seek evidence about standards and assessment methods in its forthcoming evaluation of the Apothecaries' examinations. It will certainly ask how the society addresses the difficult issue of standard setting when the pool of candidates is probably of lower overall ability than that in a medical school—a problem noted by an inspector in 1942 (addendum to the minutes of the GMC, 1942-6, p 386). It will want to know what explicit criteria have been established for setting pass levels in each subject in view of the overwhelming evidence that examiners, left to themselves, are not good at this—pace Dr J B Ilangaratne, a belief in the infallibility of experienced examiners is not sustained by the research.

The inspectors will also require figures for the reliability of the various components of the examinations in terms of interexaminer agreement and consistency. And they will find it helpful to know the results of analyses of the written and clinical components, based on generalisability theory, to help them estimate the precision of the whole procedure. An inquiry of this nature would certainly not lead me to conclude that "the GMC is an inadequate guardian of the standards," as Dr W F Whimster suggests.

Such questions are particularly appropriately addressed to an external examining body which, having no background information on its candidates, can be expected to adopt a most professional approach to the issues of reliability, validity, generalisability, and fairness. Answers to these questions would have more effectively refuted the criticisms in my original paper, themselves based on both the theory and practice of assessment, than unsubstantiated assertions that the society "has provided a diploma of a high standard since 1815" like some sort of royal grocer.

Although about 20 students a year qualify solely by virtue of one of the non-university licensing bodies (not 93 as stated by me in a word processed error), many more who have failed their final university examinations use this qualification to avoid being referred for a period of extra study, before retaking their university examinations. The figures from Cambridge show that over a third of students who failed one or more parts of the final examination in 1979-84 passed one of the non-university licensing bodies' examinations before finally succeeding in their own universities' exams; 84% of them passed the LMSSA examination.

Typically this mechanism allowed these students to practise six or 12 months earlier than would have been possible otherwise. Worryingly, a few of these passing students continued to fail their university examinations years after qualifying in this way.

The question whether such an additional route to medical qualification should exist at all for British students remains. Mr Southwood suggests that it is a great waste of public money if medical students "are denied the chance of qualifying after a long period of study." Dr Philip H Willcox (25 April, p 1096) puts in a plea for hard up students who "need to qualify quickly to obtain hospital appointments by a certain date" and who "cannot afford a delay of six months." Dr N R C Robertson (p 1096) offers a perplexingly double edged view, that the mechanism is needed because "finals are a poor way of assessing fitness to practise." These are strange arguments indeed. Surely the students have had a chance of qualifying, through their medical school? The question is, how many chances should they have? And it is arguably an even greater waste of public money, ultimately, if unfit students are permitted to qualify as doctors. But in a time when many think that we are training too many doctors, perhaps this policy decision should be taken by a more publicly accountable authority than the society.

Although in the eyes of the master I am only "a medically unqualified and uninformed source," by my definition of a back door, this is one. And it is up to the GMC's education committee to collect and evaluate evidence about the charge of possible lower standards. Our aim must be to ensure that our system of certification is as accurate as possible, in so far as fit students must pass while unfit ones fail. Sentimental appeals against "homogenisation" of examinations (25 April, p 1096) do not help. What is needed is support for increased professionalism in this important subject.

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Lower oesophageal contractility as an indicator of brain death in paralysed and mechanically ventilated patients with head injury

SIR,—Like Drs M E Sinclair and P M Suter (11 April, p 935) we have also been studying patients with severe brain damage but have failed to find an association between the absence of spontaneous lower oesophageal contractions and brain stem death. In three of four patients studied to date, all of whom were receiving mechanical ventilation but no sedative or neuromuscular blocking drugs, spontaneous lower oesophageal contractions persisted after the presence of clinical criteria of brain stem death had been confirmed.¹

The first patient, a 35 year old man, was admitted after acute haemorrhage into an inoperable intracranial tumour. Oesophageal monitoring was started shortly afterwards, when motor responses to noxious stimuli were present and the pupils were reacting to light; the frequency of spontaneous lower oesophageal contractions was 2.1/min. Twenty four hours later the patient's condition deteriorated suddenly: he became unresponsive and his pupils were found to be fixed and dilated. Though the frequency of spontaneous lower oesophageal contractions decreased, they were still present at a frequency of 0.3/min when brain death was formally diagnosed.

The two other patients, both young men, had sustained severe head injuries in road traffic accidents. The presence of clinical criteria of brain stem death was confirmed 36 hours (case 2) and 72 hours (case 3) later; in addition, no identifiable peaks were present when the brain stem auditory evoked responses were recorded.² Over the next hour the average frequencies of spontaneous lower oesophageal contractions were 3.8/min and 1.3/min, respectively.

These findings contradict those of Drs Sinclair and Suter. One possible explanation is that their patients were paralysed and hyperventilated and sedation was "given as indicated clinically," a state similar to that during anaesthesia, when suppression of oesophageal contractions might be expected.³ Indeed, we regard as potentially dangerous their implication that a test that "may be very useful in the early identification of brain death" may be used in patients who are sedated.

Our findings also fail to support the suggestion that an intact pathway between brain and oesophagus is necessary for spontaneous lower oesophageal contractions to occur, although impulses transmitted through the vagi could modulate intrinsic activity in the oesophageal nerve plexus, altering the frequency of such contractions. The absence of spontaneous lower oesophageal contractions during deep anaesthesia, when cortical and brain stem electrical activity is modified but not obtunded,⁴ may reflect a combination of decreased vagal activity and direct depression

of the oesophageal nerve plexus by circulating anaesthetic drugs.

Irrespective of the mechanism by which spontaneous lower oesophageal contractions are generated, we cannot accept the suggestion of Drs Sinclair and Suter that measuring such contractions provides a means of predicting, or even confirming, the presence of brain stem death.

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Alcohol and ischaemic heart disease in middle aged British men

SIR,—There is a well established association between moderate alcohol intake and reduced incidence of ischaemic heart disease, which has been well reviewed.^{1,2} Several misconceptions exist among doctors, however, concerning this association.

There is a widespread belief that the association is present only in geographical studies, but in fact the early geographical evidence has been confirmed by case-control and cohort studies. In addition, many doctors believe that the association is probably spurious owing to the association of teetotalism with other risk factors or to sick people giving up drinking, but it has been shown to be present even when these factors have been meticulously accounted for. Furthermore, the association is often said to be of no practical importance, but if it is causal people who switched from moderate drinking to teetotalism would increase their personal risk of ischaemic heart disease by 25%.

Many papers have found the association to be present but not to a significant extent and have presented this as evidence refuting the larger and more precise studies in which it has been found to be significant. A finding of a significant difference is not, of course, refuted by finding the same difference to be present but not significant in another study; the two studies are quite consistent, but one is better designed than the other, or larger.

Such misconceptions may be widespread because of the extremely inconvenient nature of the association. If it is causal it will constrain public health measures against alcohol abuse considerably, as the most effective measures would probably be those that reduced total consumption, which would be unethical, and on balance harmful, if they caused large numbers of moderate drinkers to abandon drinking, increasing their risk of ischaemic heart disease. At present, while the association may or may not be causal, the dilemma is even more acute. Do we ignore it and pursue non-selective antialcohol policies, taking the risk that by doing so we are killing people? Or do we limit ourselves to measures selectively aimed at heavy drinkers, knowing that by doing so we are certainly restricting the effectiveness of our attack on alcohol abuse, causing definite ill health on the strength of indefinite evidence?

The paper by Professor A G Shaper and colleagues (21 March, p 733) claims to present findings that conflict with the idea of a protective