fourth ventricle, I can still depend on ultrasound to recognise changes in cardiac rate and rhythm.

I would strongly recommend the neurosurgical anæsthetist not to reject ultrasonic monitoring because of fear of interference from the diathermy apparatus. If interference is a problem, the surgeon should be encouraged to use a different diathermy machine.

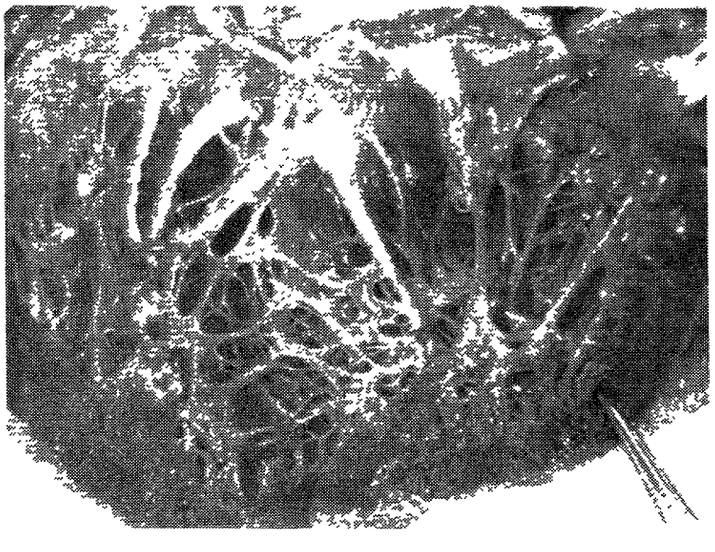
Department of Anæsthetics,
The Royal Infirmary,
Sheffield S6 3DA. W. N. C. McCLEERY.

ENDOCARDIAL CALCIFICATION

SIR,—We describe here the clinical feature, electrocardiogram, and hæmodynamic studies in an unusual case of endocardial and myocardial calcification, which was discovered at necropsy in a 30-year-old man who presented clinically with cardiac failure due to cardiomyopathy involving the left side of the heart.

Clinical examination revealed signs of congestive heart-failure —ventricular hypertrophy and a short systolic murmur at the apex. The electrocardiogram showed atrial fibrillation with large, coarse, slow F waves. There was striking left-axis deviation with a mean frontal QRS vector of −60° and a mean frontal T vector of +100°. There was prolongation of the terminal parts of the QRS complexes with the first 40 milliseconds almost opposite in direction to that of the remaining terminal portion. The precordial QRS voltages were increased, and the tracing indicated left ventricular hypertrophy with in addition anterior hemiblock. The chest X-ray showed a cardiothoracic ratio of 67% with significant left atrial enlargement.

Cardiac catheterisation showed elevation of left atrial and left ventricular end-diastolic pressure (30 mm. Hg). No mitral-valve diastolic gradient was observed. There was moderate passive pulmonary hypertension. The cardiac output was normal.



Appearance of heart at necropsy.

Left atrial cineangiography revealed grossly enlarged left atrium with no evidence of mitral stenosis. The left ventricular cineangiogram showed normal left ventricle, and it contracted well except for a small segment on the anterior aspect. No calcification was seen on screening.

The heart-failure responded satisfactorily to digoxin and diuretics for two years, after which the patient was readmitted in severe congestive heart-failure. During this admission his ventricular-rate slowed down to 30 per minute, which caused syncope. Electrocardiogram showed atrial fibrillation with slow ventricular rate. Transvenous intracardiac pacing was done without improvement in the circulation, and he died.

At necropsy the heart weighed 450 g. The trabeculæ carnæ in the region of the septum and the posterior wall were transformed into calcified columns approximately 4 cm. long and 0·5 cm. wide, with a coarsely nodular external surface (see figure). The calcification was most striking in the endocardium and the superficial few millimetres of the immediately adjacent myocardium, and only microscopic foci were evident in the deeper layers of the heart muscle. The endocardium between the areas of calcification appeared normal.

There was some calcification of the mitral-valve ring. The papillary muscles of the mitral valve were small and fibrotic. There was a small patch of granular superficial calcification on the wall of the left atrium. There was some hypertrophy of the left ventricular wall and on section the myocardium showed patchy fibrosis. These areas were a few millimetres in diameter and were confluent in places.

Dystrophic calcification in the endocardium may be associated with hypokalæmia,[1] infection, infarction, granuloma formation, parasitic infestation, and degeneration of tumours.[2] Endocardial calcification was not suspected during life in this patient, and in retrospect the investigations that were done throw no light on its possible cause, except for the chance finding of echovirus type 21 in the fæces on one occasion. The possibility exists that this lesion followed a viral myocarditis. The distribution of the calcification is very unusual, being mainly endocardial and maximal in the trabeculæ carnæ.

