Ultrasonic appearance probably indicative of imminent fetal death

One of the difficulties in recognising fetal death by ultrasonic scanning in early pregnancy is that the negative finding of no fetal heart movement must always be less convincing than a positive finding. Furthermore, Robinson[1] found only one instance of irregularity of the fetal heart in ten cases scanned before their pregnancies became missed abortions, and he concluded that it was not yet possible to predict fetal death by sonar. I report an ultrasonic appearance that I have noted on at least 14 occasions always in association with subsequent fetal death.

Case reports

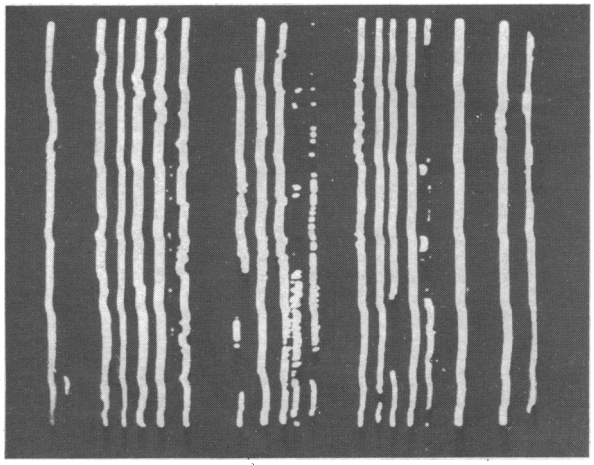
Case 1—A 33-year-old housewife had had one previous pregnancy 2½ years earlier. She was first seen in the current pregnancy 12 weeks after her last menstrual period. Two weeks later she had a slight brownish vaginal discharge, and on clinical examination her uterus was felt to be small for dates. She was therefore referred for ultrasonic scanning. The scan was performed 14 weeks after her last period using a Diasonagraph NE 4102 (Nuclear Enterprises, Edinburgh) with a sound frequency of 2·5 MHz and pulse repetition frequency of 600 Hz. Fetal crown rump length was only 50 mm, and a fine vibration was noted originating from the region of the fetus (figure). The vibration was intermittent and variable in rate but about 1200-1500 Hz. On repeat scan three days later no fetal heart movement or vibration could be detected. An assay of the serum concentration of the β subunit of human chorionic gonadotrophin gave an inappropriately low value and it was decided to evacuate the uterus surgically. This was carried out four days later, the products being histologically compatible with a missed abortion.

Case 2—A 27-year-old housewife was referred for termination of pregnancy. An ultrasonic scan (details as in case 1) carried out seven weeks after her last menstrual period showed an intrauterine gestational sac containing a fetus with a crown rump length of 14·7 mm. The fetal heart movement was easily detected and had a rate of 150 beats/min. Termination of the pregnancy was agreed and, with the patient's consent, it was decided to perform this by the intravaginal administration of a pessary containing an ester of 15-methyl-prostaglandin F2α.[2] As part of the trial of the abortion method further ultrasonic scans were performed seven hours after insertion of the pessary and two days later. On the earlier of these scans no fetal heart movement could be detected within the partially fragmented gestational sac; but on the later scan very slow, irregular movements of fetal heart type were seen in the region of the fetus, and superimposed upon these were intermittent rapid vibrations, the rate of which was about 1500 Hz.

Comment

In both cases described the fetal echoes were too small to allow further identification of the source of these vibrations, but when the phenomenon has been observed in larger fetuses it appears to originate from the thoracic area in the position where the heart movement might be expected. The rate of the vibration is, of course, much faster than the heart rate but it does not appear to be an artefact since it is localised to the region of the fetal echoes and is intermittent, usually lasting for only one or two seconds at a time. Furthermore, I have never observed this appearance in pregnancies which subsequently progressed normally.

The mechanism of production of this phenomenon is unknown but possibly the biochemical changes associated with imminent fetal death give rise to myocardial fibrillation and it is the resulting movements which are detected. I should be most interested to hear of any similar observations and to learn of any alternative hypothesis, either physiological or electronic, to account for this appearance.



Time-position scan (time base movement 25 mm/minute) showing rapid vibrations in central echoes.

I thank the consultant staff of Charing Cross Hospital for referring their patients for ultrasonic scanning, and in particular Mr M G Elder (now Professor Elder) for allowing me to include details of the two illustrative cases.

