Correspondence

Letters to the Editor should not exceed 500 words.

Undescended Testicle

SIR,—Your leading article on "The Undescended Testicle" (15 May, p. 1259) presents a very one-sided picture and points to little more than the views of one man over one period—early childhood. Your author has completely ignored a number of significant studies in the mechanism of testicular descent and maldescent which have appeared in recent years, from a number of workers. The mechanics of the normal process have been described in several papers, and my own findings together with an explanation of the mechanism of ectopia testis were given in an Arris and Gale lecture to the Royal College of Surgeons.¹ These views fit reasonably closely with experimental and clinical studies carried out by Raynaud in France and Wells, Koop, and Minor among others in the U.S.A.

We have shown that several factors are attendant upon testicular descent. Firstly, a mass of mesenchyme, the gubernaculum testis, which is a structure totally different from that usually described in textbooks of surgery and based on Lockwood's putting into the foetus fibrous bands which he found in cases of ectopia testis. This gubernaculum mesenchyme permits the development of the processus vaginalis and cremaster muscle and also, just before testicular descent, expands to an enormous extent to dilate the inguinal canal and scrotum. The actual descent occurs with a relative shortening of the gubernaculum mass and rapid lengthening of the vas deferens and testicular vessels. These changes have been shown to occur as a result of the high gonadotrophic stimulus to the foetus, from mainly placental sources, acting upon the interstitial cells of the testis to produce foetal androsteroids. Failure of testicular descent can thus, very briefly, be attributed to failure of the testis to respond to the gonadotrophic influence or of the other target organs to the testicular androsteroids. After birth the child ceases to have significant gonadotrophic stimulation and thus the androsteroid output of the testis, which is high just before birth, falls away to a very low level. Hence the descent process can only be consolidated over the first few weeks of life, as Scorer has shown in his writings. But thereafter both Scorer and the writer of your leading article beg so many questions that their credence is often in doubt. No consideration is given to the perfectly reasonable and demonstrable phenomenon of the testis with a low target response to gonadotrophins, which consequently secretes a lower than needed level of androsteroids, or to low response secondary target organs, the gubernaculum, vas deferens, testicular vessels, etc., which can also lead to a perfectly salvable cryptorchid state. We often see, for instance at operation, a testis which cannot descend owing to a shortage of vascular or vasal length: but foetal growth of these structures is stimulated by androsteroids.

Such a concept of the disorganizations of testicular descent can be shown as acceptable experimentally in animals and clinically in man, and also fits in with the patterns found in other fields of endocrinology. It is quite correct that bilateral undescended testes may be deficient, but perhaps not totally or irretrievably so, given a proper post-natal stimulus. Why should we wait to treat these children for eight anxious years (for their parents), when the more logical time to use hormonal stimulation is soon after birth while the mechanism is still at the correct phase of development? Treatment at that time fits in with Scorer's clinical observations as well as my own experiments and experience. The unilateral cryptorchid testis may be an extension of the above, but probably only if it is a right one, this being the later one to descend. In this case hormone therapy is worth trying; but I would agree with your reviewer in the case of the left testis and many right ones also.

On other aspects also the views expressed can hardly be substantiated. Evidence is put forward, for instance, that the cryptorchid testis is usually deficient because *after operation* Scorer found such to be the case, forgetting entirely how delicate is the blood supply of the child's testis, a feature well enough known by paediatric surgeons.² The prepubertal testis is very susceptible to vascular damage or spasm, with consequent irreversible changes, so that it can be argued that the operative procedures could have caused the abnormality which Scorer found.

Undoubtedly many of the comments made by your author are correct, but others are wide of the mark. I feel that if such an article were published under a name it would then be accepted as an individual's views, but an unsigned review showing such lack of balance and respect for other opinions in a still controversial field is good for neither your journal nor medicine.—I am, etc.,

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- Backhouse, K. M., *Ann. roy. Coll. Surg. Engl.*, 1964, **35**, 15.
- Fahlstrom, G., Holmberg, L., and Johansson, H., *Acta chir. scand.*, 1963, **126**, 221.

* * * The aim of our leader was to discuss diagnosis, prognosis, and management, and not operative techniques nor the mechanisms responsible for testicular descent and maldescent, and therefore we did not refer to Dr. Backhouse's important contributions. However, it appears that on at least one, and probably the most important, aspect Dr. Backhouse and we agree—namely, that the management of the undescended testicle is a controversial subject and that until we

know more about the natural history of cryptorchidism and about the effects on fertility of different forms of treatment, there cannot, and should not, be agreement on the best management. We also agree that operative technique is always of great importance—Ed., *B.M.J.*

SIR,—Whilst it cannot be disputed that the normal spermatogenesis is the major aim of treatment with bilateral cryptorchidism, it was a surprise to observe that the risk of neoplasia was not considered in discussing the treatment of unilateral cryptorchidism in your leading article (15 May, p. 1259).

