treatment to monitor, as well as the consideration of keeping the whole device small and easily assimilable, the idea of a clock-face layout surrounding the current medical record card seemed the most economical (see figure).

The check-list clock.

There are twelve headings, six to do with the diagnostic process, four to do with the diagnostic process, and two involving both. They are as follows.

Presentation of the complaint. (Why has the patient come? Why now? Is there a firm traditional diagnosis? If not, what is the matter?)

Medical history.

Family history.

Recent life events. (For example, childbirth, marriage breakdown, bereavement, etc.)

Personality factors. (For example, stoicism, introspection, aggression, keeping doctor at arm's length.)

Doctor-patient relationship. (Does the doctor feel any strong emotion, such as anger, in the presence of the patient? If so, is it a symptom of the doctor or the patient? Is there any disinclination of the doctor to do a particular examination (for example, vaginal, rectal)? If so, why?)

Allergies. (Any external allergies, drug allergies or intolerances, psychological state likely to produce reactions to treatment?)

Drug vetoes. (Common examples listed.)

Drug interactions. (Common examples listed.)

Drug hierarchies. (For example, aspirin—indomethacin—newer antirheumatic drugs.)

Unconscious factors in prescribing. (Does a departure from the normal (strength) hierarchy in prescribing patterns reflect something in the doctor-patient relationship? For example, does the prescription of a potent drug reflect the doctor's need to be potent with that particular patient or does the prescription of a cheap preparation reflect the doctor's contempt?)

Working diagnosis and its therapeutic plan. (Is there, potentially, a fatal pay-off? How ill is the patient? (If there is doubt, has the blood pressure been taken?)

Initial experience with the check-list clock, placing the current medical record continuation card in the central part of the frame, has shown that this kind of review of the consultation conducted initially in an unstructured way has led to important changes in both diagnosis and treatment through the stimulation of further recall and understanding of important information and interaction which had previously been missed.

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When is significant not significant?

SIR,—The work of Sir Richard Doll and Mr Richard Peto (4 June, p 1433) raises several points of interest.

With figs 1 and 2 the reader is asked to bias his judgment in favour of a relation which the authors appear intent on proving; the broken lines used for five confidence intervals enforce this bias. However, the basis for this choice of "reliable" and "unreliable" SMRs is not indicated. Definition of statistical methods being poor, the authors would appear to have calculated regression lines using 11 pairs of variables. However, these calculations (using, presumably, the mean values for each specialty) fail to take into account the often large scatter of data about each group mean, this influencing in turn the error associated with the estimation of regression line gradients. Thus in using apparently grouped data for their calculations the authors would seem to be biasing the analysis, again in favour of demonstrating a significant relation between variables.

In their subsequent letter Mr Peto and Professor Doll (23 July, p 259) state that "moderate statistical significance (such as $P < 0.05$) [in association] often does arise as an artefact of chance." This statement is worthless. From the statistical estimation one is able to suggest that the observed data would have occurred by chance in less than one case in 20; if one is willing to accept the *predefined* risk of erroneous conclusion, then one can but conclude that certain relations have, or have not, been demonstrated in the sample group. It is irrelevant whether a causal relation can be established. The authors fail to define their level of significance *before* the analysis of their observations; as a consequence of this omission Professor Doll and Mr Peto in their paper appear to have succumbed to the serious error of choosing only those statistical estimations which suit their purpose, as suggested by Professor Hugh Dudley (2 July, p 47). Moreover, they conclude that "the excess of ischaemic heart disease and chronic bronchitis in single-handed general practitioners and the deficiency of ischaemic heart disease in hospital physicians and surgeons, and of chronic bronchitis in hospital physicians, may be attributed respectively to their above and below average consumption of cigarettes" (my italics); herein lies the common error of implicating causal relations between variables linked only by statistical estimation.

The example of throwing paired dice, as given by Mr Peto and Professor Doll in their letter, although correct probability theory, is an example of poor logic and misuse of statistical estimation. Of course the probability of throwing a double-six is about 0.03; however, this does not even suggest that the dice are biased because the probability of throwing any pair of numbers (for example, 4 on dice A and 1 on dice B) is about 0.03. The very suggestion that the judgment should be made on the basis of a single throw implies a failure to grasp an elementary principle of statistics—namely, that statistical estimations can be made only on the basis of populations of figures. The words of a great statistician, the late Sir Ronald Fisher, would be of undoubted value here: "... Nevertheless, in a real sense, statistics is the study of populations, or aggregates of individuals, rather than of individuals. Scientific theories which involve the properties of large aggregates of individuals, and not necessarily the properties of the individuals themselves ... are essentially statistical argu-

ments, and are liable to misinterpretation as soon as the statistical nature of the argument is lost sight of."

