of $2.9 \times 10^9/l$ and a haemoglobin concentration of $10.9g/dl$. Her blood film report was: "Leucopenia. Neutropenia with normocytosis. Platelets adequate. Red blood cells showing anisocytosis and normochromia." On repetition six days later her white blood cells had recovered to $5.3 \times 10^9/l$ and the haemoglobin concentration was $11.5 g/dl$; her film report was "Hypochromia of red cells but white cells and platelets normal." A third blood count seven days later showed her white blood cells to be $6.7 \times 10^9/l$ and her blood film was reported as normal.

At the time of this marked leucopenia her fasting blood sugar, urea, and electrolytes were normal; calcium was lowered at $2.08 mmol/l$ ($8.32 mg/100 ml$); her protein and albumin concentrations were marginally low at 63 and $33 g/l$ respectively. Liver function tests were normal and the serum thyroxine concentration was at the lower end of the range at $65 nmol/l$ ($5 \mu g/100 ml$).

Thereafter she was examined by a consultant physician, but was not considered to be clinically hypothyroid or to show evidence of any serious condition. The patient was commenced on ampicillin when all other drugs were stopped, and following the reported leucopenia this was continued for six weeks. She has now made a full clinical recovery from a discrete episode of leucopenia, apparently following administration of mianserin hydrochloride.

ANNE M MCHARG
JAMES F MCHARG

Royal Dundee Liff Hospital,
Dundee

Creatine kinase MB estimation in myocardial infarction

SIR,—The study by Dr S P Joseph and others (10 February, p 372) on technetium imidodiphosphonate scanning in myocardial infarction was of considerable interest. Additional diagnostic help is undoubtedly needed in the many patients in the coronary care unit who have equivocal electrocardiogram and enzyme results. The incidence of such borderline cases has been estimated to be as high as 20% of admissions to coronary care units.¹

The authors, however, give scant reference in their discussion to the value of creatine kinase MB (CKMB) in such situations. Estimating concentrations of CKMB is a technically much simpler diagnostic method than myocardial imaging and also permits the diagnosis to be made earlier. It is also probably more accurate. CKMB disappears from the serum 36-48 hours after onset of chest pain² (not 24 hours as Joseph *et al* state) and so a serum sample must be obtained within this period, but this should not present difficulties. A further advantage of CKMB estimation is that it can be measured retrospectively in selected cases presenting diagnostic problems so long as serum samples have been kept frozen. Such selected use of myocardial imaging would not be so easy four to five days, say, after admission.

Our group (at the Victoria Infirmary, Glasgow)³ measured CKMB in 38 patients with borderline myocardial infarction. The ECG was non-diagnostic, the total CK concentration was raised but no greater than $400 U/l$ ($n=100 U/l$), and the serum aspartate aminotransferase concentration was no greater than $63 U/l$ ($n=42 U/l$). CKMB estimation diagnosed myocardial infarction in 14 cases and excluded it in 24. Retrospective analysis, taking CKMB concentrations as the yardstick,

suggested that the original clinical diagnosis had been wrong in about 12 cases.

Certainly further diagnostic methods would be helpful in the coronary care unit, but in this capacity CKMB would seem to have advantages over myocardial imaging.

DONALD MELVILLE

Department of Cardiology,
Northwick Park Hospital and
Clinical Research Centre,
Harrow, Middx

¹ Krauss, K R, *et al*, *Archives of Internal Medicine*, 1972, **129**, 808.

² Roberts, R, *et al*, *Lancet*, 1977, **2**, 319.

³ Melville, D I, *et al*, submitted for publication.

ABC of Ophthalmology

SIR,—Myopia is the result of inadequate correlation between the converging power of the refractive media (mainly the cornea) and the axial length of the eye. In most myopes the axial length is within the range found in eyes of normal refraction and myopia has occurred because the cornea has failed to flatten sufficiently during growth. Viewed in this light, Mr T Stuart-Black Kelly's claim (20 January, p 198) that myopia is caused by "increased intravitreal pressure" becomes a little implausible. This same fact is the explanation of the alleged influence of hard contact lenses in preventing progress in myopia. Every contact lens practitioner is familiar with the temporary moulding effect of contact lenses on the cornea, but it is equally well known that the effect is transient.