[1] Robinson, H P, *British Journal of Obstetrics and Gynaecology*, 1975, **82**, 855 and 856.
[2] Dutt, T P, Blair, M, and Elder, M G, *American Journal of Obstetrics and Gynecology*. In press.

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Paracetamol poisoning in children

The number of patients admitted to hospital because of paracetamol poisoning has increased since 1968,[1] and in 1975 there were 105 deaths due to paracetamol.[2] A further 107 deaths occurred due to paracetamol in combination with other drugs.[2] There are no official statistics available for the incidence and severity of paracetamol poisoning in children, and we therefore reviewed information collected by the National Poisons Information Service (NPIS) over a three-year period.

Patients, methods, and results

In 1975-7 the NPIS received 3139 inquiries about paracetamol poisoning, and 580 of these concerned children under the age of 13 years. Questionnaires were sent in each case to the relevant doctor asking for details of the age and sex of the patient, the brand of paracetamol and the quantity ingested, the results of plasma paracetamol estimations and liver function tests, the treatment given, and the outcome. Replies to 116 questionnaires were received.

Analysis of the age and sex distribution (see table) showed that the patients fell into two main groups (1 to 4 years and 11 to 13 years) with different sex ratios. Of the 86 patients aged 1 to 4 years, 24 ingested paracetamol tablets, 34 ingested paracetamol elixirs, and 22 ingested Distalgesic tablets (paracetamol 325 mg and dextropropoxyphene 32·5 mg); the figures for the 27 patients aged 11 to 13 years were 16, 0, and 5 respectively (brand not stated in 12 cases). In 49 of the 86 patients aged 1 to 4 years the quantity of paracetamol taken was known, and in only eight cases did it exceed 5 g. Among 11- to 12-year-olds, however, the amount of paracetamol was known in 23 cases, and in 18 of these the amount exceeded 5 g.

Paracetamol taken alone caused vomiting and drowsiness in 15 patients. Of the 22 1- to 4-year-olds who took Distalgesic tablets, 18 had no symptoms, one suffered abdominal pain, one vomited (Distalgesic and iron tablets), one was drowsy (Distalgesic and prochlorperazine), and a 4-year-old girl who ingested 20 to 25 Distalgesic tablets became unconscious. Five children aged 11 to 13 years took Distalgesic tablets: three were asymptomatic and two became unconscious (one took 30 tablets and the other took a barbiturate as well).

Plasma paracetamol concentrations were determined in 52 patients, and in 15 cases paracetamol was not detected. In three cases the paracetamol concentrations fell above a line joining 200 mg/l at 4 hours and 80 mg/l at 12 hours after ingestion when plotted on a semilog paracetamol-time scale. In only one of these patients did the maximum plasma aspartate amino-transferase concentration (SGOT) exceed 250 IU/l: a 13-year-old girl ingested 15- to 20-g paracetamol 12 hours before hospital admission. The initial paracetamol concentration was 221 mg/l and she was given 5 g of methionine over the following 21 hours. Hepatic encephalopathy developed

Age and sex distribution of 116 children with paracetamol poisoning

Age (years)	No of patients (male:female)	Total No of patients
1-4	44:42	86
5-10	2:1	3
11-13	6:21	27

with a maximum plasma SGOT of 3129 IU/l; after haemodialysis on two occasions she made a full recovery.

Liver function tests were performed in 19 patients aged 1 to 4 years: 16 patients had normal results and three patients had maximum plasma SGOT concentrations not exceeding 250 IU/l. Ten of the 19 patients aged 11 to 13 years had normal liver function values; one patient had a maximum plasma SGOT concentration of over 250 IU/l but less than 1000 IU/l; and two patients, one of whom has already been discussed, developed plasma SGOT concentrations greater than 1000 IU/l.

Twenty-seven patients received gastric lavage, 60 ipecacuanha syrup, 19 methionine, and 20 supportive therapy only. All patients made a satisfactory recovery.

Comment

It is remarkable that with one exception there were no serious complications in the 116 children reviewed and that all made full recoveries. The child who developed hepatic encephalopathy was given methionine at a later stage than recommended,[3] which may have contributed to the encephalopathy. The patients seemed to fall into two groups: children aged 1 to 4 years, who had an equal male:female ratio and ingested small quantities of paracetamol with minimum liver damage; and children aged 11 to 13 years with a female:male ratio of 3:1 who ingested larger quantities of paracetamol with a greater likelihood of liver damage. The peak incidence of all forms of poisoning in young children is known to occur at 1 to 4 years, and this probably represents true accidental poisoning; in contrast, the high incidence of paracetamol poisoning at 11 to 13 years is likely to be due to self-poisoning. From these findings it is clear that small children rarely require treatment with a specific antidote, though older children of about 11-13 years of age may require active measures. It would seem prudent to administer to this latter group the adult dose of oral methionine (2·5 g every four hours to a total of 10 g), so long as no more than 10 hours have elapsed from the time the drug was ingested.

