'Stand ye in the ways and see and ask for the old paths where is the good way and walk therein and ye shall find rest for your souls' (Jeremiah VI, 16)

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Depression and myalgic encephalomyelitis

We read the letter from Lev (November 1989 *JRSM*, p 693) with interest, but see a danger in using assumptions as to aetiology in definition of study groups. Operational definitions not making this assumption will produce replicable findings and progress towards better definitions and understanding of aetiology.

Definitions of depressed control groups are difficult, for example the following need to be controlled:

(1) Demographic variables
(2) Severity of depression symptoms: inappropriate control groups for ME patients would be severely depressed inpatients. Outpatient depressives are not too dissimilar in severity.
(3) Psychotropic medication: this is less likely to be given to ME patients where treatment is not agreed and could modify symptoms to be compared.
(4) Psychiatric history: in possible ME patients a previous significant psychiatric illness prior to fatigue symptoms leads to difficulty in studying this symptom and produces too much overlap with depressed controls.
(5) History of febrile illness: to minimize overlap, one must also control for preceding febrile illness in otherwise typical depressive illness.

Comparison of control groups should be serial, not cross-sectional as physical symptoms and markers may fluctuate, as may fatigue and depression.

Assessment of depressive symptoms is difficult, as Lev points out, due to non-specific 'biological' symptoms of depression. However, psychic ones such as pessimism should not overlap and could be assessed.

The concept of fatigue is poorly understood, as is its assessment. The paradigm of pain research has much to offer, where 'dichotomization' of physical and psychological components is not thought useful, but assessment emphasizes all components of the experience of pain. Thus, psychometric assessment of fatigue, for example, its severity, frequency, and pattern may be a future research area. Using such a paradigm, our initial findings of differences in fatigue in the two groups are because depressed patients are predominantly anergic, but 'ME' patients have more variability and unpredictable onset of fatigue relative to the severity of exercise attempted. Lack of motivation overlaps in both groups, explicable in Lev's own terms as due to a reaction to a chronic illness.

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Wind of change II - medical journals in Britain in 1988

In the lead-up to his delightfully whimsical construction (December 1989 *JRSM*, p 774) of a case report by William Wordsworth on the clinical effect of massed daffodils, Dr Jackson mentions his inability to accept Medawar's statement that 'There is no such thing as unprejudiced observation'. Medawar is unfortunately no longer with us to explain or defend this statement but I believe it to be part of his appreciation of the larger truth that there is no such thing as pure observation uncontaminated, as it were, by inference. Only the newborn babe can be truly objective; very quickly, observation becomes a complex of seeing things and drawing inferences about them and therefore can be surprisingly unreliable and inaccurate.

It becomes virtually impossible to separate what we see from inference although an awareness that observation consists of these two elements probably helps. We are commonly prejudiced by what it is in our interest to see; hence the need for blind techniques. If Dr Jackson is not entirely familiar with these ideas about the unreliability of observation, he would, I believe, enjoy a book *The Anatomy of Judgement* (Hutchinson, London, 1960) by ML Johnson Abercrombie, happily still in print (Pelican Books, London), in which she demonstrates with fascinating and disturbing examples that things are not always as they seem to be and, simply because we are not expecting them, we may fail to see things that are staring at us.

Such a book helps to explain why Medawar, and Popper before him, were so scornful of the inductive process of reasoning. Dare I suggest that both these profound thinkers were over-iconoclastic and threw away the baby as well as the bath water? Even though the observations on which the synthesis of a hypothesis is traditionally based may be intricately contaminated by inference, it is still useful to think of the inductive-deductive processes as a staircase on which thought goes rapidly and repeatedly back and forth, upstairs inductively to the hypothesis and downstairs deductively.

It seems to me that these thought processes must have some relation to observations, facts or reality whatever we call it and that the hypothesis or inspiration (Medawar) does not arise from nowhere as seems to be implied by Medawar's hypothetico-deductivism.

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Subarachnoid haemorrhage: can we do better?

The arguments and conclusions of Johnston *et al.* (December 1989 *JRSM*, p 721) are largely irreproachable, but the interpretation of the published CT and MR images of case 3 is questionable.

The contrast enhanced CT shows a small circumscribed rounded opacity typical of a posterior communicating aneurysm. The adjacent mass is of brain density, surrounded by a zone of low attenuation, consistent with a 10-day-old haematoma. The T_2-weighted MR shows high signal in the central area of the mass with a surrounding narrow band of low signal, probably due to methaemoglobin with surrounding haemosiderin. The likely diagnosis is therefore a partly re-absorbed medial infratemporal haematoma.

A single T_2-weighted MR image gives incomplete information: fresh blood has no paramagnetic properties, but deoxyhaemoglobin formation in intact erythrocytes shortens the T_2-relaxation time. Subsequent lysis with methaemoglobin formation