Lactic Acidosis Complicating Late-pregnancy-onset Diabetes

SIR,—I was interested to read the letter of Drs. H. F. Woods and D. H. Williamson (22 February, p. 511). It is a pity they did not apply the same degree of criticism to the work of Daughaday and others[1] which they quote and whose three patients with "non-ketotic acidosis and diabetes" had no lactate estimation performed in one instance, and had other good reasons for the lactic acidosis which occurred in the other two. May I also suggest that an arbitrary figure of 7μM/ml. obtained in the laboratory before the diagnosis of lactic acidosis can be accepted would have helped little in the clinical management of the patient we described (14 December, p. 707) since to wait for such a high level would have resulted in the death of our patient when the lactate level rose to 6·91μM/ml.[2] Indeed, the high "arbitrary" figure of 7μM/ml. required by Tranquada and Grant[3] probably accounts for the 39 deaths in their series of 46 patients who had good reasons for developing lactic acidosis apart from the presence of diabetes, as was accepted by Tranquada and Grant themselves. Accordingly, I would repeat, "If untreated, lactic acidosis may become severe and jeopardize the life of the patient and/or the foetus. The diagnosis of this condition should be considered whenever a diabetic patient presents with a strongly positive Acetest and a negative ferric test."—I am, etc.,

BRIAN LIVESLEY.

King's College Hospital,
London S.E.5.

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2 Egan, J. D., Jordan, M. C., and Sullivan, J. F., *American Journal of Cardiology*, 1968, **22**, 516.
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Tumoral Calcinosis

SIR,—I was most interested in the article by Dr. S. McClatchie and Dr. A. D. Bremner on tumoral calcinosis (18 January, p. 153). In 24 years of practice in southern Africa I have seen and operated on many cases of this condition, which until now I had assumed to be calcification of trochanteric bursae due to sleeping on mats on the hard floor. My cases have been almost exclusively confined to females, and to the trochanteric regions, often bilateral. It was a surprise to me to read that the lesions could be found on other parts of the body. I confirm the finding of white chalky fluid which looks like an aqueous suspension of procaine penicillin, and I confirm also that the lesions are sometimes perforated and chronically infected.

My treatment has always been to excise the lesions down to bone, and this has proved successful in the first instance, but, like Drs. McClatchie and Bremner, I find it impossible to follow them up. The last case, on which I operated two months ago, absconded before her stitches were removed.—I am, etc.,

HAMILTON CURRIE.

Chitambo Hospital,
P.O. Kanona,
Zambia.

SIR,—Tumoral calcinosis (18 January, p. 153) is indeed an intriguing condition. We believe we have established some of the pathogenetic factors of this disease by comparing tumoral calcinosis with "Kikuyu bursa," a lesion in the subcutaneous tissue of the back caused by chronic mechanical trauma due to carrying loads suspended from a head-strap.[1] As a result of this special tribal way of carrying one or more bursae develop on the back which are similar to periarticular bursae. An interesting point is that the histopathological changes in the subcutaneous tissue of an early Kikuyu bursa and of a decubitus ulcer are also similar.[2]

Kikuyu bursa: x-ray photograph of an excised surgical specimen.

A striking feature of Kikuyu bursae is the presence of calcium deposits in quite a number of these bursae. Very often also haemosiderin is detectable. It appears that both the histological and radiological findings in calcified Kikuyu bursae and tumoral calcinosis are identical. It seems therefore likely that both conditions have also some causative factors in common: chronic mechanical trauma, which subsequently leads to bursa formation and calcification.

Finally, the fact that tumoral calcinosis, a solid, partly multiloculated cystic lesion, is very often localized in the periarticular tissue raises the question whether tumoral calcinosis is not a calcified periarticular bursa.—I am, etc.,

J. W. KOTEN.

Department of Anatomy and Pathology,
Institute of Tropical Medicine,
Kinshasa, Congo Republic.

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Acute Abdomen in Children

SIR,—The plea from Dr. R. H. Jackson (1 March, p. 575) for further work on the causes of acute abdominal pain in childhood must be cordially welcomed. My paper (1 February, p. 284) was limited to recording our observations, and at the moment further comment must be speculative. Dr. Jackson is right to hope that further virological research may improve the understanding of these syndromes. This does not alter the current position in clinical diagnosis, which must include the fact that attempts to diagnose acute mesenteric adenitis at the bedside are a mistake. This does not imply that it will never become correct to do so.

The label "mesenteric adenitis" makes the doctor think medically; he may then tolerate and continue to watch signs which can equally indicate a surgical disease. Such distinctions between medical and surgical causes for signs of peritoneal irritation in the right iliac fossa cannot at present be safely made. This brings us back to one of the main points in both Dr. Jackson's letter and my own paper—that it is the careful assessment of the physical signs which counts most in deciding management.

I willingly accept Dr. D. J. M. Bruce's criticism (1 March, p. 575). The present paper was written very much as a sequel to the first one on the same subject,[1] in which considerable attention was paid to the vital importance of rectal examination, to the technique of this examination in children, and to interpretation.—I am, etc.,

PETER F. JONES.

Royal Aberdeen Hospital
for Sick Children,
Aberdeen.

REFERENCE

1 Winsey, H. S., and Jones, P. F., *Brit. med. J.*, 1967, **1**, 653.

Splenic Leishmaniasis

SIR,—We would like to record the following observation.

The serum from five patients with splenic leishmaniasis showed a high titre of antistreptolysin-O units, possibly associated with the hyperglobulinaemia known to occur in this illness. In each case the diagnosis of leishmaniasis had been reached through finding Leishman-Donovan bodies in marrow puncture. Antistreptolysin-O (A.S.O.) units were estimated by the Rantz and Randall method, using Wellcome reagents.[1]

One patient, a man of 54, had 1,250 u./ml. Another, a boy of 11, had over 2,500 u./ml., when first examined and over 10,000 when re-examined 14 days later. A boy of 3 years had 1,250 u./ml.; another boy of 6 had 833 u./ml. The A.S.O. titre was not estimated on a boy of 2½ years when he first came under observation, when the diagnosis of leishmaniasis was made. He was tested four months later when he returned to hospital suffering from a relapse and showed a titre of 1,250 u./ml.; retested eight days later the titre was 833 u./ml., a one-tube difference. There were also two patients in which the A.S.O. titre was not markedly high. One, a young man of 18, had only 250 units; another, a boy of 3, had 125 u./ml. Two patients whose A.S.O. titre was estimated six and five and a half months respectively after they had been treated and cured showed only 125 and 250 u./ml. respectively.

As far as could be ascertained, in none of these cases had there been a streptococcal infection.—We are, etc.,

EMANUEL AGIUS.
ROSEMARY PEPPER.

Bacteriology Department,
St. Luke's Hospital,
Malta, G.C.

REFERENCE

1 Rantz, L. A., and Randall, E., *Proceedings of the Society for Experimental Biology and Medicine*, 1945, **59**, 22.

Measles in the Tropics

SIR,—I have just received, and read with great pleasure and profit, Dr. David Morley's account of severe measles in the tropics. One must agree with him that the ultimate control of this disease is both possible and urgently necessary, but realism suggests that it is likely to be with us for many years yet.

I should therefore like to mention two further points in the management of the disease that we have found to be of value