We thank Dr I. Spencer, Preston Hospital, North Shields, for referring the patient and for permission to publish this case; Dr P. Szekely, Dr R. G. Gold, Dr F. Jackson, and Prof. B. Tomlinson for their invaluable contributions; and Mrs M. P. Johnson for her assistance.

Regional Cardiovascular Unit,
Newcastle General Hospital,
Newcastle upon Tyne.
S. J. JACHUCK
H. DAYA
C. KAVAKAS.

AUTOERYTHROCYTE SENSITISATION IN A YOUNG BOY

SIR,—We have observed a 10-year-old boy in whom autoerythrocyte sensitisation, or psychogenic purpura, developed. This syndrome, which was first described in young women by Gardner and Diamond,[3] consists of recurrent episodes of painful ecchymoses commonly associated with multiple somatic complaints and characteristic anxiety and depression. Our patient also had an increased concentration of plasma-cryofibrinogen during acute attacks, which reverted to normal in remission.

The patient had a 5-month history of recurrent lesions which began with sharp localised pain progressing through erythema and heat to purpura in a few hours. They occurred on the front of the shoulders, legs, and arms, usually lasted 4–5 days, and recurred every 6–10 days. During these episodes he also complained of intermittent pain in the abdomen and legs.

Physical examination showed blotchy purpuric areas scattered over both upper arms. These areas were slightly indurated and tender to touch, but did not blanch with pressure. The remainder of the physical examination was normal. The hæmoglobin was 14·5 g. per 100 ml., the white-blood-cell count was 5900, and the platelet-count was 288,000. The erythrocyte-sedimentation rate was 14 mm. in one hour. Complete clotting studies, rheumatoid factors, antinuclear antibodies, lupus-erythematodes-cell preparation, and C-reactive protein were negative. C3 was 190 mg. per 100 ml. Serum concentrations of IgG, IgA, and IgM were 12·25, 1·30, and 1·70 mg. per ml. respectively. Immunoelectrophoresis was normal.

Psychological testing demonstrated superior intelligence (Wechsler). This depressed and anxious child was able to express his hostility only in a very passive/aggressive manner, was unperturbed by his somatic symptoms, and used his illness to gain parental attention. He also displayed infantile behavioural traits.

Skin-tests were done on two occasions with the patient's blood. Each test produced typical lesions (2·5–6 cm.) with 0·1 ml. of whole blood and with red blood-cells lysed by freeze-thawing. No lesions were produced by lysed buffy coat or saline solution. In addition, new lesions occurred while the patient was in hospital and under continuous observation by a physician.

1. Littman, M. S., Meadows, W. R. *Circulation*, 1963, **28**, 938.
2. Hudson, R. E. B. Cardiovascular Pathology. London, 1965.
3. Gardner, E. H., Diamond, L. K. *Blood*, 1955, **10**, 675.

During an acute attack, the patient's plasma contained 0·9 mg. per ml. of heparin-precipitable cryofibrinogen (normal range 0·56±0·1 mg. per ml. in adults [4]). The re-dissolved precipitate formed a precipitin arc with anti-fibrinogen antiserum in agar gel; there was no reaction with antisera to IgG, IgA, or IgM. During remission, the plasma-cryofibrinogen was 0·06 mg. per ml., while the serum-IgG fell to 6·80 mg. per ml.; IgA and IgM were essentially unchanged. There was a rise in cryofibrinogen during two different attacks of purpura.

Cryofibrinogenæmia during acute attacks may be related directly [4] or coincidentally to activation of the kinin-generating system in inflammatory lesions [5] or may be the consequence of fibrinolytic breakdown of the purpuric lesions.[6]

Our patient, a 10-year-old boy, displayed the same typical personality traits described in young women with this type of purpura. How this personality is related to the pathogenesis of the purpura is unknown.

Department of Pediatrics and Communicable Diseases, University of Michigan Medical School, Ann Arbor, Michigan 48104, U.S.A.

ELIZABETH M. KURCZYNSKI
JAMES T. CASSIDY
RUTH M. HEYN.

GASTROINTESTINAL BLEEDING AND ERYTHROCYTE AUTOANTIBODIES

SIR,—The hypothesis of Dr Jerne and his colleagues (Jan. 13, p. 79) that gastrointestinal bleeding might provoke erythrocyte antibodies is interesting, but an alternative explanation should be considered. Substantial gastrointestinal bleeding usually evokes a reticulocyte response from the normal marrow. Transferrin on reticulocytes may cause a positive Coombs' test.[7] Could not the reticulocytosis resulting from bleeding lead to the positive direct antiglobulin tests ?

Radioisotope Laboratory, Cincinnati General Hospital, Cincinnati, Ohio 45229, U.S.A.

