clinically relevant examples than the purely scientific questions posed by Montgomery and colleagues.

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1 Montgomery H, Hunter S, Morris S, Naunton-Morgan R, Marshall RM. Interpretation of electrocardiograms by doctors. *BMJ* 1994;309:1551-2. (10 December.)

Formal teaching can reduce serious errors in interpretation

EDITOR,—We were not surprised by the results of Hugh Montgomery and colleagues' study of doctors' ability to interpret electrocardiograms.¹ We have audited the ability of senior house officers to interpret electrocardiograms in an accident and emergency department.² We found that only 114 of 245 traces were interpreted correctly but that management was seriously affected by misinterpretation in only eight cases. After formal teaching and the issue of guidelines serious errors of misinterpretation fell by half to four out of 242 (1.6%; confidence interval 0.7% to 4%).

We also tested the ability of cardiographers, cardiac technicians, nurses in the coronary care unit, anaesthetic registrars, and medical senior house officers to interpret electrocardiograms; we used a standard set of 20 electrocardiograms. All the subjects were asked to report the rate, rhythm, QRS axis, and abnormalities. Answers were graded 1-4, where 1=complete concordance; 2=minor disagreement (for example, first degree heart block); 3=disagreement (for example, left ventricular hypertrophy); and 4=appreciable disagreement potentially affecting management (for example, ST segment elevation). All the paramedical staff were better than the doctors and made fewer serious errors (table; $\chi^2=27.2$, $P<0.01$).

We believe that these results are explained by the fact that formal training in interpretation of electrocardiograms is given to all the groups of staff that we tested except the doctors. It is an indictment of medical education that doctors can interpret electrocardiograms less well than paramedical staff. Should this interpretation be part of the undergraduate curriculum? We believe that it should certainly be part of postgraduate training for doctors in clinical specialties. It is particularly important when decisions about giving thrombolytic treatment to patients with acute myocardial infarction depend partly on interpretation of the electrocardiogram. If general practitioners begin to use thrombolysis in the community they must be capable of recording and interpreting an electrocardiogram.

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Grade given to interpretation by different groups of staff of sample of 20 electrocardiograms (ECGs)*

Grade*	Registrar (n=6)	Nurse in coronary care unit (n=6)	Cardiac technician (n=6)	Cardiographer (n=5)	Senior house officer (n=6)
1	53	46	51	51	47
2	13	19	8	6	10
3	18	27	43	26	33
4	36	21	23	17	30
Total ECGs	120	120	120	100	120

*See text for details of grades.

Continuous ambulatory electrocardiography in elderly people

EDITOR,—I J Räihä and colleagues' paper on the predictive value of continuous ambulatory electrocardiographic monitoring in elderly people digresses and therefore arrives at several misleading and clinically unsound recommendations.¹ In a retrospective study the authors found several abnormalities on Holter monitoring that were associated with a high odds ratio for the risk of cardiac death over 10 years. The subjects had a mean age of about 75.

The predictive value—as distinct from the odds ratio—is a statistical term indicating the likelihood that a patient in whom the result of a test is abnormal will have the disease in question (or, in this case, a poor outcome). With respect to the three factors found by Räihä and colleagues to be associated with a poor outcome, 95 patients had >100 ventricular ectopic beats during the day, of whom 32 died of cardiac causes over the next 10 years; 88 had multifocal ventricular ectopic beats at night, of whom 31 died of cardiac causes; and 79 had sinus pauses of >1.5 s during the day, of whom 34 died of cardiac causes. Thus the predictive value that a given patient with one of the identified factors on Holter monitoring would die of heart disease over the ensuing 10 years was only slightly greater than 1 in 3.

Even if we assume that these markers, which were defined arbitrarily and post hoc, are indeed valid, what possible benefit could there be in determining that a given 75 year old has a 1 in 3 chance of dying of heart disease during the next 10 years? To imply that Holter monitoring should be performed routinely to stratify the risk in elderly people and to recommend that "patients with these findings should be evaluated for silent ischaemic heart disease and sick sinus syndrome" seems the height of folly. The cardiological community generally accepts that giving antiarrhythmic drugs to patients with ventricular extrasystoles does more harm than good, and Räihä and colleagues themselves state that "the benefit of treating silent ischaemia in elderly patients is unclear." Adherence to the authors' clinical recommendations would not only be economically wasteful but also give rise to much unnecessary anxiety in a population already so concerned by its declining sense of wellbeing.

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1 Räihä IJ, Piha SJ, Puukka P, Sourander LB. Predictive value of continuous ambulatory electrocardiographic monitoring in elderly people. *BMJ* 1994;309:1263-7. (12 November.)

Weight in infancy and coronary heart disease in adult life

EDITOR,—C H D Fall and colleagues' paper assumes that low weight at 1 year is a result of undernutrition and that greater weight is a result of better nutrition.¹ Unfortunately, weight gain in the first year of life is not as straightforward as this.

When weight profiles of individual babies are examined in the early months considerable shifts across centile lines take place in both upward and downward directions as the baby leaves behind intrauterine influences on growth and grows under the direction of his or her own genetic machinery.² In a study of weight gain over the first year in 66 normal term infants only 12% kept roughly in the same "centile channel" in which they were born; 28% showed downward crossing of centiles, while most showed upward crossing of centiles.³ The greatest shift occurred in the first three months.

Some babies of low weight at 1 year will therefore be those who were light at birth, for physiological or pathological reasons, and who maintain this phenotype. Some will have become small, having crossed various numbers of centile channels. These have not all necessarily failed to thrive but have simply recaptured their true growth phenotype. At the same time, there are those of higher weight who have continued a high weight profile at birth while others have reached this weight, having caught up from being relatively light at birth. It is therefore important to know more about the detailed patterns of growth in the babies in the authors' study who weighed ≤ 18 lb (≤ 8.2 kg) and ≥ 26 lb (≥ 11.8 kg). Is metabolic disadvantage relevant to those whose weight continues in a low channel or in those whose weight has fallen from a higher centile and, if so, which type? Likewise, what of the protection conferred on those who are of higher weight at 1 year?

There is further confusion. In some of the earlier studies by the authors low birth weight was linked with later risks of high blood pressure and ischaemic heart disease.⁴ Yet a typical pattern of early weight gain in growth retarded neonates is to catch up—to reach a higher centile than they were at birth.² This phenomenon is also found in some infants of appropriate weight for gestation. Some heavier children at 1 year will therefore probably be those who have caught up from intrauterine growth retardation. On the one hand, these are considered to be at risk of later cardiovascular disease, yet we now hear that they seem to be advantaged in being less vulnerable to later coronary artery disease.

It is an intriguing possibility that early growth may somehow be related to programming of later disease. But the literature on the subject contains contradictions that need to be explained.

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Simple aspiration for spontaneous pneumothorax

EDITOR,—Editor's Choice in the issue of 19 November describes the clinical trial reported by John Harvey and Robin J Prescott—of simple aspiration versus intercostal drainage for spontaneous pneumothorax in patients with normal lungs¹—as "methodologically strong." I challenge that view.

Firstly, patients "with signs of a tension pneumothorax" are said to have been excluded from the trial, presumably because intercostal tube drainage is considered to be mandatory in all such cases. But the criteria for that diagnosis are not stated, and at