The fundamental problem lies in the apparatus itself; equipment currently available for the use of the handicapped is poorly designed and often unsuited to the child's needs. (In the Wolfson Centre series, 19 out of the 31 appropriately chosen chairs required structural modification.) Much of the equipment is traditional in concept and style, taking no account of modern technology, anthropometry, or ergonomics, being designed mainly by engineers with no clinical contact with the patients involved. The designer of apparatus for this specialized group of patients must apply much stricter criteria to his data collection and design method.¹ In design terms, being handicapped means that the patient has a reduced flexibility and cannot adjust to poor ergonomics as can a normal child. A design must be based on the problem reduced to its simplest form. The concept of a "chair" is irrelevant; it is the postural requirements of the patient that the designer must attempt to satisfy.

The second major fault of currently available apparatus is its complexity. Most chairs are multivariable and can be adjusted to anatomically and ergonomically nonsensical positions. True flexibility can be achieved only from the concept of a dynamic environment in which the child can develop. Simple adjustments which always maintain correct posture reduce the inappropriate use of chairs. Fewer structural modifications are required, which eliminates some of the delays in delivery. In the use of the Cell Barnes chair,² which is purpose-designed for the severely handicapped, no structural modifications have been required in over four years' use in a clinic seeing about 100 new patients per year. Most children can be appropriately equipped with the standard chair; necessary modifications are minor and can be achieved immediately in the clinic with use of direct moulding techniques with plasazote or similar materials to provide individual, specifically corrective forms. Adjustments can be made at each visit with the improvement in the child's posture and control.³

With the present Department of Health tendering system, manufacturers are reluctant to spend money on the development of ergonomically designed equipment which departs from conventional design for the handicapped as they may not receive a contract and would be unable to recoup development costs. Until attention is drawn to the positive harm done and the waste of resources resulting from the unsuitable and misapplied use of apparatus, and until adequate incentives are offered to designers and manufacturers, this regrettable state of affairs is likely to continue.—I am, etc.,

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- ¹ Taylor, R. H., *Community Health*, 1972, 3, 162.
- ² Green, E. A., and Taylor, R. H., *Physiotherapy*, 1969, 55, 376.
- ³ Green, E. A., and Taylor, R. H., *Physiotherapy*, 1971, 57, 68.

Tragic Dilemma

SIR,—I am a doctor and have been the parent of a child handicapped similarly to the one with which your leading article "Tragic Dilemma" (9 December, p. 567) is concerned.

When I first noticed—and it took several

months for the condition to become apparent—that my child was suffering from something so serious, the world went dark around me. And after the tests proved that an operation might offer some hope, I experienced almost a triumph: something could be tried for him. Those were the days of the first neurosurgical efforts on such conditions and, inevitably, each patient had to serve as a guinea-pig as well.

I remember the five times I held my son's hand as he was directed towards the operating theatre and left him on the trolley to pass through the swing doors. I remember him returning to the ward, going through the suffering of the first postoperative days, undergoing investigation after investigation, receiving one injection after the other. I have always been fully aware of the "physical" tragedy his life was until his death a few years later. If left alone, he would have certainly died much sooner. By going on with whatever medical treatment was available then we were only prolonging his day-to-day suffering. But this suffering was definitely reduced and there was the belief that perhaps, were he to survive, the quality of his life was being improved. I have never regretted what I did then.

Some years later I worked in a hospital where a little girl with my child's illness was treated successfully. Recent advances and knowledge based on experience had made her capable of running around, chattering happily, developing like any other normal child, enjoying life—herself and her parents. Then, even more than ever before, I felt as a parent and a doctor that my child's life and death were, after all, justified.—I am etc.,

MEDICAL PARENT

Anaesthesia in Sickle-cell States

SIR,—We have read the paper by Professor K. A. Oduro and Dr. J. F. Searle (9 December, p. 596) entitled "Anaesthesia in Sickle-cell States: A Plea for Simplicity" with interest. The insertion of the ambiguous word "simplicity" into the title makes it possible that anaesthetists might interpret the article as a reassurance that the anaesthetic management of sickle-cell states requires no particular skill. This would be regrettable because, on reading the text, one becomes aware that the authors are in fact advocating something very different. They recommend a painstaking preoperative preparation, a most carefully administered anaesthetic, and efficient postoperative care which includes postoperative oxygen for 12–24 hours.

