology or neurology. Different observers have produced different conceptions of the disease because modes of observation, and the points from which observation takes place, differ. It is also noted that the debate over the causation and treatment of MS has occurred between a large and powerful social group and a weak and marginal one. The effects of this power inequality on the production and assessment of knowledge about MS are investigated. The significance of the case study for Bury's misgivings as to the value of social constructionism is discussed, particular concern being taken to clarify the notions of instrumental realism, of reflexivity, and of the alleged dispensability of medicine.

Introduction

Recently M.R. Bury has radically questioned the value of social constructionism for medical sociology (1986). The present authors have already responded to this wide-ranging critique (Nicolson and McLaughlin 1987). We have argued that social constructionism as a whole is not in fact vitiated in the manner Bury contends. We have also set out some of the theoretical arguments in support of a
particular variety of social constructionism, namely the ‘strong programme of the sociology of knowledge’ (Bloor 1976: 4–5, 1981; see also Barnes 1972, 1977 and 1982). However, as we noted in our original paper, abstract or theoretical discussion is not the most effective means of demonstrating the heuristic value of social constructionism. The strengths of the approach are best displayed by its deployment in empirical case-study. The present article aims to complement our theoretical discussion by providing such a study.

A constructionist sociology of knowledge tries to explain the production of knowledge in terms of social and historical factors – the cognitive and technical skills which specific groups of scientists possess, their training, the equipment they have at their disposal, and so on. Knowledge is also understood in terms of the particular purposes it is devised to serve. The sociologist stresses, not that input from the non-social world is irrelevant, but that the processes whereby that input is generated, and incorporated into new scientific knowledge, are selective ones. Input from the phenomena under study is filtered according to the equipment, skills and priorities of the observer. Furthermore that input falls not upon a blank slate but upon a set of previously existing beliefs about the world. Scientists cannot therefore devise scientific theories solely in the light of their direct immediate experience of phenomena. They base new knowledge upon the relevant data and upon their pre-existing beliefs and theories. We understand the unknown in the light of what we already know – which, of course, in turn has its roots in training and in prior socialisation. Different observers, therefore, produce radically different cognitive worlds because modes of observation, and the points from which observation takes place, differ.

The interaction between experience and pre-existing theory has been subjected to intense philosophical scrutiny (see Duhem 1945, Hesse 1974, Quine 1964). There is nothing in the above account of the production of scientific knowledge which differs in principle from the results of these investigations. It is important to note, however, that the social constructionist’s attention is not focussed solely upon individual differences between observers, important though these often are. Of principal interest are differences of social situation such as differences in training and education, and differences in collectively sustained priorities of inquiry and use.

These particular social circumstances are often exposed to prominence in scientific controversy. It is frequently the case that the protagonists on either side of a controversy bring different skills
to bear on the problem in question, employ different sorts of equipment, and so on. So each side constructs its own distinctive account of the phenomenon under investigation. Controversies, thus, often neatly display the contingent factors which govern the production of scientific knowledge. They have accordingly become a favourite focus of study for historians and sociologists (see Collins 1981, and for a medical example very comparable to the present one, see Granshaw forthcoming). It is with such a controversy that we are here concerned – a long-standing and recently revived debate as to the aetiology of multiple sclerosis.

In our earlier article we addressed ourselves to five ‘difficulties’ with which Bury challenged social constructionism, namely the difficulties of incoherence, realism, reflexivity, relativism, and sincerity and humanity. We will argue that our case-study demonstrates that the particular form of social constructionism which is here employed is not, in actual empirical practice, insensitive to these problems. Nor is it fatally handicapped by them. In our discussion we will also offer some further clarification of the methodology we advocate. In particular we feel it might be helpful to specify how the realism of the social constructionist is to be distinguished from the varieties of realism advanced by philosophers of science.

The vascular theory of the causation of multiple sclerosis

Multiple sclerosis is a condition in which nerve function is impaired (for an authoritative description see Walton 1985). The most widely accepted theory of its causation is that it is a disorder of the body’s immunological defences. Due to some unknown cause, possibly disturbance by viral infection, components of the immune system attack the myelin sheaths which normally surround the nerve fibres. This produces demyelination and impairs axonal conduction of nervous impulses. A rival theory, however, proposes that the cause of MS lies not within the immune system but with dysfunction of the vascular system, that is lies in the blood vessels. The vascular theory is, in fact, the older of the two, having been first proposed by the French physician and anatomist Charcot in the 1860’s (Charcot 1868). Ribbert (1882) suggested that the cause of MS lay in the blocking of the capillaries by minute thrombi. The modern vascular theories may be seen as variants of this basic idea. In the nineteen-thirties and forties vascular theories were quite extensively investi-
gated. The classic exposition was by Putnam (1933). Several other possible aetiologies have attracted interest, including dietary, toxin and spirochaetal hypotheses (for recent revivals see Wolfgram 1979, Gay *et al* 1986). But, in the nineteen-fifties, the immunological theory gained a dominance it has retained ever since. More recently however fresh interest has been shown in the vascular theory. Dr Philip James (1982), for example, has recently proposed that MS may be caused by emboli of fat causing transient blockages of the microcirculation of the nervous system.

The debate surrounding the causation of MS is an extremely complex one with several different medical factions, not to mention several different grant-giving bodies, involved. We do not therefore claim that the following remarks constitute a complete outline of the controversy. However we do believe that attending to the training, skills and backgrounds of the scientific personnel involved can aid our understanding of the medical knowledge on which the debate hinges.