The argument that close work causes myopia wilts before the unassailable proposition that myopia causes close work. A failure to make this distinction in causal relationships has characterised the controversy for well over a century.

When writing for a non-ophthalmological readership, Mr J Stuart-Black Kelly would do well to avoid unqualified statements such as those in the second paragraph of his letter, which, in lumping together those cases of myopia arising from simple dimensional anomaly (the enormous majority) and those having a pathological basis, are likely to cause a great deal of unjustified alarm and perhaps unjustified hardship to young people.

R M YOUNGSON

Department of Ophthalmology,
British Military Hospital,
Hong Kong

SIR,—I am grateful to Mr K D Foggitt (17 February, p 489) for giving a correct description of the mechanism for accommodation. However it is described, the important mental picture to possess is that the elasticity of the lens is the major limiting factor and that it is the loss of this and not any "weakness" or "eyestrain" which causes reading difficulties in middle age.

As for colour values, I believe they are subtly altered when visual acuity is improved with glasses. The duochrome test is an example of this. This has, of course, nothing to do with inherited disorders of colour vision.

I applaud Mr Foggitt's comments on myopia. I agree absolutely with his reasoning and conclusions, but would add two further arguments against the proposition that near vision is implicated as a cause. Firstly, non-reading techniques were in use in special

schools for myopes before the second world war for many years. These were abandoned when it became clear that the myopic process was not affected. Secondly, monocular myopia is acquired and progresses very commonly in children, yet reading is normally a binocular activity. What is the nature of the local resistance in one eye which prevents myopia and of its absence in the other eye? Do we fit bifocal lenses to one eye and plain lenses to the other? Perhaps all children of myopic parents should be given bifocals on school entry.

Until large-scale statistically sound evidence is available—and this would be a very complicated exercise indeed, running perhaps for 20 years or more—nearly all is theory. Such hard facts as are available do not indicate any other way forward. It is my belief that children should be spared the role of guinea-pig even though myopia can have disastrous consequences, unless the strongest evidence supports an experiment and that their parents should not be burdened with the fear of irresponsibility if in most cases they ignore the undeniable attractions of the will-o'-the-wisp.

P A GARDINER

Guy's Hospital,
London SE1

College of Anaesthetists?

SIR,—The Association of Anaesthetists of Great Britain and Ireland is canvassing support for the Anaesthetists' Academic Foundation with a view to establishing a college of anaesthetists independent of the Royal College of Surgeons.

We are concerned that the council of the association assumes widespread support for an independent college of anaesthetists although no polling has taken place for a number of years. At least in this part of Essex the concept is anathema and we wonder how much active support there is nationally and whether the general body of the specialty is apathetic. The various documents issued by the association office play to a considerable extent on the emotional argument and at times remind one of the party political broadcast. We are particularly concerned about the reporter in *Hospital Doctor* (24 January, 1979) who quotes extensively from an interview with the president of the association, and we must register bewilderment at some of the sentiments stated.

In our view there is no advantage to be gained by change of the faculty status within the College of Surgeons. The faculty acts as an independent body in all important respects. It influences standards by its hospital visiting programme through the Hospital Recognition Committee. It organises educational events and it determines the regulation of the FFARCS examination and itself grants diplomas under the new charter. It has been suggested that examination fees and fellows' subscriptions are paid straight into college funds and that anaesthetists derive little benefit therefrom. Nothing could be further from the truth. The examination is set to a high standard and is expensive to run. The examination hall has considerable overheads and the ratio of candidate to examiner is high (36 candidates and 18 examiners currently each day so that each candidate is independently assessed by five pairs of examiners). Fellows of the Faculty of Anaesthetists enjoy the same privileges within the college as any surgical or dental fellow. The building is prestigious, is in a convenient site for the main railway termini, and houses artistic treasures. There is a fellows' common room and a cafeteria where lunch may be