In the Liverpool Region nearly 5% of testicular tumours arise in maldescended testes, whilst A. L. Dean¹ has obtained an incidence as high as 13.5%. Furthermore, the prognosis for these patients is considerably worsened than for those with neoplasms arising in a scrotal testis, and many would agree with the contention of Szymanowski in 1868, recently reiterated by Tresidder and Williams,² that the only certain way of curing abdominal disease in an abdominal testis is by undertaking the preventive procedure of abdominal orchidectomy.

It would be beneficial to see this dictum more widely recognized and extended to all truly ectopic (but not merely retracted) testes. Orchidopexy does not protect the testis from subsequent neoplasia: in our last ten cases of ectopic testicular tumours a previous orchidopexy had been performed in two.—I am, etc.,

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- Tresidder, G. C., and Williams, E. J., *Brit. J. Surg.*, 1962, **50**, 329.

Myopathy in Metabolic Bone Disease

SIR,—We read with interest the article by Dr. J. W. Prineas and his colleagues (17 April, p. 1034) on the myopathy associated with metabolic bone disease. In 1964 we gave an account of three patients in whom a proximal muscular weakness developed after Billroth II resections for gastroduodenal ulcer.¹ Recently we have treated an additional patient presenting the same clinical picture.

The muscular pareses occurred 2–15 years after gastric surgery and were localized to girdle and proximal limb muscles. Electromyographic tracings indicated myopathy in these cases, but definite histological abnormalities were lacking. Malabsorption could be demonstrated in all four cases, and the laboratory data as well as the calcium-balance studies suggested a chemical osteomalacia. Three of the patients were given treatment with vitamin D, which resulted in improvement of the pareses.

We feel that the skeletal musculature may be severely afflicted by a metabolic bone

disease. Gastrectomized patients seem to be especially prone to develop such a myopathy in view of the high frequency of calcium disorders after gastric operations. However, a complete recovery may follow upon treatment with vitamin D. The early diagnosis in the cases is therefore of practical importance.—We are, etc.,

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REFERENCE

- ¹ Ekbom, K., Hed, R., Kirstein, L., and Åström, K. E., *Acta med. scand.*, 1964, 176, 493.

Health Centres

SIR,—It was refreshing to read (*Supplement*, 12 June, p. 249) some healthy criticism of the health centre concept—that “sacred cow” of so many who plan for the future of general practice. Dr. G. J. Addis rightly points out that scientific medical practice demands complex equipment for its satisfactory pursuit, and for this nothing smaller than district hospitals can be the focus.

Most authorities will admit that disillusionment among general practitioners can be traced to its origin in their divorce from the hospital service with its clinical incentives and stimulating contacts. When the family doctor returns as an integral part of the staff of a district hospital, having beds of his own under consultant supervision, some consistent future may appear for general practice.

Health centres, on the other hand, differ little in their essence from present-day surgeries. They are still mere coteries of general practitioners equipped indeed with nursing and ancillary staff but a world apart from the scientific atmosphere of a modern hospital. Only when surgeries can be conducted from hospital out-patients departments and the visiting list be arranged as part of that comprehensive service Dr. Addis envisaged (embracing public health, midwifery, care of the chronic sick, geriatric cases, and child-welfare) shall we begin to see a radical new conception of general practice emerge.

If the personal nature of the relationship between the general practitioner and his patient suffers somewhat under this system can we be sure that it is so supremely vital a necessity for the practice of modern medicine? Or is this, too, merely another “sacred cow?”—I am, etc.,

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“Early Curable Stage” of Rheumatoid Arthritis

SIR,—Dr. J. W. Todd (29 May, p. 1436) refers to the presidential address given by Mr. Angus Ogilvy at the Annual General Meeting of the British Rheumatism and Arthritis Association. He reports him correctly as saying that “thousands were suffering needlessly because of a widespread belief, even among some doctors, that little or nothing could be done to mitigate the crippling effects of rheumatism.” Mr.

Ogilvy spoke to his members wisely, knowledgeably, and factually. Surely Dr. Todd cannot deny the truth of this statement reported in *The Times* of 28 April. If he does he can never meet his patients on social occasions or talk to the man or woman who suffers from “rheumatism.”

Every day the secretary of the B.R.A.A. receives letters from our members or from the general public asking for advice, saying that their doctors have told them that they have arthritis, that nothing can be done for them. Often this dismal pronouncement has been made after a few questions, and, to the patient, a seemingly cursory examination. The social aspect of their trouble has not been discussed, and they have not been advised to attend hospital for a specialist opinion. They have accepted their family doctor's judgment, and some of them in consequence have suffered years of pain, increasing disability, have lost their jobs, caused distress to their families, and have become “crippled.”

