change in the circumference of finger joints has become a standard method of assessment in rheumatology trials; yet it depends on selection of the right type of patient if it is to be effective.[5] A different kind of misuse of randomised clinical trials is that they have become part of promotional campaigns; there is now a huge literature comparing antirheumatic drugs with each other. Yet papers can easily be selected to show that the effect of a drug A is greater than drug B, which is greater than drug C, which is greater than drug A: such an Escher spiral must have required considerable effort by clinicians, but whose interests have been served?

So Cranberg's argument that retrospective controls may be valid is worth considering. Until recently this might have meant reliance on the memory of subjective evidence; but data are now collected in more objective form and, as Cranberg says, are more easily retrieved. To be sure, strictures about methods and their use in randomised controlled trials apply just as forcibly to the use of historical controls, but they do not sway the evidence in favour of the randomised trial. An argument often advanced against comparisons based on retrospective data is that patterns of disease change. But how rapid are such changes in relation to the periods concerned? And if change is rapid, perhaps for environmental reasons, then a randomised trial may be invalidated just as much as any other.

In his wide-ranging paper that includes comments on randomised controlled trials Black concludes that "in spite of these reservations, I would agree that if a controlled trial is practicable and can produce a result, it is a most valuable contribution to progress. . . ." That and no more: the controlled trial has been placed on too high a pedestal and needs to be brought back to earth.

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[3] Dollery, C T, *Journal of the Royal College of Physicians of London*, 1977, **11**, 226.
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Who carries the can?

Clinical medicine, and especially therapeutics, advances by a process of evolution, in which only the changes proved to be of value survive. New ideas and new drugs are introduced and assessed and either accepted into the body of orthodox medicine or rejected. In recent years, for example, beta-blockers and bypass grafting have become accepted treatments for coronary heart disease, while anticoagulants and hyperbaric oxygen have fallen into disfavour.

Unfortunately these evolutionary principles do not seem to be applied to changes in the organisation of medicine. Only rarely is any attempt made at a pilot study: more often a committee makes its report and new measures are introduced with no built-in system for their evaluation. An innovation may be introduced with all the fervour of a divine revelation —but the conviction of enthusiasts is a poor substitute for objective evidence based on soundly constructed experiments. More important, when practical experience suggests that an innovation does not work there is no mechanism for it to fade silently away.

So the disenchantment evident in Appleyard and Maden's criticism of multidisciplinary teams (p 1305) is not really surprising. These teams were introduced into clinical medicine as part of a new gospel—consensus management—which was envisaged as a bright new solution to some of the discontents in the Health Service. No doubt there are circumstances in which consensus management does work, but making clinical decisions is not one of them. As the authors explain, the fundamental defect in the concept of team management is that individuals will follow decisions with which they disagree only if they are forced to do so. If the team is arranged in the old-fashioned hierarchical fashion, with the doctor as boss, he can enforce such decisions; but in the current set-up a social worker or psychologist who disagrees with the team decision remains free to obstruct it by refusing, for example, to arrange for special education for a disturbed child. Neither the legal nor the ethical implications of obstruction of this kind have yet received enough attention.

Whatever the defects of the old system, in which the doctor was an autocrat who took advice from his colleagues but then made his own decision, it had two important merits. Firstly, the patient knew who was treating him. All too often nowadays patients are made confused and miserable by obvious conflicts and anomalies in their treatment by different health professionals. Secondly, when things went wrong, the doctor carried the can—and at worst had to appear in court and possibly pay damages or answer charges before the General Medical Council. Few of the new health professions have established procedures (comparable to those for doctors and nurses) for dealing with unprofessional or unethical conduct by their members. And where does the legal responsibility lie in these days of team management? The recent spate of civil liability cases in which damages have been awarded against health authorities suggests that they will be held liable for errors and omissions made by the teams they employ—but spreading responsibility so widely must encourage individuals to believe that they were not to blame. In traditional clinical medicine doctors (most often) learnt from their mistakes: the new-style medicine of modern management seems to be designed in such a way that no one has to admit that the mistake may have been his or her fault.

Asymptomatic complete heart block

The widespread practice of recording routine electrocardiograms in almost every patient attending the medical outpatient department has led to general physicians seeing many more asymptomatic patients with complete atrioventricular block. In these circumstances the risk of syncope in individual patients is unknown; but some clinicians have taken the view that it is always sufficient to warrant prophylactic insertion of a pacemaker.

Atrioventricular block may be due to lesions at any of three sites: the atrioventricular node, the bundle of His, or the bundle branches.[1] Structural abnormalities affecting the His-Purkinje system are generally believed to be progressive, so that Adams-Stokes attacks are more likely to develop in such cases; in contrast, abnormalities affecting the atrioventricular node have a more benign course.[2] The introduction of His bundle electrography has allowed cardiologists to define

the site of the block more accurately and so to increase our understanding of the conventional, surface electrocardiogram,[3] but use of the surface electrocardiogram alone to distinguish nodal from His-Purkinje disease remains difficult and may occasionally be misleading. Nevertheless, some guidance[1] may be given by the width of the QRS complex (narrow complexes suggesting that the block is nodal) and the response of the heart rate to atropine and to exercise (failure of response to either of these manoeuvres suggesting His-Purkinje disease). If the heart rate is over 50 per minute that, too, may indicate that the block is located in the atrioventricular node.

