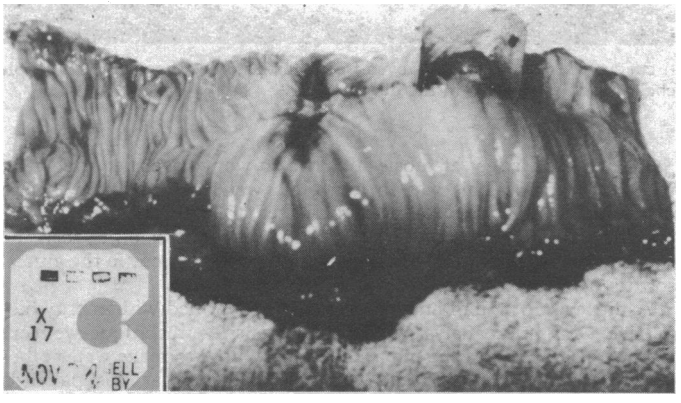
glycaemic treatment presented with a four month history of recurrent colicky abdominal pain and intermittent vomiting. Two years previously a small intestinal lipoma, which had caused an intussusception, had been resected successfully. Examination elicited slight tenderness in the right hypochondrium. Barium meal and follow through examination and oral cholecystography failed to identify the cause. At elective laparotomy various adhesions were divided and a stricture in the terminal ileum identified and resected. When the specimen was opened a bread wrapper clip was found firmly embedded in the intestinal wall and surrounded by a 2×3 cm area of mucosal ulceration. The patient made a satisfactory postoperative recovery and subsequently remained symptom free.



Case 1. Excision specimen of small intestine showing bread wrapper clip clasping and perforating full thickness intestinal wall with adjacent areas of mucosal ulceration.

## Comment

Inadvertent ingestion of foreign bodies—particularly by elderly or edentulous patients—is common but complications are apparently rare.[1 2] Our two cases suggest that ingested plastic bread wrapper clips tend to grip portions of the intestinal wall between their "jaws." Ensnaring of less than the full thickness of the intestinal wall by the clip probably causes localised necrosis and sloughing of the mucosa. As a result the clip is released back into the lumen, where it can again become attached to the mucosa at a more distal site. This sequence of events may have accounted for the areas of mucosal ulceration seen in case 1 (figure) proximal to the perforation and for the clinical presentation of intermittent abdominal pain in both patients. If the full thickness of the intestinal wall is grasped perforation (case 1) or stricture (case 2) may occur. Perforation and subacute intestinal obstruction caused by these clips have been described.[3 4]

In view of the apparent ease with which these clips may be accidentally swallowed and their tendency to damage the small intestine, bakers should seek safer methods of sealing bread wrappers. At the very least, a warning of the dangers of swallowing these clips should be placed on the bread wrapper.

With the continuing vogue for prepackaged food, physicians should consider ingested foreign bodies as a cause of recurrent abdominal pain, especially in elderly or edentulous patients.

We thank Mr W P Small for permission to report case 2.

[1] McPherson RC, Karlon M, Williams RO. Foreign body perforation of the intestinal tract. *Am J Surg* 1957;**94**:564.
[2] Bunker PG. Role of dentistry in the problems of foreign bodies in the air and food passages. *J Am Dent Assoc* 1962;**64**:335-7.
[3] Rivron RP, Jones DRB. A hazard of modern life. *Lancet* 1983;ii:334.
[4] Jamison MH, Davis RWW, Maclennan I. A plastic bread-bag clip—cause of intermittent intestinal obstruction. *Br J Clin Pract* 1983;**37**: 402-3.

(*Accepted 7 March 1984*)

**University of Edinburgh Medical School, Teviot Place, Edinburgh EH8 9AG**

N J BUNDRED, MB, BS, research fellow, department of clinical surgery
R A S BLACKIE, MB, MRCPATH, lecturer, department of pathology
A N KINGSNORTH, MS, FRCS, lecturer, department of clinical surgery
O EREMIN, FRACS, FRCSED, senior lecturer, department of clinical surgery

Correspondence to: Dr N J Bundred.

# Value of test for antinuclear antibodies in rheumatic diseases

Antibodies to nuclear antigens that fluoresce on testing are useful in diagnosing connective tissue diseases[1] but are also seen in patients with rheumatoid arthritis, when their presence is thought to predict a more severe form of the disease.[2] Tests for antinuclear antibodies are increasingly being carried out, and we evaluated the usefulness of the test in a retrospective study.