[1] Volans, G N, *Journal of International Medical Research*, 1976, **4**, suppl No 4, p 7.
[2] Office of Population Censuses and Surveys, *Mortality Statistics: Accidents and Violence*. London, HMSO, 1975.
[3] Crome, P, *et al*, *Lancet*, 1976, **2**, 829.

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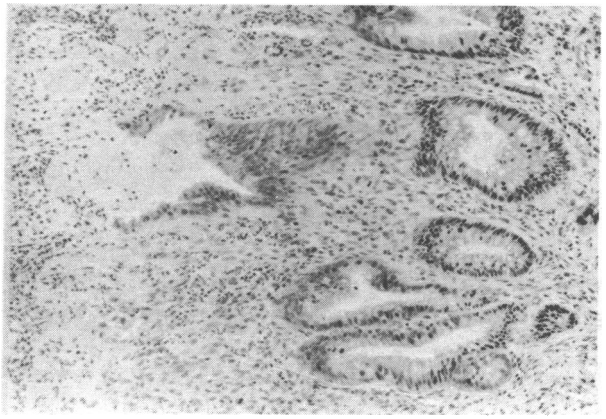
Pseudomembranous colitis in a colostomy

Pseudomembranous colitis is now a well-recognised complication of antibiotic treatment,[1] but has also been reported after surgery and in patients with chronic debilitating diseases.[2] The aetiology is not yet proved but circumstantial evidence suggests that it results from an alteration in colonic bacterial flora allowing overgrowth of a pathogenic bacterium.[3] A case of pseudomembranous colitis causing colostomy dysfunction without any preceding antibiotic treatment is described.

Case report

A 69-year-old woman was admitted to hospital with a three-month history of gradually worsening intractable diarrhoea and leakage from her colostomy. She had a history of Crohn's disease necessitating a left hemicolectomy four years before this admission. It was expected that the colostomy would require to be refashioned. On admission the mucosa of the colostomy was noted to have white-yellow plaques and endoscopy showed similar lesions within the ascending colon. A biopsy specimen of the plaques on the colostomy showed the typical features of pseudomembranous colitis. There were focal areas of glandular disruption and necrosis with associated acute inflammation and an overlying membrane of mucin, polymorphs, and fibrin typical of a type 2 lesion (see figure). A biopsy specimen of the surrounding mucosa appeared normal and there was no evidence of



Glandular disruption associated with mucin and polymorph pseudomembrane. (H and E. × 100.)

Crohn's disease. Antibiotics had not been prescribed by her hospital doctors or her general practitioner in the preceding two years. Culture of faeces failed to grow *Clostridium sordellii* or *Cl difficile*. Treatment with metronidazole (1·5 g a day) resulted in disappearance of the plaques and improvement in the patient's symptoms within seven days. The patient was discharged without requiring surgery.

Comment

The diagnosis of pseudomembranous colitis rests on the awareness of the condition and the typical histological features of an appropriate mucosal biopsy specimen,[4] and is supported by a rapid improvement in the patient's condition after treatment with vancomycin or metronidazole.[5] This case shows that pseudomembranous colitis may occur in the absence of a preceding history of antibiotic treatment and may be an unreported cause of colostomy dysfunction.

I thank Mr H I Tankel for permission to publish.

[1] Ecker, J A, *et al*, *American Journal of Gastroenterology*, 1970, **54**, 214.
[2] *British Medical Journal*, 1978, **1**, 669.
[3] George, R H, *et al*, *British Medical Journal*, 1978, **1**, 695.
[4] Price, A B, and Davies, D R, *Journal of Clinical Pathology*, 1977, **30**, 1.
[5] Dink, H T, Kernbaum, S, and Frottier, J, *Lancet*, 1978, **1**, 338.

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Correction

Prevalence of latent perhexiline neuropathy

In the paper by Dr Alain Sebille (20 May, p 1321) the name of L Rozensztajn, MD, associate professor of experimental medicine, Institute of Cardiology of the Hôpital Saint-Antoine in Paris, was omitted as a coauthor.