The use of these variables to distinguish dangerous from benign complete heart block has recently been questioned.[4] In the 29 patients studied by Lane and Kennelly[4] the resting heart rate did not distinguish symptomatic from asymptomatic patients with either wide or narrow QRS complexes. Intracardiac recordings of the site of the block were also unhelpful. They claimed that the best test for identifying patients at risk of serious symptoms was the response of the heart to right ventricular pacing. In technical terms, the mathematical product of the ventricular escape time and the length of the overdrive cycle seemed to have a predictive value. Nevertheless, while there were statistically significant differences between groups the overlap was too great for this test alone to be of clinical value in individual patients.

For the time being, therefore, the physician wanting guidance on whether or not to advise prophylactic pacing for his patients will have to continue to use his clinical judgment. One group of patients who may not require permanent pacing are those whose block is localised to the atrioventricular node and in whom the heart rate accelerates with atropine or exercise. Failure of the ventricular rate to be suppressed by right ventricular pacing strengthens the argument. Nevertheless, whenever a decision is made not to advise insertion of a pacemaker the patient's progress must be followed carefully.

In patients in whom treatment is considered justifiable the best treatment is implantation of a permanent pacing system using either an endocardial or epicardial electrode. Should pacing not be possible, patients whose heart rate is increased by intravenous isoprenaline[5] may benefit from treatment with a long-acting preparation of the drug.[6]

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Drug toxicity studies and the clinical pharmacologist

We have no reliable estimate of the number of hospital admissions in Britain each year attributable to drug reactions. One report[1] suggested that about 3·5% of medical admissions are of this type, with a further 2·5% attributable to deliberate drug overdosage[1] (relatively few patients being admitted to wards other than medical—or paediatric—for these reasons[2]). Most drug reactions are mild, but a few may be severe enough to threaten life—a possibility more likely in patients with serious or terminal illness.[3] Nevertheless, in such circumstances these risks may seem worth taking when balanced against possible benefits.

Early in the 1960s Wade and his colleagues in Northern Ireland showed how variable was the prescription of oral hypoglycaemic drugs in the province.[4] Such variability has since been confirmed for many other drugs elsewhere. It stems as much from differences between doctors as from fluctuations in the local prevalence of disease. More recently McDevitt and his colleagues[5] and the Liverpool Therapeutics Group[6] have shown that many patients in domiciliary practice who take digoxin regularly do equally well without it. Yet here is a drug with a substantial risk of undesired effects. No doubt before long some patients with cardiac failure will be reported not to need long-term diuretic treatment once the initial episode has been treated adequately. Similarly, at least in the United States, most maturity-onset diabetic patients who adhere to their diets have been shown not to need oral hypoglycaemic agents.

Clearly we need more studies of drug-prescribing habits, aimed particularly at discovering the reasons why some doctors are enthusiastic and others conservative drug prescribers. Also needed are more studies into long-term use of potentially toxic drugs such as steroids (both oral and topical), anticoagulants, and diuretics. Despite much interest in the last few decades there have been few systematic studies on the frequency of adverse drug reactions in defined populations. The Boston Collaborative Drug Surveillance Program has a wealth of information and continues to publish regular reports. Much simpler studies can, however, be undertaken for a more limited number of drugs.[7 8] Unfortunately, few groups in Britain have undertaken this type of work, though the recent report in the *BMJ* of acute toxicity from frusemide in patients in hospital is an example of the type of study we urgently need.[9] The problems of quantification of risk are much greater in outpatients. Here Britain can take justified pride in the pioneering work done by Skegg and Doll,[10] with their study of over 40 000 patients in several general practices in the Oxford area.

In the future this type of study must, surely, become the province of the clinical pharmacologist. Professor M Rawlins of Newcastle has recently suggested that there is a case for devolution of some of the functions of the Adverse Reaction Subcommittee of the Committee on Safety of Medicines, which is based at present in London. At least part of this work could be undertaken more readily by regional clinical pharmacology centres with their improved contacts with local prescribers. Such centres could collaborate in collating spontaneous reports of adverse effects from prescribers and share responsibility for conducting studies on acute drug toxicity. This could be done at minimal cost and would rapidly increase our knowledge of the subgroups of the population at high risk of reasonably common serious adverse drug effects. The experience gained could then be put to immediate practical use since it would help in developing the postmarketing surveillance studies we so urgently need for new products.

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[9] Lowe, J, *et al*, *British Medical Journal*, 1979, **2**, 360.
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