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Hallucinations in polymyalgia arteritica

SIR,—Polymyalgia arteritica has been commendably treated in some of your recent issues. One feature I did not observe: the occurrence of hallucinations in connection with this disease. During the last few years that I was head of the Medical University Clinic in Uppsala, we saw some 50 cases of polymyalgia arteritica. Visual hallucinations were not common but did occur: an old lady came into the clinic escorted by a St Bernard dog which was entirely fictitious. She wanted the dog placed in the bed next to her and this permission was of course granted. The following morning, after she had received, the previous day, a rather massive dose of cortisone, the dog had disappeared, much to her embarrassment. The subsequent treatment kept the dog away from her.

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Congenital dislocation of the hip

SIR,—With reference to Mr A J Harrold's excellent review of the problems in congenital dislocation of the hip (CDH) (23 April, p 1071) I would like to present a deviating opinion on a few points.

It is true that the incidence of CDH has increased in those clinics which have introduced the technique of Ortolani and Barlow in examining all newborn infants. It is not true, however, that this increase of incidence is due to "overdiagnosis," including completely healthy infants, as it has been demonstrated¹ that the condition in these children is closely related to CDH. All these children have unstable hips, some of which are dislocated or dislocatable at the moment of examination. There are no means, however, of distinguishing between those hips which will recover spontaneously and those in which complete dislocation will develop later on.

Should all these children be treated and is treatment harmless? Not only failures but also a dangerously high complication rate in the treatment of CDH have been reported.²⁻⁴ All these misfortunes, however, have occurred in children who have either been treated late or with rigid fixation. In a long-term follow-up⁵ 8-16 years after treatment it has been demonstrated that proper treatment is completely harmless.

Acetabular dysplasia is a common finding among the parents of children with CDH.⁶ A child with a minor degree of hip instability that will allow only partial dislocation (subluxation) can remain symptom-free throughout childhood. The resulting incongruence will lead to underdevelopment of the acetabular roof (dysplasia)⁷ and perhaps to early arthritis.⁸⁻¹¹ Treatment in a harmless splint for a few months will probably prevent this course.

The literature on the difficulties of early diagnosis and treatment is abundant. During the past few years, however, it has become evident that the difficulties can be overcome and by

now it is a common experience that most cases of CDH can be diagnosed at birth¹²⁻¹⁵ and that proper treatment will lead to the development of completely normal hips.³

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- ¹³ Finlay, H V L, et al, *British Medical Journal*, 1967, **4**, 377.
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Management of intractable hiccup

SIR,—I hesitate to add to the list of drugs and other treatments of hiccups compiled by Dr B W A Williamson and Mr I M C Macintyre (20 August, p 501), but they mention methylamphetamine only to record failure in the one case they describe. I have used it to treat hiccups scores of times during the past 25 years and have rarely seen it fail to relieve the symptom. I inject the drug very slowly intravenously until the hiccups stop, usually after 6-12 mg has been given.

Unfortunately, as a consequence of restrictions introduced because of social abuse, it has become increasingly difficult to obtain the drug in this hospital, especially after normal working hours, so I now use chlorpromazine 25 mg intravenously. However, most hiccups that I see occur during or shortly after recovery from general anaesthesia for upper abdominal operations, when it can be a nuisance to the surgeon or distressing for the patient. Chlorpromazine may not be desirable at these times, but I have found that pentazocine 30 mg intravenously stops these hiccups quickly and effectively. It stops the more prolonged hiccups that occur in uraemia and the diaphragmatic irritation of pneumonia and sub-diaphragmatic infection or haemorrhage, but its short action (1-3 h) makes it less practical in these situations than chlorpromazine.

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Microangiopathy and diabetic gangrene

SIR,—In your leading article (18 June, p 1555) you assert that microvascular changes in the legs of diabetics predispose directly to gangrene. There is no functional test for the presence of microangiopathy in the leg and the histological evidence is conflicting. Nielsen¹ concluded that microangiopathy was not a cause of pedal gangrene. Du Plessis² reported

that half his patients with foot lesions had histological changes and Moore and Frew³ claimed an association between vascular changes and the presence of foot lesions, although this was true only if the most severe cases of microangiopathy were considered. My own studies have demonstrated capillary and arteriolar changes in half the diabetics studied but not more commonly in patients with ulceration or gangrene of the foot.

