we are pleased to note that in another report Moulds and Denborough, using the in-vitro method introduced by us, showed that the same patient's muscle exhibited one of the cardinal features of this myopathy—namely, contracture on exposure to halothane. Central-core disease, so far as is known, does not occur with the contracture seen on anaesthetic agents. Some years ago we examined a mother and a daughter by the lengthy procedure of motor-point muscle biopsy, and both were shown to have central-core disease. Have you used this for general anaesthesia without incident.

In your leading article on the "Pathology of Malignant Hypertrophy" (3 February, p. 249) you showed that you were aware of the non-specificity of the structural changes that have been described so far, but we wish to draw attention to what appears to be a thoughtless remark, in which you say that "it would be interesting to see whether any further susceptibility to the chemicals, or not general, anaesthesia..." show similar histological changes. This could lead to susceptible patients or their relatives being subjected to muscle biopsy, without benefit or risk, as biopsy is indicated, it should include examination at the motor-point (so that nerve terminals as well as muscle cells can be examined), a full histochemical and electron microscopic examination, and above all in-vitro examination of the susceptibility of muscle to anaesthetic agents. Patients are prepared to travel long distances for this examination, and we now have an experience of 20 cases in 10 families. We no longer use local anaesthesia, which is uncomfortable for the patient, impossible in children, and usually interferes with the identification of the motor-point. We employ general anaesthesia and electron microscopic examination, introduced by us, has proved consistently reliable in experienced hands.

Biopsy examination of patients for susceptibility to malignant hypertrophy should not be undertaken lightly, but if it is to be done at all it should be comprehensive.

We are, etc.,

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Cost of Drugs

SIR,—Those whose duty it is to teach medical students have been urged to include some account of the cost of the drugs in their courses of instruction. This is not so simple a matter as may at first appear when one remembers that the "cost" of an illness includes the social benefits as well as the N.H.S. costs and that the "cheapest" drug must therefore be the most effective one. The D.H.S.S. and its precursor the Ministry have for many years issued information sheets with tables or bar diagrams comparing brief lists of drugs of comparable action and dosage, for the N.H.S. of an average prescription or of an equal number of units, capsules, tablets, etc. This method of comparison ignores effectiveness and may therefore be misleading if one accepts the implication that the drug mentioned first in the list (the cheapest in terms of cost to the N.H.S.) is the drug of choice. There is a disclaimer (in small print) that it is not suggested that the drugs mentioned thus far be used in this way, but that the choices of a general practitioner, or one then has been the purpose of this long continued exercise?

A recent example, ECL 106/69 No. 14/72, which is devoted to the cost of antibacterial drugs, illustrates the point. The last page of the issue is devoted to drugs used in urinary tract infections. The cost of 25 tablets each of six frequently prescribed drugs is given:

- Ampicillin 91p.
- Nalidixic acid (Norflox) 2.51p.
- Neomycin (Bacitracin or Septrin) roughly equal at £1.71p.
- Nitrofurantoin (Furadantin) last at £1.38p.

Now before I incorporate this information into the text, I referred to the N.H.S. costs, i.e., the use by the cost to the N.H.S. of the procedure of drug therapy. As in most cases, the cost of the drug is a minor part of the total cost. The cost of the drug is needed to help the practitioner, but it is not all the cost of the drug.

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Sarcoid Heart Disease

SIR,—Your leading article (16 December, p. 627) and the work of Ghosh et al have rightly emphasized that sarcoid heart disease is a serious condition. As a cardiac pathologist I agree that in appearance the aggregates of sarcoid follicles may easily be confused with myocardial fibrosis secondary to ischaemic heart disease. I also agree that the presence of lesions in the right atrial wall, where ischaemic scarring rarely occurs, is of help in differentiating the two conditions at necropsy. However, in my opinion, certain histological features are also helpful in diagnosing sarcoid heart disease, as in the following case.

A 67-year-old woman suffered for three years from complete heart block with recurrent Adams-Stokes seizures and episodes of cardiac arrest and ventricular fibrillation. She deteriorated progressively and died, notwithstanding endocardial pacemaker by catherer. At necropsy fairly large, whitish, hard areas were seen in the myocardium of the ventricular septum and left ventricular walls. There were similar nodules in the lungs, spleen, and some mediastinal lymph nodes, and also throughout the microscopic picture was that of sarcoidosis. Involvement of the right atrium also became evident on histological examination of the conducting system. The node of Tawara, whose out-line was still recognizable, had been almost completely destroyed by sarcoid granulomatous tissue (figs. 1, 2) and other parts of
the system in the ventricular septum were also severely affected, further establishing the aetiology of the heart block.