The family doctor is only partly to blame. The underlying or main cause of his poor “judgment” is the inadequate teaching, and the improper presentation of the rheumatic diseases by the physicians, whose privilege it is to teach him during his undergraduate days at his teaching hospital. This is possibly aggravated by the desire of many of our specialist colleagues to satisfy the “thirst” of their audiences at B.M.A. lectures and other medical meetings with the presentation of the rheumatoid diseases as intriguing and involved problems in genetics and biochemistry, and the showing of rare cases, which, although he may search for a long time, the family doctor is unlikely to see again. People who suffer from arthritis are seldom, if ever, asked to tell their doctors something of “their side of the story.” A willingness on the part of the doctor to spend a few minutes to listen to his patient's story, followed by a clinical examination, a sincere attempt to explain the reason for his symptoms, reassurance, an appreciation of the “social” factors involved, and when uncertain a referral to hospital for “expert advice” would, I am certain, reduce Mr. Ogilvy's thousands by at least one nought.

“If rheumatoid arthritis is not diagnosed early many more do become crippled.” This implication is correct. I do not know what would be the “natural history” of the disease in each patient that I saw and discussed with my registrar at this morning's out-patients. We do teach, and I think rightly, that two in ten get well without any disability even if we do nothing, or in spite of our intervention; one in ten or probably less will become “crippled” even if the very best treatment is instituted; of the remaining seven the “activity” of their disease, their ability to cope with it, and the sort of life that they will be able to live, does depend on our recognition of the disease, preferably in its early phase, and on our giving proper advice which is accepted and carried out, sometimes over many years, by the patient and his doctor.

Dr. Todd's middle-aged lady may well have had a “lesion” localized to the flexor tendon which would have disappeared after a local injection of cortisone, but this would be no excuse for omitting a clinical examination and a “follow-up.” Naturally physiotherapy and surgery have only a limited but

well-defined role in the management of these patients. This is becoming clear to those of us physicians who in hospital have instituted joint clinics with our orthopaedic, neurological, and plastic surgery colleagues.

“Should she be referred to a psychoanalyst on the assumption that the condition is psychosomatic? This suggestion needs no comment.” But it does; very seldom do I ask the help of a psychoanalyst, but some two years ago I spent a very instructive evening at the Royal Society of Medicine as the guest of the “psycho-somatologists” when they discussed “rheumatoid arthritis.” The “follow-up” has already shown its value. The members of our “rheumatoid arthritis” club at Mount Vernon Hospital speak frankly of their “inner aggression” and frustration. Pain and joint swelling have subsided following the “release” of tension by discussion, and sometimes by the breaking of crockery. We are studying these matters in the activities-of-daily-living unit in our occupational-therapy department, but as yet we have not instituted a “double-blind control,” and asked for a research grant from our regional board to foot the bill.

As my friend Sir Austin Bradford Hill has often said: “If I have given you the wrong answers it is probably because you have asked me the wrong questions.” Dr. Todd's questions and the reason why he asks them demand thought and action.—I am, etc.,

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Hyperpyrexia with Antidepressant Drugs

SIR,—In accord with recent correspondence¹⁻³ suggesting that antidepressant therapy may carry the risk of serious hyperpyrexia reaction, I would like to record the following experience:

A 41-year-old nurse was admitted to the hospital in April 1965 in *extremis*. Relatives stated that she had been in good health several hours previously. The patient was known to have been mildly depressed as a result of her son's disciplinary problems; however, she gave no impression of being despondent. In the past she had dieted for mild obesity and had been known to supplement her diet with amphetamines.

On the night prior to admission the patient had gone on duty and carried out her usual nursing chores in a normal fashion. At 3 a.m. a co-worker had noted that the patient looked “fidgety,” and by 6 a.m. the patient was seen to be sweating profusely. She called her husband about one hour later and asked him to meet her as she felt ill. At 7.30 a.m. the patient was still able to converse freely; however, her fidgeting became more pronounced and she was drenched with perspiration. By 9 a.m. she was incoherent, wildly making involuntary, dystonic movements of all extremities, and assuming the opisthotonos posture.

At the time of her admission at 10 a.m. her pulse was 130/min., blood-pressure 110/70 mm. Hg, and respirations 35/min. and stertorous. She maintained opisthotonos, continued to make writhing movements, grimaced, and was unarousable. The pupils were widely dilated and fixed and her fundic arterioles were “thread-like” in appearance. The optic disks were flat. Bilateral Babinski sign was present. Her temperature was 106° F. (41.1° C.).

At this time the patient's belongings were searched and a small pill box was found to