schedules, etc, are required. Unless proper management skills are acquired by laboratory staff more and more of these roles will be taken over by outside administrators. Medical laboratories are complex organisations which require a great deal of scientific skill allied to a high level of managerial skill—by and large the organisation works well. It is unlikely that the vast changes which have occurred in laboratories over the past 20 years have been equalled anywhere else in the Health Service, either for the rate at which they have occurred or for the ease with which they have occurred, a matter on which the professional bodies concerned should be congratulated. There has been the right training—the right skills have been there when they were needed, unlike the rest of British industry, which even now is short of skilled workers. My hope is that the professional bodies will realise what has been achieved by co-operation in the past and will come even closer together to continue the record of success and unity within medical laboratories.

The past 20 years have seen great change; who knows, the next 20 years may see even greater changes. For this reason the professions within medical laboratories should welcome HSC(1S)16, which with minor modifications is already practised in the large proportion of the leading laboratories within the National Health Service.

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SIR,—We noticed with interest the statement in your leading article (21 October, p 1108) that "divisions between the work of junior scientists and technical staff (medical laboratory scientific officers, MLSOs) are blurred."

Ignoring the gratuitous misuse of the terms scientific and technical we would like to make an observation. On the last occasion that we advertised for a senior medical laboratory scientific officer in chemical pathology we received an application from a senior biochemist. Could it be that there is no distinction between the work of the "scientists" and MLSOs and that the appearance of blurring only occurs where artificial distinctions are contrived?

B WEEKS
R P WELCH

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SIR,—Your leading article (21 October, p 1108), while giving an admirably objective review of the proposals for reorganisation within the NHS scientific service, regrettably deteriorates towards the end into a rather sad attempt to irritate further—or perhaps it might be more correct to say "resurrect"—antagonisms which have sometimes existed between medical and non-medical staff in hospital laboratories.

Perhaps it might be worth pointing out in the context of your naval analogy that the *Ark Royal* has now been scrapped and there is no longer a captain in command of her. Similarly, many of us hope that the old inter-professional antagonisms are also well on the way to being obsolete and that instead of a crew of officers and ratings (or, as a few people tend to see it, "us and them") the pathology service is moving into an era when laboratories are indeed managed by consensus as a team effort directed towards the ultimate benefit of the patient.

Irritating and unsubstantiated comments such as that to the effect that graduates only enter the medical laboratory scientific officer grades because they are "unable to obtain posts as scientists" are doubly regrettable both for their inaccuracy and for their apparent attempt to keep alive an antagonism which can benefit nobody and which few enough of either professional group seem to desire. The attitude of most medical laboratory scientific officers towards pathologists was made perfectly clear in the Institute of Medical Laboratory Sciences' statement "Pathologists and ourselves."[1] From this it is perfectly clear that the profession has a deep-seated desire to work in friendly partnership with pathologists but that it does suffer from time to time from misinterpretation of its attitude by some of those with whom it seeks to co-operate.

Surely there can be no suggestion that clinical pathologists feel their role in any way threatened by the non-medical staff who do the vast bulk of the scientific work of medical laboratories. If that role is in any way threatened it would seem to many of us that the threat comes rather from the "scientists" and biochemists to whom you refer who are employed under Whitley Council PTA regulations rather than the large majority of graduates entering the service who are employed under PTB regulations as medical laboratory scientific officers.

It is a pity that those who seek to perpetuate dissension between the two main professional groups in the medical laboratory do not come forward and identify themselves by name in order that their colleagues among both medical and non-medical staff may know just who it is who still seeks to stir the troubled waters of a recent past which most of us believe would be better forgotten.

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[1] *Gazette of the Institute of Medical Laboratory Sciences*, 1977, **21**, 238.

Effect of propranolol on HDL cholesterol concentrations

SIR,—We were interested in the report of Dr P J Jenkins and others (5 August, p 388) concerning the inverse correlation between plasma high-density lipoprotein (HDL) cholesterol concentration and the severity of coronary artery disease. However, in the same issue Dr A Helgeland and others (p 403) reported an association between low HDL cholesterol and treatment with propranolol and thiazide diuretics. This raises an important question, since subjects with severe coronary artery disease are more likely to be treated with propranolol.

We have studied 37 male Caucasian patients with coronary artery disease who were euthyroid, non-diabetic, and on no lipid-lowering agents. The diagnoses were: myocardial infarction (12), myocardial infarction and angina (13), angina (10), and aortocoronary bypass graft (2). Propranolol in the dosage range of 40-240 mg/day was being administered to 16 patients for treatment of angina and/or hypertension. Fasting blood samples were obtained on three different occasions separated by at least a four-day interval. The results obtained are shown in the table.

Only two subjects in the propranolol-treated group received thiazide diuretics, and the HDL cholesterol concentration in both was higher than the mean of the group.