Philip James works at the Wolfson Institute for Occupational Health which is associated with Ninewells Hospital, Dundee. He specialises in the medical problems associated with working underwater. He is thus extremely familiar with decompression sickness. Many of the symptoms of decompression sickness that involve the central nervous system are similar to those of MS. Accordingly James drew on his knowledge of, and experience with, decompression sickness in order to try to understand MS (James 1982: 380, 1983). Decompression sickness provided him with a model whereby the unknown causation of MS could be conceptualised. The neurological features of decompression sickness are generally held to be due to the presence of gas emboli in the microcirculation of the nervous system and so James proposed a similar causative mechanism for MS. Noting that many of the demyelination lesions associated with MS occur along the course of small veins, he suggested that the neurological symptoms of MS might also be triggered by blockages of the microcirculation – in this case caused by particles of fat. Such blockages might produce lowered oxygen tension in the venule downstream from the embolus. This might, in turn, James suggested, result in damage to the walls of the blood vessels, allowing substances in the blood to leak out into the interstitial fluid causing tissue damage and eventually disturbance of nerve function. The individual fat emboli are transient but blockages might recur at specific sites due to the emboli having preferred pathways through the microcirculation. Alternatively even a single blockage event
might be sufficient to produce a persisting weakness in the wall of the blood vessel, thus rendered that particular portion of the blood/brain barrier more susceptible to further damage.

This schema provides an explanation of why demyelination occurs most commonly in the relatively poorly vascularised parts of the nervous system – a feature of the disease for which none of the other aetiologies provide an adequate interpretation (Allen 1981: 171). It also allows for the possibility that the full mechanism of the development of MS may be multifactorial. For example, individuals might differ, for genetic or other reasons, as to the degree and duration of the damage they sustain from sub-acute fat embolism. Other factors (for example, exposure to infection, stress, or environmental toxin) might interact with the effects of fat embolism to produce the crucial pathological event – namely breaching of the integrity of the blood/brain barrier.

Multiple sclerosis is therefore, according to James’s theory, a consequence of sub-acute fat embolism. Acute fat embolism disease, following trauma of adipose tissue or bone marrow, is a well-characterised condition in its own right. Acute fat embolism disease also has several features in common with MS. In particular, petechial haemorrhages, which have been described as ‘the classic physical findings which firmly establish the diagnosis of fat embolism’ (Peltier 1970), are also a common feature of MS. Acute fat embolism thus provides James with another useful disease model for MS. He can base aspects of his conception of the aetiology of MS on the relatively well-understood aetiology of acute fat embolism in much the same way as he is able to extrapolate to MS from decompression sickness. It might be said that we often understand the unknown not merely in terms of the known but in terms of what is most familiar, in terms of group- and individual-specific knowledge and skill.

James is also very experienced in the use of hyperbaric oxygen in the treatment of decompression sickness. Furthermore oxygen is also an effective treatment for acute fat embolism. It is understandable, therefore, that James and his colleagues in Dundee should follow the promptings of their two principal disease models and endorse hyperbaric oxygen as a possible treatment for multiple sclerosis.

It is interesting that Italian workers investigating decompression sickness have also and independently noted its similarity to multiple sclerosis. They too have made the suggestion that hyperbaric oxygen might be a useful therapy (Palotta et al 1980, Palotta 1982).
Such investigators would, of course, have similar skills to those possessed by James. If one accepts the model of the social construction of medical knowledge which has been presented here, one is not surprised that Palotta and his colleagues should conceive of the underlying processes of disease in a similar way.

It should be noted that James’s proposal of a new vascular theory followed rather than preceded experimentation with hyperbaric oxygen in the treatment of multiple sclerosis or related conditions. The first report claiming benefit from HBO in cases of MS was published in 1970 (Boschetty and Cernoch). This was followed by a small flurry of studies in the late-seventies (Baixe 1978, Neubauer 1979, 1980). James’s sub-acute fat embolism hypothesis may be seen as an attempt to provide a theoretical rationale for the new therapy. One might say that skills preceded ideas – in the sense that general professional orientations, and commitments to forms of practice, structured specific conceptualisations of therapy and aetiology. All the major protagonists for hyperbaric therapy in multiple sclerosis first used HBO for some other reason – Neubauer in the treatment of osteomyletis; James, Formai and Palotta in the treatment of the medical problems associated with deep-sea diving; Yeagar, Maxfield Sr. and Maxfield Jr. through their use of compression chambers in conjunction with radiation therapy (Neubauer 1983).

James, due to his training and experience in underwater medicine, is accustomed not only to using hyperbaric oxygen but also to conceiving of pathogenesis in vascular, hydraulic and structural terms (interview – James/McLaughlin 1982). His theory of the causation of MS is accordingly vascular, hydraulic and structurally localised. It is also important to note that he lays particular stress on those features of MS which occur outside the central nervous system such as the petechial haemorrhages in the skin (James 1982: 380). This emphasis illustrates the fact that, to him, MS is not solely, or even primarily, a disease of the myelin sheaths or any other part of the nervous system. It is for him primarily a disease of the vascular system which only secondarily produces neurological effects.

James’s suggested mechanism of the causation of MS is not necessarily generally accepted even among the advocates of hyperbaric oxygen therapy. However, it provides an important exemplification of how workers outside the mainstream of immunology and neurology have adopted alternative conceptualisations of the disease. Boguslav Fischer and his colleagues (1983), for instance, when reporting the results of their very positive clinical trial of HBO, were formally agnostic as regards theories of the
causation of the disease. However in their discussion they gave great prominence to its vascular aspects:

The dominant anatomic feature of multiple sclerosis is the relation of plaques to the venous component of the vascular system. The plaques, at least in the early stages, are almost always perivenular, extending as slender sleeves of demyelination.

They concluded by suggesting that hyperbaric oxygen might act by alleviating areas of persistent local hypoxia, presumably (although they did not say so specifically) produced by some form of vascular dysfunction.

Support for the idea of early vascular involvement in the aetiology of MS has also come from researchers working with the tomographic scanner. One of the standard techniques used in sectional radiography is a process whereby a radio-opaque dye is injected into the bloodstream, thus enabling any leakage through the walls of the blood-vessels to be readily observed. In other words tomographers possess well-developed skills in the study of lesion events within the vascular system. When these skills have been applied to the investigation of MS they have revealed fresh vascular features. Small asymptomatic lesions have been identified and studied in vivo for the first time (Aita et al. 1978). Professor Sears and his group claim that their radiological observations of new lesions in patients with active MS provide confirmation of the early involvement of blood/brain barrier dysfunction (Sears et al. 1982). They argue that the blood/brain barrier defect observed in MS is not ‘simply an inflammatory response to active demyelination’ but is, on the contrary, a precursor of both lesion development and demyelination. In other words they imply that vascular causes produce neurological effects. Here again it is evident that the possession of specific cognitive and technical skills has structured actors’ conceptions of the disease process.