These findings are characteristic of the lesions and the functional impairment that the inorganic particles of the skin, have been identified as sarcoïd granulomas.

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Fig 2—The code of Tawara, recognizable above the abaxial surface of the skin, is not replaced by sarcoïd granulomatous tissue. (Azan x 12.)

seems to be peculiar to sarcoïd heart disease, as stated in your leading article.—I am, etc.,

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Cutaneous Sarcoidosis in Venepuncture Sites

SIR,—Dr. B. W. Hancock (23 December, p. 706) reports six cases of sarcoidosis which first showed itself by the development of granulomas at the site of previous venepunctures for blood donation. This phenomenon must be an extremely rare one. In a personal series of 260 cases of sarcoidosis, seen over a period of 25 years, there have been 20 examples of granulomatous involvement of cutaneous scars, only one of which related to a venepuncture site.

A woman aged 59 presented in 1969 with typical sarcoid nodules on the skin associated with bilateral hilar lymphadenopathy, and the Kveim test gave a positive reaction. She had been a blood donor. Ten years previously she had been treated for anaemia with a series of iron injections, some of which had been administered intramuscularly into the buttocks and some intravenously into the left antecubital veins. During the five months preceding the appearance of the erythema nodosum she developed two painful prominent red nodules in the scars overlying the site of the previous intravenous injections, and two weeks before the erythema nodosum appeared she also developed deep tender nodules in the gluteal muscles. She was not given corticosteroid therapy. The erythema subsided in six weeks and the bilateral hilar adenopathy after six months, and during this time the left antecubital skin nodules also subsided. Biopsy was not carried out on the cutaneous nodules, but there could be little doubt that this was an example of sarcoïd granulomatous involvement of scar tissue both in the gluteal muscles and in the skin over the left antecubital veins.

The granulomatous involvement of scars in the course of sarcoidosis appears to occur chiefly where the scars are relatively old and firm (and possibly containing particles of silica) and not where there has been merely some recent minimal skin trauma. Patients with sarcoidosis, during their investigation, are invariably subjected to diagnostic tests including venepuncture, yet sarcoïd granulomas do not develop at the sites of these venepunctures. The explanation for the phenomenon among blood donors may well be that in this procedure a local anesthetic is usually used, and in the skin over the vein, a large-bore needle is used and remains in the vein for a longer period, and the same site is often used for successive blood donations. Hence the greater trauma to the veins and extravasation of blood and its subsequent organization leads to more scarring than occurs after the relatively trivial trauma involved in simple diagnostic venepuncture. Moreover, the blood donor needle would in the past have been used repeatedly, and been sharpened on a grinding stone which might have left the slightest trace of silica or some other element on it. The scarring resulting from some of the iron preparation finding its way outside the vein would also explain the phenomenon in the case described above.

The speculation by Dr. G. MacGregor in his letter (10 February, p. 357) as to whether “the intracutaneous scar [of the patient’s blood] [might be] as effective in diagnosing early sarcoidosis as an injection of Kveim homogenate” has already been answered by the work of Refvem and also by Hurley and Shellby, who performed intracutaneous tests with numerous substances (including beryllium, silicon, etc.) as well as with homologous blood and found no evidence of a granulomatous reaction in a large series of sarcoidosis- and normal controls, thus disposing of the theory of the terrain sarcoidique or sarcoïd diathesis as an explanation of the aetiology of sarcoidosis.—I am, etc.,

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Gastric Decompression after Abdominal Surgery

SIR,—Your leading article on gastric decompression (27 January, p. 189) deals admirably with a much-discussed problem. Probably there will never be complete agreement on the precise indications for decompression, but I believe that when decompression is necessary the case for gastrostomy rather than nasogastric aspiration is overwhelming. I have used gastrostomy routinely in preference to a Ryle’s tube since 1964 in every patient (a total of 200-300) in whom at operation I have expected would need postoperative decompression.

I have never seen an infected gastrostomy wound. I use a whistle-type catheter brought through a separate stab wound. The main incision is dressed separately and is devoid of drains, stoma, or any other extraneous foreign bodies. There is no obvious reason why a clean gastrostomy should cause this to be infected, nor do I find an unduly high incidence of this complication. I have no detailed records on the incidence of postoperative chest infections, but anaesthetists have welcomed the absence of a Ryle’s tube in patients needing postoperative ventilation, and physiotherapists and nurses find it easier to treat patients who have a gastrostomy.

Patients who have had gastrectomy and nasogastric decompression invariably say that they would never again have a Ryle’s tube postoperatively. It is customary to see a patient drinking tea the day after