It would be of interest to know if propranolol therapy can account at least in part for the lower HDL cholesterol observed in subjects with severe coronary artery disease.

D STREJA
DAVID MYMIN

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Findings (mean ± SEM) in 37 male patients with coronary artery disease

	Propranolol	No propranolol	Significance
No of subjects	16	21	—
Age (years)	55 ± 2	53 ± 2	NS
Weight (kg)	77 ± 2	79 ± 2	NS
Sum of four skinfolds (mm)	46 ± 2	46 ± 3	NS
Plasma glucose (mmol/l)	5·38 ± 0·17	5·38 ± 0·22	NS
Plasma insulin (mU/l)	15·1 ± 1·4	14·9 ± 1·1	NS
Plasma triglycerides (mmol/l)	2·33 ± 0·18	2·34 ± 0·57	NS
Plasma cholesterol (mmol/l)	5·70 ± 0·21	6·03 ± 0·23	NS
Plasma LDL cholesterol (mmol/l)	3·76 ± 0·23	3·86 ± 0·23	NS
Plasma HDL cholesterol (mmol/l)	0·86 ± 0·03	0·98 ± 0·04	$P<0·05$

Conversion: SI to traditional units—Glucose: 1 mmol/l ≈ 18 mg/100 ml. Triglycerides: 1 mmol/l ≈ 89 mg/100 ml. Cholesterol: 1 mmol/l ≈ 39 mg/100 ml.

Terminal symptoms in children dying suddenly and unexpectedly

SIR,—The Department of Health and Social Security's multicentre study of postneonatal mortality must be welcomed as a major long-term commitment to the elucidation of the causes and prevention of cot deaths. However, the study group's preliminary report (4 November, p 1249) has a number of aspects which give cause for concern. These relate both to the methodology of the study itself and to the conclusions regarding intervention that derive from it.

In respect of methodology, as the investigators acknowledge, the retrospective assessment of symptoms is difficult and is especially problematic in this context because parents are likely to search for symptoms to explain their child's death. The symptoms sought are also likely to be open to considerable variation in interpretation—for example, "irritability" or "altered character of cry." The controls selected were apparently matched only for age, although other variables (for example, sex, social class) might be expected to affect the frequency of symptoms and their reporting. Further, the symptoms recorded for the controls were those present at the time of interview, which might be more readily recalled than or differently interpreted from the retrospective data obtained for the study group. Taken jointly, these criticisms would seem to cast some doubt on the reliability of the findings regarding the extent and severity

of the symptoms preceding sudden and unexpected deaths at home when these are compared with their general rate of occurrence in the child population. Since the conclusions of the study are based on these findings some independent assessment of their reliability is desirable.

The conclusion of this paper is that the prevention of cot deaths is dependent firstly on the recognition by parents of the significance of certain non-specific symptoms and on their decision to involve the primary care services, and secondly on the efficacy of medical intervention at this stage. Consideration does not appear to have been given to the difficulties that will face parents who are asked to undertake such close surveillance of their children or the anxieties that this is likely to cause; nor has any estimate been made of the presumably very large proportion of cases in which this will prove "unnecessary." Conversely, the implications of the increased work load for the primary care services has yet to be determined. Concerning intervention, the investigators recognise that the value of either hospital referral or drug therapy is at present doubtful, leaving close supervision and observation of the child at home as the only alternative. It has yet to be established what this can achieve.

By the nature of the condition, sudden and unexpected death in children is an exceedingly difficult area of study, especially in its community aspects, and many of the criticisms raised here are clearly recognised by the study group and are the subject of further investigation. But because of these unresolved difficulties it would seem premature to urge the "need to improve the recognition by both doctors and parents of non-specific symptoms as markers of severe illness in young children and their understanding of the necessity for rapid and appropriate action."

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Thyroid disease and pregnancy

SIR,—We read with interest your leading article on this subject (7 October, p 977). You state that during pregnancy "free thyroid hormone concentrations are normal." The paper[1] to which you refer states that "direct measurements of free serum T4 provide values similar to those seen in the non-pregnant state . . . but there is some evidence that free T3 levels fall below normal and remain low until term."

We have recently completed a study of free T4 in the serum in the three trimesters of pregnancy using the Corning Immophase (^{125}I radioimmunoassay) method. The data provided with the kit currently show that the normal range and the range of values obtained during pregnancy are the same. Our results, which are in conflict both with those to which you refer and also with the Corning data, are summarised in the table. They show that in the second and third trimesters approximately one-third of the values obtained will be in the hypothyroid range.