Despite careful anaesthetic management, there were six postoperative deaths in the 505 patients in the series. Although sickling was thought to have played some part in the death of only two of these patients, one cannot categorically exclude it as a contributory factor in the other four. It was difficult to relate anaesthetic death and sickle-cell haemoglobin from the figures given in the article because there were no data on a comparable series of anaesthesia in non-sickling patients.

With their great experience in this field Professor Oduro and Dr. Searle believe that "a simple anaesthetic technique together with good postoperative care can provide safe general anaesthesia for patients with sickle-cell states" (our italics). In a recent review¹ we concluded that "even if every

precaution is taken, anaesthesia for a patient with sickle-cell disease may prove a hazardous and at times a fatal undertaking." This apparent difference in opinion is at least in part explainable because the vast majority of patients with sickle-cell haemoglobin (that is, with a sickle-cell state) are sickle-cell trait carriers. It is generally agreed that the anaesthetic risk for the sickle-cell trait carrier during routine surgical procedures must be extremely small. In contrast the anaesthetic risk for the small minority of patients with sickle-cell haemoglobin who have sickle-cell disease (that is, sickle-cell anaemia, sickle-cell haemoglobin C disease, and sickle-cell thalassaemia) will be very much higher. For example, there were only 42 patients in Professor Oduro and Dr. Searle's series known to be in this latter category and one of them, a 22-year-old woman (having a McMurray osteotomy) died six hours post-operatively with a "sickle-cell crisis."

In order to minimize the anaesthetic hazards, even in the emergency situation, the anaesthetist in Britain should attempt to divide all patients who have been shown to possess sickle-cell haemoglobin into the low-anaesthetic-risk sickle-cell trait carrier and the high-anaesthetic risk patient with sickle-cell disease. It is fortunate that this differentiation can usually be achieved by simple laboratory tests such as a solubility test to recognize sickle-cell haemoglobin combined with a reticulocyte count. The exact diagnosis of a patient provisionally labelled as having "sickle-cell disease" can be arrived at the next day using more refined techniques such as electrophoresis. The information that the patient has sickle-cell disease should result in a reappraisal of both the diagnosis and the need for operative interference. Even if a careful general anaesthetic is subsequently administered to a patient with sickle-cell disease, it would be unwise for the anaesthetist to imagine that this will always prove to be a safe procedure.—We are, etc.,

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- ¹ Howells, T. H., Huntsman, R. G., Boys, J. E., and Mahmood, A., *British Journal of Anaesthesia*, 1972, 44, 975.

Sarcoid Heart Disease

SIR,—Your recent leading article (16 December, p. 627) draws attention to our report¹ of six new cases of fatal myocardial sarcoidosis. Since that report was completed, from further inquiries I have collected a total of 44 additional new cases in the United Kingdom. My inquiry was prompted by the fact that I had seen, during life, three of the six patients in our original report and, while I was aware of the existence of sarcoid involvement of the myocardium, I had failed to make the aetiological diagnosis. This suggested to me that the condition was more common than is recognized, and the reaction of many colleagues confirms this impression. They have neither made the diagnosis nor thought of its possibility.

Of the total of 50 patients, 17 are from the East Anglian area and these have all been seen by me personally. Most of the other cases come from a relatively small number of centres and this suggests that the diagnosis is overlooked elsewhere. While all

the cases fulfilled the criteria of sarcoidosis as defined by Scadding,³ the majority of patients did not present in the usual way with sarcoidosis, and indeed a survey of chest clinic and eye and skin department material has been unprofitable in revealing cases with myocardial involvement. These chiefly present with the cardiac problems of heart block, difficult tachyarrhythmias, frequent ventricular extrasystoles, sudden death, and congestive cardiac failures, in that order of frequency.