EDWARD B. SILBERSTEIN.

WHY ARE SMALL-BOWEL TUMOURS SO RARE ?

SIR,—The article by Dr Lowenfels (Jan. 6, p. 24) interested us greatly, and his observations and conclusions are in keeping with our own.

We agree about the low incidence of cancer of the small intestine, both as a primary and secondary growth. This was an incidental finding during our treatment of rheumatic diseases with an extract of the whole mucosa of the small intestine. One of us (A. R.) has used this treatment for 30 years and the other (J. McC.) for 20 years. The total number of cases treated is 1734 and the albumin/globulin ratio in the serum was estimated in most of them, a fairly constant finding being a diminution of the albumins and increase in the globulins.

Some patients who already had cancer of the colon before treatment with intestinal extract began appeared to live much longer than expected, even when the surgeon had already noted the presence of secondaries. The number of such cases is small, but the effect was striking.

All our cases have been followed closely for up to 20 years and, so far as we know, not one of the 1734 patients developed carcinoma in any part of the body during treatment.

We believe that the enzymes in the small intestine are probably responsible for the low incidence of cancer in that organ and that the administration of intestinal extract seems to some extent to inhibit the development of secondary growths elsewhere, even when a primary growth is already established in the large intestine.

Redcot, 22 Grove Avenue, Wilmslow SK9 5EG. ARNOLD RENSHAW.

Wairoa, Catrine, Mauchline, Ayrshire. JOHN S. MCCRAE.

NUTRITION AND THE DEVELOPING BRAIN

SIR,—Your leading article [1] reviewed the excellent publication on this subject by the Ciba and Nestlé Foundations.[2] A monograph on this subject from the PanAmerican Health Organisation [3] is a very useful counterpart to this, because it deals in rather more detail with the long-term effects of malnutrition on physical and intellectual development in man.

On the biochemical and experimental evidence we must accept that malnutrition imposed at a critical period of brain development is likely to cause permanent impairment of structure, and therefore perhaps of function as well. Dobbing [4] has emphasised that in man this critical period extends from the later part of pregnancy to the age of about 2 years or more, so that it covers most of the age-range in which malnutrition is common. However, I think it is important not to overlook the other side of the picture—that some degree of rehabilitation can occur. The malnourished child seems to have a remarkable capacity for physical catch-up: an example is given by Graham [5] from Peru, where the initial circumstances are particularly unfavourable—very severe malnutrition at an early age.

It is, of course, much more difficult to tell whether mental or intellectual catch-up is ever complete, because one cannot know the original potential. The study in Jamaica by Hertzig et al.[6] showed that children who had been severely malnourished in infancy, some 5 years later were *on average* significantly inferior in their mental development to their sibs or to comparison children in the same schools. However, the late Prof. Herbert Birch, in a Special University Lecture which he gave in this school last year, pointed out that the failure among ex-patients was not inevitable: although on average their performance was worse than that of the other groups, if one looked at individuals there were some who reached the level of the controls. As far as could be determined, success or failure in this bore no relation to the original nutritional state. It might be argued that these were exceptional individuals who, if they had never been malnourished, would have reached a level above the average. We can never know the answer for certain, but it is significant that in the Jamaican study the malnourished children who returned to homes in the towns performed better in all the tests than those who lived in the country. Many of the papers in the P.A.H.O. symposium [3] (e.g., those of Birch, Cravioto, and Monckeberg) draw attention to the importance of maternal and social stimulation in conditioning the child's mental development, even though he has been malnourished.

Therefore I cannot agree entirely with the suggestion in your leading article [1] that it is really an academic

4. Epstein, W. V., Tan, M., Melmon, K. L. *Arthritis Rheum.* 1968, **11**, 478.
5. Ratnoff, O. D. *in* Immunobiology (edited by R. A. Good and D. W. Fisher); p. 135. Stamford, Connecticut, 1971.
6. Agle, D. P., Ratnoff, O. D. *Archs intern. Med.* 1962, **109**, 685.
7. Jandl, J., et al. *J. Lab. clin. Med.* 1960, **55**, 662.

1. *Lancet*, 1972, ii, 1349.
2. Lipids, Malnutrition and the Developing Brain. Amsterdam, 1972.
3. Nutrition, the Nervous System and Behavior. Scientific Publication no. 251. PanAmerican Health Organisation, Washington, 1972.
4. Dobbing, J. *Lancet*, Jan. 6, 1973, p. 48.
5. Graham, G. G., Blanca Adrianzen, T. *Pediat. Res.* 1971, **5**, 691.
6. Hertzig, M. E., Birch, H. G., Richardson, S. A., Tizard, J. *Pediatrics*, 1972, **49**, 814. See *Br. med. J.* Jan. 20, 1973, p. 126.