What has been provided above is a sociological explanation for why certain workers prefer a vascular theory; indeed we have provided a sociological explanation for the specific characteristics of James’s version of the vascular theory. We believe that it is possible, at least in principle, to provide a similar explanation for their opponents’ preference for the auto-immune theory. For the moment however we will confine ourselves to some very brief remarks.
The auto-immune theory

The proponents of the auto-immune theory have well-developed expertises in conceptualising and investigating the hidden molecular activity of viral and immunological agents (see, for example, Mims 1983). Furthermore they have their own laboratory disease model which embodies this distinctive mode of thinking about multiple sclerosis. Just as the vascular theorists can point to the similarities between MS and decompression sickness, so the auto-immune theorists can point to the similarities between MS and Experimental Allergic Encephalomyelitis – a disease which is induced in laboratory animals by injections of nerve tissue. The symptoms of E.A.E, as it is called, are presumed to be due to increased immune activity against such tissue, resulting in damage to the host’s own nervous system.

Unlike specialists in underwater medicine, immunologists are not generally accustomed to thinking in hydraulic or vascular terms. When neurologists think structurally about the pathogenesis of MS it is in terms of the structure of the nervous system. Accordingly they do not place much emphasis on those features of MS which occur outside the CNS – the petechial haemorrhages so significant to James are not even mentioned in Walton’s (1985) account of the disease. Some investigators, such as Professor Adams, have however attended carefully to those lesions which occur in the tissue surrounding the ventricles of the brain (see Adams 1977, Adams and High 1982). The existence of such lesions has been regarded as providing strong presumptive evidence for the direct filtering of a causative agent into the nerve tissue from the cerebrospinal fluid (Allen 1981: 170). To Adams, therefore, the vascular system is not the only, or necessarily the major, route by which the putative cause of MS reaches the nervous tissue. Multiple sclerosis is not therefore primarily a vascular disorder, according to this view of its causation.

As a neuropathologist Adams is a representative of a group of workers who possess very developed skills in the structural conceptualisation of neurological disease. It is interesting that neuropathologists were among the first to respond to James’s challenge to the orthodox conception of multiple sclerosis (for review see Bates 1986). It was quickly pointed out that the ‘demyelination’ seen adjacent to fat embolism is a ‘form of Wallerian degeneration with axonal damage and does not resemble the primary demyelination of MS’. In other words the plaques
associated with acute fat embolism are structurally different, in ways which are significant to neuropathologists, from those of MS. Similarity of lesion structure is taken to be a indicator of similarity of pathogenesis. From this viewpoint, it would seem likely that the lesions of acute fat embolism are produced by a different mechanism from those of MS. Therefore the one is not a good model for the other. The expertise of the neuropathologists is centrally relevant to the defence of the orthodox clinical and neurological view because of its conjunction with the structural emphasis of James's own theory. Bates (1986), for example, employed the neuropathological evidence as a central, strategic component of his attempt to discredit the vascular theory.

It is interesting also that the structural perspective of neuropathology has led several workers in that field to note the frequency with which MS plaques are associated with small veins (Allen 1981, Brownell and Hughes 1962). This is an aspect of the disease which, to the lay person, seems to receive surprisingly little attention in the standard clinical accounts of the disease. Unlike James, however, neither Allen nor Brownell and Hughes take the further step of voicing heterodox opinion as to the cause of this connection. Adams (1977) has, on the contrary, tried to accommodate this finding into the standard view of the disease by suggesting that immunological attack may cause damage to the myelin sheaths and the walls of the veins simultaneously. Neuropathologists might be said to have little incentive to propose unorthodox mechanisms - not generally having a commitment to alternative forms of therapy or, indeed, to any form of specialist expertise outside neurology and pathology.

In the immunological conception of multiple sclerosis laboratory workers are well supported by their clinical colleagues. This is an example of a quite general process affecting many aspects of modern medicine. As Humphrey (1982) has pointed out, 'a major consequence of the widespread acceptance of immunological concepts in medicine has been to alter the way in which disease processes are thought about'. He noted that this did not only affect the perceptions of immunologists and laboratory workers in related fields. There was a tendency, he contended, for clinicians, when faced with puzzling clinical conditions, to give preference to immunological explanations rather than any of the wide variety of possible alternatives.

Furthermore, the clinicians who treat MS patients have always been neurologists - who tend routinely to conceive of MS as being essentially, as well as symptomatically, a neurological disorder.
Also the therapeutic implications of the auto-immune theory are generally pharmacological – involving modes of treatment with which most neurologists are already very familiar and in which they possess developed skills. A new drug treatment for MS, even the trial of a new drug, could be accommodated into existing clinical routines with little difficulty. The viral/auto-immune theory, with its emphasis on events which occur within the nervous system, thus has a close, one might almost say a symbiotic, relationship with clinical neurology. On the other hand few neurologists understand how a decompression chamber works, fewer still would be able to supervise its use. HBO therapy, as presently constituted, cuts against the grain of the current routines of clinical care in neurology. It does not generally take place in wards, theatres or out-patient departments. If it was to become a standard form of therapy, new equipment would have to be bought, additional facilities and accommodation provided, new skills acquired, a new order of technicians trained. It is easy to understand why such innovations are not taken lightly.

