meeting of the European Radiological Congress at Edinburgh. Should these figures confirm the trend, as expected, then an impressive case can readily be made out for the cost-effectiveness of the machine in this and probably in other similar departments.

The fact that clinicians have been quick to recognize the tremendous advantages of the new technique is probably reflected in the doubling of the numbers of patients attending the neuroradiological department. For not only is the initial diagnostic information so valuable, but there is now an easy and harmless technique of checking patients' progress, whether for diagnostic or postoperative purposes. This again is leading to better patient care.

Our experience at this hospital would therefore tend to show that newly developed techniques do not necessarily proliferate without considerable benefit elsewhere. The E.M.I. scanner has been hailed as a tremendous advance, and in our view it has more than fulfilled expectations. It has simplified our diagnostic approach, made considerable inroads into the numbers of traditional neuroradiological investigations, improved the standard of patient care, and saved a significant number of inpatient beds. Administrators can take heart that this is not just another expensive way of increasing hospital costs and investigatory complexity but a new and exciting way of saving lives and money.—I am, etc.,

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Cataracts with a Glucose-6-phosphate Dehydrogenase Variant

SIR,—Nearly a decade ago one of us was involved in reporting in these columns the demonstration of glucose-6-phosphate dehydrogenase (G6PD) deficiency in the erythrocytes of an Australian boy of Maltese descent who was found at the age of 4 years to have bilateral lamellar cataracts.¹ This boy differed from most G6PD-deficient males of Mediterranean extraction tested here in that his erythrocytic G6PD activity was approximately 20% of normal, whereas most other deficient males had very much lower enzyme activity; at no stage before or since has he experienced any haemolytic or other manifestation of this deficiency.

Soon afterwards two further cases were reported of cataract formation in G6PD-deficient boys: Helge and Borner² noted bilateral cataracts in a 4-year-old German boy with non-spherocytic haemolytic anaemia and virtually no G6PD activity; and Westring and Pisciotta³ observed cataracts in late infancy, severe chronic haemolysis aggravated by salicylates, and an ill-defined neurological disease in a Puerto Rican Caucasian male whose erythrocytic G6PD activity was markedly impaired and electrophoretic mobility substantially reduced.

We have now demonstrated that our own patient with cataracts and his 80-year-old Maltese maternal grandfather have a slow-moving electrophoretic G6PD variant (see fig.). We intend to characterize this enzymatic variant further, but report the observation briefly at this stage so as to reopen the question of a possible predisposition to

Starch gel electrophoretic variant of G6PD; (a) normal B⁺ (b) propositus (c) maternal grandfather of propositus.

cataract formation in certain G6PD-deficient subjects, perhaps particularly those with the rare, slow-moving variants.—We are, etc.,

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² Helge, H., and Borner, K., *Deutsche medizinische Wochenschrift*, 1966, 91, 1584.

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Myocardial Injury and Death in Acute Infarction

SIR,—Success in the management of acute cardiac arrhythmias has resulted in the emergence of heart failure and cardiogenic shock as the two main causes of mortality in acute myocardial infarction. Both complications, as pointed out in your leading article (15 February, p. 353), could be correlated directly with the amount of resultant necrotic myocardial tissue. Whatever the antecedent pathology, these two and many other complications, immediate and late, could in fact be attributed to the ensuing injury and necrosis of the myocardial parenchyma. Therefore the pursuit of any means to reduce the mass of necrotic tissue^{1,2} seems a logical development and merits commendation. The objective therein may be considered radical in comparison with those tried so far. All patients having a myocardium destined to infarct or actively infarcting could benefit by reduction of the mass of necrotic tissue. We have data pointing to the existence of another category of patients in whom death of myocardium could be prevented altogether. That would, of course, be achieved when we succeed in combating myocardial injury and death. Meanwhile, at this stage, we wish to draw attention to the existence of such a category by presenting our evidence.

In the course of a study using many clinical, haematological, and biochemical correlative data to determine the degree of confidence with which a diagnosis of acute myocardial infarction could be made on electrocardiographic (E.C.G.) criteria only, we^{3,4} came across a group of patients who seemed to have infarcted while receiving treatment for it. Out of 198 patients admitted to hospital and discharged with a final diagnosis

of transmural or Q-wave-bearing acute myocardial infarction we found that about one-quarter developed E.C.G. changes (ST-segment elevations with or without abnormal Q waves and T-wave inversions in a specified number of leads) diagnostic of acute infarction 24 hours after the onset of the acute episode. However, according to known E.C.G. and morphological correlates of acute injury and death of myocardium^{5,6} based on many experimental and necropsy studies, the permissible latent period for diagnostic E.C.G. changes to appear following ischaemia lies between two and six hours. On this basis we estimate that a proportion actually greater than 25% who were discharged with a final diagnosis of full-thickness acute myocardial infarction were not destined to have an infarct or rather could have been prevented from having an infarct from that episode, provided also that we knew how to deal with the condition.

The fact that such infarcts developed in spite of therapy would suggest the continual operation of factors capable of promoting infarction in the myocardium. Further, these factors do not seem to be abolished by current modes of treatment. The finding that 12 (7.5%) males and 8 (19.8%) females who later developed full-thickness infarcts had only ischaemic changes on the first day, indicating continuous progression of these infarcts, goes further to support this view.⁵ A few other less commonly occurring events^{4,8,9} in association with acute myocardial infarction also lend support to this view. They are (1) the extension while under therapy of an infarct beyond its original boundary, (2) the secondary occurrence of an acute myocardial infarct in patients who are being actively managed for acute coronary insufficiency, and (3) possibly reinfarction in a fresh site of those who have had a recent infarct. In point of fact the category we are trying to delineate, but classified now by others as cases of acute myocardial infarction, seems to fall in between those who would infarct without question and category (2) referred to above.

Thus direct and indirect evidence seems to support the existence of a category of patients with acute myocardial infarction which is preventable and also suggests the existence of factors promoting infarction which we have not been able to control so far. Further study of this group of patients and the introduction of methods of treating them could be expected to yield rewarding results in lowering morbidity and mortality due to acute myocardial infarction.—We are, etc.,

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