SIR,—I was interested to read the report of sudden death after exercise reported by Dr. R. C. R. Connor (4 October, p. 30). It is unfortunate that he does not mention whether voluntary muscle tissue was examined histologically, for clinically the history suggests that the cause of death may have been exertional rhabdomyolysis. In this condition the heart muscle is normally spared, though a case reported by Favara *et al.*¹ did show myocardial damage.

Sudden death, which is not uncommon in rhabdomyolysis, is thought to be due to the hyperkalaemia which follows the acute muscle cell damage, and which may result in a fatal cardiac dysrhythmia.—I am, etc.,

D. C. L. SAVAGE.

Department of Child Health,
University of Dundee.

REFERENCE

- ¹ Favara, B. E., Vawter, G. F., Wagner, R., Kevy, S., and Porter, E. G., *American Journal of Medicine*, 1967, 42, 196.

Congenital Anomalies of the Vas Deferens

SIR,—Now that sterilization by vasectomy is a common procedure it would be useful to know what congenital anomalies of the vas may be encountered.

Recently I divided and ligated a perfectly normal right vas deferens and was then surprised and embarrassed (the operation was under local anaesthesia) to be unable to find any trace of a vas on the left side. A piece of tissue was eventually removed for histological examination and was reported to be a small artery (Dr. F. C. Harris). Two subsequent semen microscopies having been reported negative for spermatozoa, the patient was re-examined, and the absence of a left vas deferens confirmed. The left epididymis was present and the body of the left testis felt normal, not atrophic.

I was recently consulted by a second patient whose wife had become pregnant following his vasectomy by "a perfectly competent urologist." The lady's reputation was saved by the finding of active spermatozoa in her husband's semen and he was given the usual explanation of duplication of the vas. When I examined him he appeared to have one vas deferens only on each side, and it seems very probable that this was a case of recanalization rather than duplication.¹

From the evidence of the first case reported above I am satisfied that congenital absence of the vas deferens can occur. I would be very interested to know whether any of your readers have first hand experience of duplication of the vas.

In my experience the most difficult part of the operation is the digital manipulation required to isolate the vas from the other cord structures. Sometimes it lies on the lateral side of the cord and sometimes anteriorly. In the latter case, if it occurred bilaterally, it might be easy to divide the same vas twice, which might be a second explanation of the cases of duplication of which one not infrequently hears.—I am, etc.,

R. E. B. TAGART.

Newmarket General Hospital,
Newmarket,
Suffolk.

REFERENCE

- ¹ Pugh, R. C. B., and Hanley, H. G., *British Journal of Urology*, 1969, 41, 340.

Warning to Travellers to Tanzania

SIR,—On entering Tanzania recently my personal supply of medicines was confiscated by the Customs. The reasons given were, firstly, that the drugs had been "made, packed, or bought" in South Africa; and, secondly, that I did not have the written authority of the Principal Medical Officer of Tanzania to import these drugs. Fortunately none were essential for my immediate well-being, and prompt appeal to responsible officials in Dar-es-Salaam ensured their return after a few days.

This incident occurred at Tunduma, a remote frontier post on the road from Zambia. It would be advisable for patients visiting Tanzania to enter only at Dar-es-Salaam, where in case of difficulty senior officials could be contacted without delay. They should also carry a letter from their doctor stating the drugs they require.—I am, etc.,

GEOFFREY H. ROBB.

Bristol General Hospital,
Bristol.

Exposure and Exhaustion

SIR,—I read with great interest Dr. L. G. C. E. Pugh's article (14 June, p. 657) on the physiological effects of prolonged outdoor exercise, particularly the last paragraph in which he discusses the possible significance of ketosis. The following case of exhaustion and exposure, seen recently under conditions rather different from those of Dr. Pugh's experiment, seems to bear out his point that in certain circumstances the resulting clinical picture may owe as much to ketosis as it does to hypothermia, especially where the causation of mental symptoms is concerned.