Twenty patients in the series have died and the diagnosis has been confirmed post-mortem. In all, other organs were involved, often to a clinically undetectable degree. In many cases sarcoid involvement of the heart was massive, and in these the classical sarcoid granulomatous tissue with large numbers of giant cells was widespread. In less massive involvement the ventricular septum appears to be the site most commonly affected.

I am preparing the full results of this study for detailed publication. A preliminary report of over 40 cases was made to the Sixth European Congress of Cardiology in Madrid.³ It was there suggested that the diagnosis of sarcoidosis should be considered in any patient presenting with a cardiomyopathy of unknown aetiology, particularly if there are frequent ventricular premature systoles, or in any patient who presents with a serious rhythm disturbance of any type. The success of steroid treatment will depend on how massive is the sarcoid involvement of the heart.

This study has been made possible by the generous co-operation of colleagues allowing me access to their material. I would be glad to have details of any further cases.—I am, etc.,

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- 1 Ghosh, P., Fleming, H. A., Gresham, G. A., and Stovin, P. G. I., *British Heart Journal*, 1972, 34, 769.
- 2 Scadding, J. G., *Sarcoidosis*. London, Eyre and Spottiswoode, 1967.
- 3 Fleming, H. A., *Revista Española de Cardiología*, Proceedings of the 6th European Congress of Cardiology, Madrid. In press.

Spin Dryer Injuries

SIR,—Mr. C. S. B. Galasko (16 December, p. 646) rightly draws attention to the dangers associated with the use of spin dryers. I realize that the pictures of trauma vary from one part of the country to another. In my experience in the north-east of England, I have seen these injuries about 12 times a year in casualty departments receiving 40,000-50,000 new patients a year. These injuries are not confined to children and are replacing the "wringer injury." As with wringer injuries, soft-tissue damage is out of proportion to the bony damage; this is due to the tremendous rotational forces involved. Unlike wringer injuries, these are not virtually confined to the appliances made by one manufacturer.

Spin dryer injuries are related to the kinetic energy involved. It therefore follows that the large drums which mostly rotate on a horizontal axis tend to produce much more severe injuries than small drums, which generally rotate on a vertical axis, where the speed of revolution is similar. Horizontally rotating drums are more accessible to children.

The most common injuries affect the fingers—generally there is dislocation at the proximal interphalangeal joint. This may be inadequately treated by the casualty officer, for the fact that one or both collateral ligaments are torn may be overlooked. However, about one-third of these injuries are compound and it is, I hope, unlikely that an inexperienced casualty officer would be left to deal with these. On occasions, multiple digits may be dislocated.

Injuries at higher levels than the fingers are generally not associated with injuries to the hand, just as injuries to the humerus are unlikely to be associated with injuries below this level. I have seen seven spin dryer injuries to forearm bones, five of which occurred in children. It is again important to realize that damage to soft tissues associated with these injuries is often much more extensive than an x-ray of the forearm would lead one to suppose. Ischaemic contracture and muscle fibrosis must be regarded as definite risks, and forearm decompression may have to be considered.

I have seen three fractures of the humerus from this cause. Once again, soft-tissue damage predominates, and it is not easy to identify the anatomy at the site of injury. I have been more fortunate than Mr. Galasko in that in my worst experience of this type of injury at this level all structures were divided apart from the neurovascular bundle containing the median and ulnar nerves. Progress in this child has therefore been quite good.