No rational resolution

It is important to note that there is no easy or obvious rational resolution to be made between the auto-immune theory and the vascular theory. As the accounts of new experiments and fresh clinical trials are published, each side is able to place its own interpretation upon the results. The early hyperbaric oxygen trials were badly organised and poorly controlled. The results were thus easy to dismiss, particularly since multiple sclerosis is often marked by spontaneous remission, regardless of medical intervention. However later, more carefully controlled trials did seem to encourage the conclusion that hyperbaric oxygen possessed some real beneficial effect (Fischer et al 1983). This provided the proponents of hyperbaric oxygen and a vascular theory with what they regarded as a vindication. Their opponents, however, interpreted the same results quite differently. Some dismissed the improvements as negligible and the tests as inconclusive. Others ascribed any beneficial effect of hyperbaric oxygen not to the removal of perivascular hypoxia but to oxygen suppression of the immune system.

That hyperbaric oxygen suppresses several immune processes was noticed by Warren and his co-workers in the late seventies. In 1978
the same team demonstrated that the symptoms of the MS model E.A.E. were alleviated, indeed sometimes completely suppressed, by hyperbaric oxygen (Warren et al 1978). Warren cautioned against these results being extrapolated to the human situation but proponents of hyperbaric oxygen as a therapy for MS saw his work as providing an empirical justification for their clinical experimentation with the treatment. To them Warren's results suggested a candidate mechanism for the efficacy of HBO, namely that the oxygen suppressed components of the body's immune system. This could be regarded as a rival mechanism to the vascular hypothesis or, alternatively, it could be incorporated into it. In an interesting reversal of Warren's position, James suggested that perhaps E.A.E. was itself an embolic disorder characterised by perivascular hypoxia, albeit immunologically exacerbated (James 1982). To immunologists, however, Warren's results merely confirmed the centrality of immune theory to the understanding of E.A.E. and therefore to MS. A drug regime specifically designed to suppress the immune response would, they claimed, be more effective than hyperbaric oxygen and would offer more hope for further refinement.

The vascular theorists, on the other hand, have their own explanation for the effectiveness of drug therapy. For example, the steroid therapy used in acute episodes of the disease might act not by suppressing the immune system but by reducing vascular permeability and hence alleviating localised oedema (James 1982: 385). Furthermore both steroid therapy and the more specific immunosuppressant regimes involve the administration of large volumes of fluid. The vascular theorists point out that the effects of such increased blood volume on perivascular conditions have never been fully investigated. Hence in the experiments of Hauser et al (1983) there may have been confusion, so it is argued, between the action of the immunosuppressant drugs and that of the introduced fluid (C. Butterworth pers. com.).

Further series of controlled trials of hyperbaric oxygen have more recently been undertaken and others are under way at the time of writing. Some of these latest trials have reported negative results. Other workers have reserved judgement, suggesting that perhaps HBO might be of benefit to particular classes of MS sufferers and not others. In Britain neurologists have proclaimed these negative or equivocal results as a vindication of their original scepticism toward hyperbaric oxygen (Bates 1986). The latest clinical trials have indeed been a disappointment for the vascular theorists and the proponents of HBO but the lack of positive results by no means
renders their position untenable. The accuracy, objectivity or legitimacy of the trials may readily be disputed. The publication of the results of each case-study has been greeted by storms of protest, mainly focussed on the alleged inadequacies of the design of the trial. One common complaint has been that the recent clinical trials have subjected all the participating patients to the same partial pressures of hyperbaric oxygen, whereas the Dundee study (James 1983a) indicated that patients required regimes of treatment tailored to individual requirements. A similar stance has been taken by Neubauer (1983) in the United States. In certain patients oxygen at 2 A.T.A. produces adverse effects which would mask any improvement in their neurological condition. These patients, it is argued, should be treated with lower pressures.

It should be noted that disputing the validity of a clinical trial is not a illegitimate tactic only employed as a desperate last throw by the losing party. Supporters of the immune theory attempted to cast doubt on the earlier positive HBO trials in an exactly similar manner. Dr Allen Downie, of the Department of Neurology, Aberdeen Royal Infirmary, was one of the most vigorous critics of James and the Dundee ARMS group. Downie argued that James could not use the results of Fischer's trial to legitimate HBO therapy since 'Not all such trials are valid . . . ' (Downie 1982). In fact the difficulties of devising an adequate clinical trial in as complex and protean a disorder as multiple sclerosis are well-known to all in the field, as are the difficulties of arriving at firm conclusions from trials however devised (Brown 1980, Silderberg 1984, see also Robinson forthcoming). Furthermore, as Chalmers (1974) has demonstrated, the results of clinical trials have only the most problematic relation to changes in medical practice, even in the case of well-understood and clearly defined disorders.

Thus, the experimental and clinical evidence has no authority in itself to resolve the differences between the disputing parties. For the time being at least, different forms of expertise, different underlying conceptualisations of the disease process, ensure the continuation of the debate. (For descriptions of similarly indefinite conclusions to other controversies, see Pickering 1981, and several of the other essays in Knorr et al, 1981.)

Knowledge and authority

Our discussion has so far concentrated almost wholly on the cognitive elements of the debate and has demonstrated how these
are structured around the participants' particular skills and expertises. However it should also be noted that in such contexts as this – where distinctions between valid and invalid medical knowledge must be made – questions of expertise interconnect with questions of power. Immunology has possession of the commanding heights of orthodox medicine (Humphrey 1982) and accordingly possesses great power – power to grant recognition, to open access to publication, to fund research. Moreover neurologists and immunologists have always dominated the Medical Advisory Committees of the British and the American Multiple Sclerosis Societies, major grant-givers in the field of MS research. The debate over the causation and treatment of MS is not, therefore, an interchange of ideas between social equals, but is a competition between a large and powerful social group and a weak and marginal one. A full investigation of the effect on the controversy of the power inequalities between the warring parties lies outside the scope of this article. It should however be noted that the high status of immunology has allowed it to create the ground rules by which knowledge about multiple sclerosis is assessed.

The same issue of the New England Journal of Medicine which published Fischer's article reporting positive results from a placebo-controlled, double-blind trial of hyperbaric oxygen also contained a paper describing a trial of an experimental drug therapy for MS (Hauser et al 1983). This investigation, led by two Boston neurologists, was not a double-blind study nor was it placebo controlled. One of the drugs employed, cyclophosphamide, is potentially a cause of major side-effects, such as serious infections, even cancer. For this and other reasons the treatment, even if proven successful, would only be suitable for chronically deteriorating patients who had failed to respond to other therapy. HBO has a variety of minor transient side-effects but in skilled hands is perfectly safe at the dosages given for MS. In principle it could be used for most patients.