A heavy man, aged 57, generally fit, got himself "bushed" on a high escarpment plateau in the Eastern Highlands of Rhodesia, and spent three nights out at a height of between 6,000 and 8,000 ft. (about 2,500 m.). He was wearing a thin shirt and shorts only, and light desert boots, and carried with him no iron-ration of food or extra salt. During the day there was hot sunshine, but at night there was frost and he undoubtedly became severely chilled, although he did his best to keep a fire going and was able to sleep intermittently. Despite being without food, he soon ceased to suffer from hunger, and fortunately he was able to quench his thirst regularly from mountain streams; but in spite of this his fluid intake was almost certainly inadequate and accompanied by salt depletion. Especially during his last day out he was troubled by intermittent hiccups and experienced illusions, in which he mistook tree stumps for people and rocks for houses, and at one time had the peculiar feeling that he was not alone; as he put it, "there must be other people in the party." He eventually found his way down a very rough and broken mountainside covered with dense and prickly vegetation to a road, where he was picked up in a condition of extreme fatigue by some passing tourists.

On examination about an hour and a half later, when he had already had a hot bath and drunk plenty of milk and soup with extra salt in it, he was found to be voluble, elated—as well he might be under the circumstances—and slightly confused mentally. His general physical condition was surprisingly good, although his limbs were covered with scratches and abrasions and his feet were badly blistered. His temperature

was 94.6° F. (35° C.), pulse rate 84 per minute, and B.P. 110/60, but the most striking sign was the extremely strong smell of acetone in his breath. This was still present next morning, although to a lesser degree, when his temperature had risen to 97.4° F. (36° C.) and his B.P. to 130/70. At this time also he was found to be 10 lb. (4.5 kg.) below his usual weight.

Looking back on the episode as a whole, the mere fact that this man took as long as three whole days to make his way down this particular hillside to the road at its foot in itself suggests that he was suffering from some degree of mental confusion as well as from physical fatigue.

To summarize, here was a case of severe exposure and exhaustion with mental symptoms playing a prominent part, in which there was clear evidence of marked ketosis. Hypothermia, if present at all, can only have been intermittent, and is unlikely to have contributed materially to the final clinical picture.—I am, etc.,

GERARD BALLANCE.

Cambridge.

Awareness during Anaesthesia

SIR,—While deploring Dr. H. L. Leaming's division of anaesthetists into academic and practical (4 October, p. 51) we wish to take issue on both theoretical and practical grounds with his simple views on anaesthesia for caesarean section. He advocates the use of nitrous oxide and oxygen supplemented by hyperventilation, and finds no rational reason for giving more than 21% oxygen "except in hyperbaric conditions." This last statement is easily dismissed: it is only in hyperbaric conditions that one can safely breathe reduced concentrations of oxygen.

There is a well-established body of evidence to show that during anaesthesia, and especially during controlled ventilation, oxygenation of arterial blood is impaired. Several workers¹⁻³ have shown that during controlled ventilation with gas mixtures containing less than 30% oxygen, episodes of arterial hypoxaemia are likely to occur in many patients. The severity of such hypoxaemia will be increased by a concomitant fall in cardiac output, as will happen during respiratory alkalosis due to hyperventilation. During caesarean section the dangerous effects of maternal hyperventilation on the fetus have been stressed by Moya and his associates,⁴ who showed that maternal alkalosis can cause severe fetal acidosis and delayed onset of respiration.

Although fetal oxygenation is not a simple function of maternal oxygenation it is obvious that a reduced arterial oxygen content in the mother will tend to be deleterious to the child. There is a well-established relationship between fetal hypoxia and such ominous signs as fetal heart changes⁵ and a low Apgar score.⁶ Dr. Leaming appears to use as his criterion of adequate fetal oxygenation the avoidance of intra-operative fetal death. This is to ignore any effects of fetal hypoxia upon the later cerebral function of the child.

Finally we would deplore his use of the term "supplemented by hyperventilation."