I must point out that all the spin dryers that I have seen do in fact have some device either to prevent the dryer being opened while in motion or to arrest the dryer when the lid is opened, though it may be possible to bypass the latter type of device. Injuries occur when the safety devices fail, failure of the former type resulting in more severe injury than failure of the latter. It is not easy to suggest what further steps manufacturers could take. I would refer those interested to British Standard 3456 B6, which specifies that with the latter type of device the drum should stop spinning within 10 seconds of the lid being opened. This standard comes into force in 1974. Which? in March 1971 reported on the mechanical safety of these devices and found that with most makes the drum in fact stopped within five seconds of the lid being lifted.—I am, etc.,

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Side Effects of Benorylate

SIR,—Benorylate is the esterification product of paracetamol and acetylsalicylic acid. It has been shown to have fewer gastrointestinal side effects, including blood loss, than aspirin or an equimolecular mixture of aspirin and paracetamol^{1,2} while retaining their analgesic and anti-inflammatory properties.³ Recorded side effects have been nausea, constipation, indigestion, heartburn, drowsiness, tinnitus, dizziness, and skin rashes. Here we report the case of a patient who was unable to tolerate the drug because of diarrhoea.

A 67-year-old woman with a three-year history of rheumatoid arthritis was admitted with severe active arthritis in her shoulders, elbows, wrists, and metacarpophalangeal, proximal interphalangeal, and knee joints. She was

found to be iron deficient; this was attributed to the salicylate therapy which she had been receiving almost continuously for two years.

She was treated with prednisolone at low dosage (7.5 mg daily), indomethacin suppositories, ferrous gluconate (Sidros), digoxin, pyridoxine, and 5 ml of Benoral (containing 1 g benorylate) in suspension was given four times daily. At this dosage the patient noticed lower abdominal discomfort within five minutes of taking the benorylate, and the discomfort passed off after a further five or 10 minutes. After three days, because of joint pain between doses, the dosage of Benoral was increased to 8 ml five times daily. At this dosage the patient noticed lower abdominal colicky pain within 10 minutes of taking the benorylate, followed within five minutes by an urgent call to stool, the motion being very loose and on occasions watery. No blood was observed and faecal occult blood tests were negative. All drugs except the oral steroids and benorylate were stopped, but the symptoms persisted for two more days until the benorylate was discontinued, since when no further diarrhoea occurred.

We feel confident that the diarrhoea was related to the benorylate and, so far as we can determine, is a hitherto unrecorded side effect.

We are grateful to Dr. D. Mattingly for permission to publish this case.

—We are, etc.,

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- 1 Cuddigan, J. H. P., *Abstracts of the VII European Rheumatology Congress*, No. 45, 7 June 1971.
- 2 Lièvre, J. A., *Information Thérapeutique*, 1969, 7, 49.

Treatment of Massive Pulmonary Embolism

SIR,—The case reported by Dr. R. J. C. Hall and others (16 December, p. 647) is further evidence of the effectiveness of streptokinase in the treatment of massive pulmonary embolism.

The conclusion that the drug was successful in lysing clot in the pulmonary arterial tree was based on clinical assessment—the patient's general state, recovery from dyspnoea, lowering of central venous pressure and pulse rate, etc. While this is probably acceptable, the absence of precise objective evidence of thrombolysis, such as could have been supplied by further pulmonary angiography or lung scanning, prevents any accurate assessment of the effective dose of streptokinase and its rate of infusion. The authors used a 41-hour infusion with a total dose which we calculate to have been 4,650,000 units. In our experience of nine cases of massive pulmonary embolism treated successfully with streptokinase—monitored before, during, and after streptokinase treatment by lung scanning—a 12-hour infusion of 1,500,000 units has been adequate to produce lysis of thrombus in the pulmonary artery. The present report states that within eight hours the patient was greatly improved. This suggests that lysis was advanced by that time. In this context it should be remembered that thrombolysis will continue for periods up to days after the cessation of streptokinase therapy. Furthermore, many of the dangers of streptokinase therapy, particularly in relation to fibrinogen rebound, exhaustion of plasminogen, and rethrombosis, are more likely to occur the longer the infusion lasts and, in fact, sometimes do occur even during pro-