The best results of the drug therapy trial were obtained from a group of patients who received cyclophosphamide plus the hormone ACTH. Of these approximately one third showed functional improvement and one third remained stable. Fischer reported significant improvement in 72% of his oxygen subjects. In other words, at first sight, Fischer's trial would seem not only to have embodied a more rigorous experimental design than the cyclophosphamide study, it also produced more positive results and involved a form of therapy with fewer side-effects and thus of wider
potential application. This was not, however, the impression that was made upon the leaders of multiple sclerosis research.

Editorial comment in the *N.E.J.M.* treated the two studies as broadly comparable. The Annual Report of the National [American] Multiple Sclerosis Society (1983), however, acclaimed the cyclophosphamide study as 'striking a positive note for MS clinical research'. It suggested that Hauser's positive results corroborated 'the theory that MS is an immunological disorder since the drug is thought to act as a brake on an abnormal immune response'. Financial support for follow-up studies was promised. The HBO trial was described as yielding 'much less impressive results'. On another occasion, Dr L. Scheinberg, Chairman of the Medical Advisory Board of the National Multiple Sclerosis Society, stated plainly that he did 'not believe the results of Fischer's trial' (Steffen and Wang 1983). From Scheinberg's point of view, however scientific Fischer's procedures might appear to be, his results were simply implausible. They did not correspond to what previous experience had led him to expect. The cyclophosphamide trial on the other hand neatly confirmed the expectations created by previous work in the mainstream of MS research. Byron Waksman, Director of Research Projects for the National Multiple Sclerosis Society, specifically emphasised that his favourable assessment of Hauser's study was a product of its continuity with earlier immunological work:

The possibility that certain neurological diseases, of which multiple sclerosis is the leading example, might be of an immunological nature was suggested in the 1930s by Pette, Schaltenbrand and others, primarily on morphological grounds. . . The production in 1932 by Rivers and his colleagues of an experimental autoimmune \[sic\] encephalitis (E.A.E.) in rabbits immunized with myelin provided the first solid experimental verification of this point of view. With the demonstration, half a century later, that MS can be arrested in the majority of patients tested, for a year or more, by the powerful immunosuppressive agent cyclophosphamide, the likelihood that MS is immunologically based has become a virtual certainty. (Waksman and Reynolds 1984)

Wolfgram (1979) has asserted that for medical knowledge about multiple sclerosis to be assessed as valid knowledge it has to accord with the protocols of immunology. Interviewing members of the Wistar Institute in Philadelphia (a major centre for MS research in America), we found that harmony with the received wisdom in immunology was indeed perceived by researchers in the MS field as being a major factor in determining whether or not research
projects got financial backing.\textsuperscript{11} One interviewee described how MS researchers at Wistar felt obliged to do work which linked up with the group projects the Institute had already set in motion. If one does not fit this social mould, it was asserted, it is ‘next to impossible’ to get funding. The shape of the mould is determined by the orthodox community and ‘they fund research only in immunology’.

The history of the hyperbaric therapy for multiple sclerosis would seem to bear out this impression. The British MS Society funded trials of hyperbaric oxygen very reluctantly and only as result of intense public pressure from the lay membership of the Society and interested parties outside (see Morris 1983). Advocates of hyperbaric oxygen in America faced similar opposition. It is hard to believe that had Fischer produced comparable results from a study involving an immunologically-active drug, there would have been such resistance to follow-up investigations.

On the basis of her study of the controversy surrounding the efficacy of vitamin C therapy in cancer, Richards (1986) has argued that not all suggested therapeutic innovations are assessed in the same way. Different rules apply according to whether the therapy in question belongs to the body of orthodox medicine (even though it might be potentially dangerous or toxic) or to marginal medicine (where it is assumed to be ineffective and dangerous until proven otherwise by the most stringent scientific tests).\textsuperscript{12} The perceived efficacy of the candidate therapy hinges on the relative political power of its supporters (see also Petersen and Markle 1979). We have seen a very similar pattern in the HBO debate.

For a variety of reasons hyperbaric oxygen is marginal medicine.\textsuperscript{13} The protocols of immunology, as employed by experienced and elite practitioners, have a large part in keeping it there. Moreover the history of multiple sclerosis treatment has been long been characterised by extravagant therapeutic claims. It is a domain of quackery and frequent accusations of quackery (see Van den Noort 1983). This means that the boundary between what is orthodox and what is not is a sensitive one. It is therefore rigorously policed. In the field of MS therapy and research simply being respectable means a great deal.\textsuperscript{14}

The burden of proof and persuasion therefore lies heaviest upon the proponents of the less orthodox therapy. Whether one sees this exercise of dual standards as necessary scepticism and a legitimate application of conservative quality control or as the suppression of innovation by established interests depends on one’s position \textit{vis-à-vis} a particular therapeutic innovation, or orthodox medicine in
general. To the sociologist it is interesting to see scientific knowledge being so obviously subjected to a calculus, the social location of which is evident.\textsuperscript{15}

**Discussion**

If the details of the above empirical example are compared with the criticisms listed in the introduction to this paper, it will be seen that many of Bury’s misgivings as to the strengths of social constructionism may be laid aside. Bury's criticisms, it will be recalled, related to questions of incoherence, realism, reflexivity, relativism, and sincerity and humanity. We hope that our account of the sociology of the vascular theory of multiple sclerosis is coherent. If it is, that is largely because our procedures are those of a single tradition of constructionist inquiry.