Mean free T4 levels (± SD) in pregnancy

	No of patients	Free T4 (pmol/l)
Controls	22	16·7 ± 2·4
1st trimester	22	16·7 ± 3·5
2nd trimester	28	12·5 ± 4·5*
3rd trimester	19	9·9 ± 2·7†

*Significant difference from control mean, $P < 0.001$.
†No significant difference from 2nd trimester mean.
Conversion: SI to traditional units—T4: 1 pmol/l ≈ 0·08 ng/100 ml.

Reports that serum free T4 concentrations were significantly lower than normal in pregnancy first appeared as long ago as 1958[2] and 1962.[3] Our results indicate that values not significantly different from normal can be expected only in the first trimester. The combination of conflicting results and a novel technique for measuring free T4 could have a methodological basis. In order to clarify the situation our sera are now being assayed for free T4 and free T3 by the dialysis method,[4] which is generally considered to be the reference method for these measurements.

If the dialysis measurements confirm our observation that the serum free T4 level falls during the second trimester and remains low until term then the widely taught concept that the free T4 concentration remains normal during pregnancy will have to be changed. Surely this will lead to some exciting new ideas about transport and action of thyroid hormones at cellular levels.

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[1] Tunbridge, W M G, and Hall, R, *Clinics in Obstetrics and Gynaecology*, 1975, **2**, 381.
[2] Robbins, J, and Nelson, J H, *Journal of Clinical Investigation*, 1958, **37**, 153.
[3] Sterling, K, and Hegedus, A, *Journal of Clinical Investigation*, 1962, **41**, 1031.
[4] Ekins, R P, and Ellis, S M, *Thyroid Research*, 1976, 597.

SIR,—I share the view of Dr R T Cooke (11 November, p 1370) that the published experience of postpartum hypothyroidism may be just the tip of the iceberg. Over the past 18 months in a general medical practice I have seen four patients[1] with spontaneously remitting hypothyroidism presenting after delivery. I have also seen other women on thyroid replacement therapy for many years whose hypothyroidism was diagnosed after a delivery.

As the natural history of postpartum thyroid disorders is not known it is important that their remitting nature should be more widely appreciated.

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[1] Hoffbrand, B I, and Webb, S C, *Postgraduate Medical Journal*. In press.

Towards positive diagnosis of the irritable bowel

SIR,—We were interested to read the article on this subject by Dr A P Manning and others (2 September, p 653), but had difficulty in interpreting the results.

Although it is stated that irritable bowel syndrome (IBS) is itself a diagnosis of exclusion, it seems curious that 27 patients in the original sample of 109 were excluded because no definite diagnosis had been reached. Did the symptoms in this group of patients, and those in the group of 14 excluded because of (perhaps coincidental) diverticular disease, differ from the symptoms of the group of 32 with IBS?

Is it not possible that patients with upper-bowel disease present with a different range of symptoms from those in patients with conditions affecting mainly the lower bowel? Twenty of the 33 patients with an organic diagnosis had conditions affecting the upper gastrointestinal tract and the differences between the symptomatology of this group and that of the group with IBS could be entirely ascribed to this distinction. Perhaps the article could be retitled "Towards positive diagnosis of upper bowel disease"?

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**We sent a copy of this letter to Dr Manning and his colleagues, whose reply is printed below.—ED, *BMJ*.

SIR,—The 27 patients who were omitted from our study were a heterogeneous group. Some had functional dyspepsia, some psychoneurosis, and in some no definite conclusion was reached in the clinic—for example, because the symptoms resolved spontaneously or the patient defaulted from follow-up. Statistically they were different from the IBS group, but it is hard to draw any conclusions from this. There were only 14 patients in the diverticular disease group—not enough for meaningful statistical comparisons with the IBS and organic groups.

We agree that the IBS patients could be considered as a group of people with mainly colonic and rectal dysfunction whereas the organic patients were heterogeneous but predominantly with upper-gut problems. This does not alter the validity of our findings. In clinical practice it is often not at all obvious if a patient's abdominal pain is coming from the foregut, midgut, or hindgut (colon pain is often felt at or above the umbilicus). To identify the hindgut as the source of pain should not only save unnecessary cholecystograms, barium meals, endoscopies, and even intravenous pyelograms but also allow the doctor to explain the probable nature of the disorder *at the first interview*. It may, of course, be necessary to exclude an organic lesion in the hindgut, but there are usually clues to this such as blood in the stool, weight loss, or sigmoidoscopic abnormalities.

In our experience patients with IBS often suffer from gastric symptoms which improve when the IBS is treated, and there is experimental evidence that upper-gut dysfunction is common in IBS. These cases of IBS are missed unless the doctor asks colon-directed questions.

Our statement that the IBS is a diagnosis of exclusion was a reference to conventional thinking. We challenge this attitude and suggest that doctors should have more confidence in their history-taking. By spending more time on the history they can not only avoid negative investigations and referrals but also increase their job satisfaction.

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