I believe that the evidence presently available is not sufficiently strong to justify your statement. Further, labelling a patient as suffering from "small-vessel disease" diverts attention from potentially remediable large-vessel disease.

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- ² Du Plessis, D J, *South African Journal of Surgery*, 1970, **8**, 29.
- ³ Moore, J M, and Frew, D O, *British Medical Journal*, 1965, **2**, 19.

Gastrointestinal symptoms of digoxin toxicity

SIR,—The article by Dr A Smith and Professor M D Rawlins (30 July, p 309) draws attention to the symptoms of nausea, vomiting, and anorexia as indications of digoxin toxicity and states that the digoxin dosage should be reduced if any of these symptoms occur. We would like to report our experience of assessing these symptoms with respect to the plasma digoxin concentration (PDC).

This laboratory offers a service for the measurement of digoxin in plasma and clinicians are asked to complete a specially designed request card when forwarding a blood sample for analysis. The request card includes a simple tick sheet for common signs or symptoms of cardiac drug toxicity, including gastrointestinal symptoms, together with a three-point assessment of their severity (minor, moderate, or severe).

Details of gastrointestinal symptoms and the PDC have been collated for 412 consecutive requests and are summarised in the accompanying table; symptomatic patients are those reported as having one or more of the symptoms nausea, vomiting, or anorexia at the time the analysis was requested.

The mean PDC of the symptomatic patients was significantly higher than that of the asymptomatic group ($P < 0.01$) and there was an association between the symptoms and increasing PDC ($\chi^2 25.23$; $P < 0.05$). However, there was a wide spread of results among patients complaining of the symptoms and 24% of these patients had a PDC of 1.3 nmol/l (1 ng/ml) or less. Furthermore, there was no significant difference between the mean PDC of those patients with minor symptoms and that of those displaying moderate or

severe symptoms ($P > 0.15$). These results are almost identical with our previous analysis of almost 2000 observations, based on a less detailed assay request card, in which an association between PDC and electrocardiographic signs of toxicity was shown.¹

Since the symptoms can be caused by congestive heart failure² they are often difficult to evaluate in patients who have been receiving digoxin for some time and could themselves be relieved by an increase in digoxin therapy. Indeed, our results suggest that these symptoms are encountered frequently in patients with low plasma concentrations of the drug and as a diagnostic aid they might be misleading.

In our experience the most prudent course of action when faced with a patient complaining of these symptoms is to determine the PDC, omitting digoxin therapy pending the result. Together with the evaluation of a carefully documented history and other clinical measurements we have found the PDC useful in assessing the probability of the symptoms being an indication of digoxin toxicity.

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- ¹ Holt, D W, PhD Thesis, University of London, 1976.
- ² Chung, E K, *Digitalis Intoxication*. Amsterdam, Excerpta Medica Foundation, 1969.

The sleepless child

SIR,—Dr H Jackson (20 August, p 509) attributes much childhood insomnia to "toddler negative behaviour" by which the child exerts his individuality and then goes on to enumerate treatment in the form of constructive play, hospital admission, or drugs, none of which seem appropriate to his putative cause.

Night crying must be a common complaint. Certainly I have dealt with many cases as a general practitioner as well as one as father of a 10-month daughter and another as uncle of a 20-month niece. My own hypothesis is that regular night crying is a Pavlovian conditioned reflex. The child wakes and cries (perhaps initially because of an ailment); the mother comes to him with loving attention and a drink; the child repeats his crying nightly and each time is quickly rewarded. With such regular reinforcement the reflex is soon established. It follows that the logical treatment is simply a matter of deconditioning.

It is explained to the mother how she has unwittingly trained her child to cry and that he must now be taught that crying is fruitless. On the appointed night the neighbours are warned that there will be an unusually protracted din but that this is entirely the fault of the doctor. The child cries. Father stamps in, flings down the side of the cot, slaps him once but firmly, flings up the cot side, and

Plasma digoxin concentration and incidence of gastrointestinal symptoms in 412 patients

Group	No	‰ of total	Plasma digoxin concentration (nmol/l)	
			Mean $\pm$ SD	Range
Without gastrointestinal symptoms	308	74.8	2.04 $\pm$ 1.16	0-7.3
With gastrointestinal symptoms	104	25.2	2.32 $\pm$ 1.34	0-6.6
Total	412	14.3	2.18 $\pm$ 1.29	0-6.6
Minor	59	10.9	2.52 $\pm$ 1.42	0.4-5.8
Moderate to severe	45			

Conversion: SI to traditional units—Digoxin: 1 nmol/l $\approx$ 0.78 ng/ml.