The above account is realist. Bury argues that social constructionists have failed to recognise the extent to which, to employ Thompson’s metaphor (1978: 210), the character of the table is determined by the ‘logic of the wood’.\textsuperscript{16} But one may accord full importance to what one might call the ‘logic of the disease’ and still produce a thoroughly sociological account of medical knowledge. It might be noted that the two competing systems of belief about MS are both here acknowledged to be firmly based upon a single shared physical reality of disease – a physical reality which both groups of actors engage and operate with actively. The sociologist of medical knowledge need not discount the scientists’ intuition that the evidence provided by, say, neuropathology derives, in some sense, from beyond the social. One need not deny that a photograph of an MS plaque on the interior surface of one of the cavities of the brain is a photograph of something which is not wholly an artefact – even if the objects photographed could be named and conceived of differently, even if both the term ‘periventricular lesion’ and the significance attached to it are social conventions, even if a medical photograph is itself a conventional representation, the making of which requires years of training and specific socialisation. It may be agreed by sociological observer as well as scientific actor that the debate is about the reality of disease. But it must equally be acknowledged that the real difference between the two sets of belief about the disease remains a social one. The contrasting perspectives on the same shared physical reality are the product of the specific trainings, backgrounds and interests of the scientists involved.
Specialists in underwater medicine ‘see’ the phenomena of disease in vascular terms; tomographers seek for new ways in which to apply their visualisation techniques; all the actors have investments in particular procedures and forms of expertise. What is more, the means by which competing statements about the real world are assessed is evidently a social one – involving not only prior belief and expectation but also power, authority and status. Acknowledging that technical medical knowledge successfully engages with the real world thus poses no threat to the power of sociological explanation.

It may be acknowledged that the above account does not attempt to demonstrate, in any strong sense, the precise nature of the interaction between scientific actor and external reality that has gone into the making of medical knowledge about multiple sclerosis. To have done so would have required a much more sophisticated level of analysis than here attempted. Our point is the lesser one that the present account is perfectly compatible with the actors’ belief that input from external reality is one of the resources that they drew upon in the construction of their belief systems.

On the other hand, we do make the strong claim that the above account demonstrates that belief is underdetermined by external reality. One of the ways this is manifested is by empirical evidence having no force by itself to cause the abandonment of belief. We have seen that the participants on either side of the MS debate have been able readily to accommodate all the available experimental findings and clinical data to their own point of view. The results of a clinical trial may be interpreted by one side as a vindication, by the other as inconclusive due to defects in the design of the trial. The fact that hyperbaric oxygen alleviates E.A.E. is regarded by one camp as demonstrating the centrality of immunological processes, by the other as requiring a reinterpretation of the aetiology of E.A.E. in vascular terms. This process is in principle endless. The rival theories are sufficiently flexible and resourceful to be, formally and actually, indefeasible. The discrediting of scientific theories is not an evidential event but a social process, predicated upon conventions of evidence, plausibility and authority.

It should also be noted that the variety of realism presented here need involve us in neither a correspondence nor a contemplative view of knowledge. Correspondence realism entails that for a theory to be correct it should possess a unique relationship between itself and the relevant features of the natural world (see, for example, Leplin 1984). The idea of a periventricular lesion is, from this point
of view, a useful one because there are such things in reality. Constructionist realism makes no such claim. Rather it adopts an instrumental view of knowledge – the idea of a periventricular lesion is a useful one because it enables us interact with physical reality in a meaningful way. Barnes’s (1977: 7) suggestion that cartography provides a very useful paradigm of knowledge may provide a useful exemplification here. A Ordnance Survey map is extremely useful in helping us find our way around the countryside but it cannot be said to mirror the physical reality of the area it covers. Maps are social conventions. A collection of contour lines looks nothing like a mountain. Nor is a contour line a useful aid to navigation because such things occur in nature. Similarly knowledge may be regarded not as a unique representation or mirror of external reality but as an instrument by which we may operate with that reality.

Instrumental realism, unlike correspondence realism, acknowledges the possibility that quite radically different conceptions of the world may be equally effective in practice. To continue our cartographic analogy – the countryside may be adequately mapped in very many different ways. The shape of the land does not uniquely determine how human beings chose to navigate or to travel. Nor does the reality of disease determine how we conceive it.

The contemplative view of knowledge, by which much philosophy of science is characterised (see, for example, the so-called ‘temperate rationalists’ discussed by Bury 1986: 152), likewise neglects the social and instrumental dimension (Barnes 1977: 1-10, 1979). It typically regards knowledge as a set of formal statements, potentially of universal validity and application (Jarvie 1984a: 178, 1984). To the sociologist of knowledge however all statements, either evidential or theoretical, are socially located and sustained. Knowledge is devised to serve particular social and technical purposes. Its meaning is determined contextually, by its employment within particular forms of life (see Bloor 1983, Wittgenstein 1953). Whether statements about the real world have any validity outside of the context for which they are devised is entirely a contingent matter. A contour line is not a true representation of nature save within a particular tradition of map-making.

The above account is not reflexive. We have studied the knowledge of the vascular theorists and not our own knowledge. However the possibility of a reflexive, social explanation of the present authors’ understanding of the dispute over the aetiology of multiple sclerosis may readily be indicated. Malcolm Nicolson received his post-graduate training in the Sciences Studies Unit of
the University of Edinburgh. During that period of instruction and
in the course of further study and teaching in Edinburgh, he gained
whatever skills he possesses in the sociological analysis of knowledge.
His initial training in biology and his subsequent employment as a
historian of medicine (in its turn structured by employment
opportunities) have both lead him to concentrate his research effort
on the history and sociology of biological and medical knowledge
rather than other aspects of social life. Cathleen McLaughlin took
her first degree in the Department of History and Sociology of
Science of the University of Pennsylvania, which teaches an
approach to scientific knowledge similar to that of the Science
Studies Unit. While on a student exchange to the University of
Edinburgh, she was taught history of science by Nicolson. Thus the
character of the above account may be seen as explicable in terms of
its authors’ skills, priorities and interests – which are themselves
explicable in terms of training, professional orientation and career
opportunity.

It should be noted that we are not here committing the
individualistic fallacy of believing (pace Gouldner 1971) that
reflexivity may be achieved simply by authors appending bio-
graphical accounts to sociological texts. Reflexivity, if it is to be a
valid part of sociological inquiry, has to be achieved collectively.
Indeed its principal focus should not be the individual investigator
but the scholarly community – for it is at that level that knowledge
and practice become embodied in distinctive and meaningful forms
of inquiry. It is also at the level of the scholarly community that the
complex of phenomena we might call cultural bias may be best
discerned. The achievement of reflexivity must thus inevitably be a
long project requiring much further theoretical elucidation and
empirical study. Our personal stories merely indicate that a reflexive
understanding of the construction of this particular sociological
account is feasible, illustrating that it would be possible to explain
Nicolson and McLaughlin’s knowledge in the same way as they have
attempted to explain the knowledge of the vascular theorists.
However, as we have already argued (Nicolson and McLaughlin
1987: 115), the personal circumstances of knowledge production are
irrelevant to whether or not a piece of empirical research is deemed
to be of good quality.

Our account of the dispute between the vascular and the auto-
immune theory is a thoroughly relativist one. No judgement is made
as to which, if any, theory is correct. Our explanation of both sides
of the dispute is, at least in principle, perfectly symmetrical. Both
sides interact with the nature of the disease according to the lights of their own positions. The above account is also relativist in the sense that it is predicated upon the assumption that no neutral point can be found from which to assess the merits of the rival theories. The present authors are, for example, quite incompetent to make any decision regarding the technical rights and wrongs of the matter. Such assessment requires an expert – but all the experts are interested parties to the dispute.

The above account obviously does not constitute a complete explanation of the beliefs of the vascular theorists or their opponents. We have merely attempted to explain the points of difference between the modern vascular theories and the autoimmune alternatives. It is, of course, true that all the actors mentioned above have much in common. Indeed viewed from a broad social or historical perspective, they share more than divides them. They have a common commitment to a modern scientific conception of the world; they all believe in the disease category ‘multiple sclerosis’, in the value of experiment and clinical trial, in the general efficacy of modern medicine. The roots of these shared beliefs are here uninvestigated. We would however contend that, in principle, the form of analysis presented here ought to be applicable not only to the specific details of individual theories but to the totality of modern medical knowledge. We would contend that the common ground between our groups of actors is itself explicable in terms of shared cultural backgrounds, shared patterns of education and socialisation. In other words with sufficient empirical work it should be possible to provide a constructionist explanation not merely of why Dr James is an adherent of the vascular theory but also why he believes in the reality of multiple sclerosis, why he recommends treatment with hyperbaric oxygen rather than treatment by witchcraft, why he believes there are such things as veins or nerves.

One of the consequences of such a perspective is that the social constructionist approach to medical knowledge need not be seen as tending or seeking to demonstrate the ‘dispensability’ of medicine (Bury 1986: 155–7). In fact by displaying the strength of the investment which has been made in modern medicine by scientists, doctors, patients and carers, constructionism provides an insight into why medicine is such a stable, important and indispensable part of late-twentieth-century social life. The evident coherence and stability of the cognitive world of scientific medicine is the product of the extent to which the participating actors share resources,
interests and commitments. It is produced by what Collins (1985: 16–8) terms 'joint and multiple entrenchment'.

Pickering (1984) has given a very detailed account of how a similar form of cognitive order emerged and developed to such a point as to constitute a powerful consensus. The new particle physics has gained such coherence that actors operate unconsciously within it, quite forgetting how the landmarks of the cognitive world they inhabit are comparatively recent social accomplishments. The labour that has gone into making the world of modern medicine stable and coherent has been orders of magnitude greater than that devoted to the world of particle physics. Very large numbers of individuals and groups have strong commitments to the maintenance of that stability and coherence. It is no wonder that, as individuals, we can no more change the categories of the world of modern medicine than we can fly to the moon. Certainly there is nothing in social constructionism which suggests that our intuition that the medical world we inhabit is given is in any way a false one. We cannot, alas, wish multiple sclerosis away, or decide that it should be alleviated by hyperbaric oxygen or cyclophosphamide. Likewise we cannot, merely by an act of will, decide that immunology should not be deemed a powerful high-status discipline. We can however ask interesting questions as to how that taken-for-granted world got the character it did, why it stays the same, and why it changes.

Nothing in the above account should be taken as impugning the sincerity and humanity of any of the actors. It is true that participants in this debate have accused each other of being motivated by considerations other than the welfare of their patients (see Steffen and Wang 1983, for criticism of the orthodoxy, and Downie 1982, for criticism of James). But our analysis of the controversy has involved us in no judgement as to the rights and wrongs of these accusations. We would rather suggest that the symmetry of the opposing criticisms provides an interesting example of the relativity of perceptions of sincerity and humanity.

On the other hand it is true that our account of the debate has accorded an important role to prestige, power, authority, and interpersonal and intergroup rivalry. But to acknowledge the presence of these features is merely to recognise that social life in the medical and scientific communities shares many of the characteristics of social life elsewhere. It is to paint the face of medical research as a human activity. This should not, in the present context, be taken as a slur against scientific medicine or medical researchers per se.
Finally the necessary relativity of a constructionist account should not be construed as setting any limits upon the potential practical relevance of the sociology of medical knowledge. In fact empirical sociological investigation is, or ought to be, centrally important to the making of decisions concerning policy and social action. If we wish to improve medicine then we ought to have as accurate as possible an understanding of how it is presently constituted and sustained. Our intention here has been to demonstrate that social constructionism provides sociology with a heuristically interesting and methodologically sound means whereby to address the research challenge posed by the remarkable social phenomena of technical, medical knowledge.

Wellcome Institute
183 Euston Road
London NW1 2BP

Acknowledgements

The authors wish to thank David Armstrong, Robert Baker, Mike Barfoot, Mel Bartley, David Bloor, Charles Butterworth, David Cantor, Richard Gillespie, Henrika Kuklick, Roy Porter, Ian Robinson, Steve Sturdy, Tilli Tansey and Andy Pickering for helpful comments on earlier versions of this paper. It should, of course, be noted that our helpers, especially the first named, do not necessarily endorse all our arguments. We would also like to thank Andrew Foley for his professional assistance. Malcolm Nicolson wishes to acknowledge the support of the Wellcome Trust during the period of preparation of this paper.

Notes

1 It should be noted however that to Duhem and Quine the choice between alternative explanations is an arbitrary one. To the constructionist such choices are structured by socially sustained priorities.
2 Bury (1987) has subsequently challenged us by stating that he did not use the phrase ‘sincerity and humanity’ in his earlier article. He did however accuse constructionists of conveying ‘the impression that care and welfare are mere facades for the interests of powerful groups’ (Bury 1986: 166). It is to this aspect of his criticism of social constructionism that our discussion of ‘sincerity and humanity’ is directed.
3 Burnsfield (1985) is a useful account for the layperson.
4 For reasons of space, detailed references to the medical literature have been omitted from the present paper. A fully-referenced version is available from the authors.
5 James’s interest in treating multiple sclerosis with HBO was initially prompted by noting that the symptoms exhibited by a car accident victim, with presumed
trauma of depot fat, bore similarities to both MS and decompression sickness (James 1982: 381).

6 Vascular theorists reply that the neuropathologists are extrapolating from the gross lesions of acute fat embolism to the much smaller and more transient lesions of sub-acute fat embolism, which might produce a different pattern of degeneration (Butterworth, pers. com.).

7 This lack of expertise was highlighted when spokesmen for the opposition to HBO, pressed in public to rationalise their antipathy towards it, claimed that it posed a danger to patients. Dr B. Waksman, Director of Research Programmes for the National MS Society, maintained that the technology was complicated and dangerous (Waksman 1982). Dr R. Kelly, Chairman of the Medical Research Advisory Committee of the British MS Society, said that the compression chambers carried a severe fire risk because 'oxygen explodes if it is brought into contact with any sort of spark' (Kelly 1983). He was evidently under the impression that the chambers contained pressurised oxygen – in fact they contain pressurised air, the oxygen being supplied to the patient by mask. Compression chambers are very safe and comparatively simple to operate. Their safety is eloquently vouched for by the smallness of the premiums charged by Lloyds to insure the Dundee chamber against all risks (James 1983b). Waksman is a leading immunologist; Kelly an eminent neurologist.

8 It would have been technically very difficult for Hauser et al to organise a double-blind placebo control for cyclophosphamide since the drug caused alopecia in all the subjects.

9 For a sociological investigation of the notion of 'plausibility' in science, see Harvey (1981).

10 The initials E.A.E. are usually held to refer to Experimental Allergic Encephalitis. That Waksman and Byron refer to the disease as 'Experimental Autoimmune Encephalitis' illustrates the extent of their conviction in the autoimmune theory.

11 Problems of confidentiality prevent us from giving full details of these conversations. More information may be obtained from Ms McLaughlin, who conducted the interviews.

12 A vivid demonstration of how harmony with the prevailing orthodoxy may permit the use of therapies that would be unthinkable under any other circumstances is provided by the recent experiments involving the treatment of progressive MS by intense irradiation of the lymphatic system, see Cook et al (1986).

13 Support for HBO as a therapy for MS has not tended to come from members of the most prestigious institutions nor have positive research reports frequently been carried by the leading medical journals. Neubauer, for example, is a general practitioner and he published in the relatively obscure Journal of the Florida Medical Association. Moreover the hyperbaric treatment has been offered in Britain largely by self-help groups such as ARMS (Action for Research into Multiple Sclerosis) and in America by fringe practitioners such as osteopaths.

14 The force of this peer pressure to conform may be seen in the fact that the Underwater Medicine Society, many of whose members advocate the use of HBO for a wide variety of conditions, have refused to endorse the HBO treatment of MS (see Underwater Medical Society 1979). The Underwater
Medicine Society is a relatively insecure professional group and the risk of being associated with quackery has frightened them off. This fact indicates that the model of scientific knowledge presented here – in which belief is seen as being structured around skills and training – must be complemented by recognition of the effect upon actors of their perception of the procedures of knowledge accreditation. While seeking to achieve maximum utilisation of their specialist skills, actors must calculate the response of their peers to any specific knowledge claim. Knowledge is structured not only by what is possible in the laboratory or the clinic but by the costs of defending knowledge claims in the wider professional domain.

15 The crucial importance of the social apparatus for the assessment of knowledge is one reason why technical knowledge cannot be qualitatively distinguished from ideological knowledge. In order for technical knowledge to be accredited it has to be able to move people as well as things.

16 When using the metaphor of the 'logic of the wood' one should remember that 'wood' is itself not a primitive category. Laypersons often negotiate about how the term is to be applied, arguing whether hardboard, chipboard or bamboo is 'really' wood. Nor has the science of botany been exempt from such problems. See Greene (1983) for fascinating insights into the long historical process of negotiation which has produced our current conception of plant organography. Where the pith stops and the wood starts is more of a social convention than one might intuitively suspect.

17 For attempts along these lines, see Pickering (forthcoming a and b).

18 It should be noted that what we term 'input from external reality' is not identical with what scientists call 'raw data'. To be called data, input has already gone through a long complicated process of selection, sorting, re-arrangement and quality control, see Latour and Woolgar (1979) and, for a classic medical example, Fleck (1976).

19 There are many case studies which display that coherent knowledge systems need not have unique characteristics. The most elegant is probably Dean (1979), see also Nicolson (forthcoming).

References


comparative case-study from the history of French and American plant ecology'.

In L. Hargens, R.A. Jones and A. Pickering (eds) Knowledge and Society: Studies in the Sociology of Science, Past and Present. 8, JAI Press.


Palotta, R. (1982) Hyperbaric Therapy and Multiple Sclerosis Naples:
Administrative Office for the Province of Naples.


Underwater Medical Society (1979) 'Uses of HBO', Pressure: Newsletter of the UMS. 8 (1).


This document is a scanned copy of a printed document. No warranty is given about the accuracy of the copy. Users should refer to the original published version of the material.