## Patients, methods, and results

We reviewed the case sheets of 470 consecutive new referrals attending this centre for over four months in 1977 in whom tests for antinuclear antibody and rheumatoid factor had been carried out. Details collected included age, sex, duration of disease, erythrocyte sedimentation rate, and the ultimate clinical diagnosis according to recognised criteria.[3] The standard test for antinuclear antibodies was performed using indirect fluorescence microscopy with rat liver as the substrate. The two stage test for rheumatoid factor using latex agglutination was carried out, and positive serum samples were retested by serial dilution with sheep red blood cells labelled with immunoglobulin (Rose-Waaler). For both antinuclear antibody and rheumatoid factor titres >1/32 were regarded as strongly positive.

We studied in more detail 102 of the patients with definite or classic rheumatoid arthritis who had been followed up for at least five years. We noted the use of second line drugs (sodium aurothiomalate, penicillamine, or levamisole), corticosteroids, or cytotoxic drugs and whether minor surgery (hand or foot) or major surgery (synovectomy, osteotomy, or arthroplasty of large joints) had been performed. Indications for drugs that modify disease remained relatively constant during the study.

The table shows the results of tests for antinuclear antibody in all 470 patients. Although results were positive in a higher proportion of patients with rheumatoid arthritis and connective tissue disease, the test was neither sensitive nor specific and strongly positive results were seen in all diagnostic categories. Titres of antinuclear antibody >1/32 were more common among those strongly positive for rheumatoid factor (28/104 patients) than those weakly positive (4/25) or negative (24/341) for rheumatoid factor.

*Results of tests for antinuclear antibody in patients with rheumatoid arthritis and other rheumatic diseases (figures are numbers (%) of patients)*

| | Negative | Weakly positive | Strongly positive |
|---|---|---|---|
| *Initial results in all patients (n = 470)* | | | |
| Osteoarthritis (n = 83) | 70 (84) | 7 (8) | 6 (7) |
| Rheumatoid arthritis (n = 156) | 94 (60) | 26 (17) | 36 (23) |
| Connective tissue disease* (n = 17) | 11 (65) | 1 (6) | 5 (29) |
| Others† (n = 214) | 185 (86) | 20 (9) | 9 (4) |
| Total | 360 (77) | 54 (11) | 56 (12) |
| *Relation of initial results in patients with rheumatoid arthritis to treatment over five years (n = 102)* | | | |
| Medical treatment: | | | |
| First line only (n = 45) | 27 (44) | 7 (41) | 11 (46) |
| Cytotoxic drugs or steroids (n = 57) | 34 (56) | 10 (59) | 13 (54) |
| Surgical treatment: | | | |
| None (n = 58) | 32 (52) | 9 (53) | 17 (71) |
| Minor (n = 19) | 10 (16) | 5 (29) | 4 (17) |
| Major (n = 25) | 19 (31) | 3 (18) | 3 (13) |

*Systemic lupus erythematosus, progressive sclerosis, polymyositis, mixed connective tissue disease, and undifferentiated connective tissue disease.
†Seronegative arthritis, gout, polymyalgia, and soft tissue rheumatism.

A five year follow up of patients with rheumatoid arthritis showed no significant correlation between the results of the initial tests for antinuclear antibody and the subsequent use of drugs that modify disease or major surgery. The same applied to the results of all tests for antinuclear antibody carried out over five years.

## Comment

Although antinuclear antibodies are found in patients with lupus erythematosus and other connective tissue diseases, they also occur in 16-34% of patients with rheumatoid arthritis.[4] Ward *et al* found that positive results related to the presence of rheumatoid nodules, vasculitis, eye lesions, Felty's syndrome, "drug toxicity," and rheumatoid factors. Condemni *et al* found a relation with nodules but not the other features, and noted instead correlation with severity of disease by anatomical functional classification.[2] Both studies considered patients with established rheumatoid arthritis already attending hospital.

In this study we assessed the prognostic value of testing for antinuclear antibody at the time of first attendance. To overcome the problems of a retrospective study of hospital case records we used use of

surgery and use of drugs that modify disease as both measures are objective and unlikely to be omitted from case notes and may be required in severe chronic destructive disease. These measures were not associated with antinuclear antibody initially or subsequently, which is particularly notable as antinuclear antibody (and correlated rheumatoid factor) may be used as criteria for selection for surgery or second line drugs. The lack of correlation strongly suggests that the presence of antinuclear antibody initially is not an adverse prognostic indicator in rheumatoid arthritis; thus the test for antinuclear antibody is of little value in the routine assessment of straightforward rheumatoid arthritis.

We thank Drs R D Sturrock and H A Capell for allowing us to study their patients, and Mrs V Eardley for typing the manuscript.

[1] Davis JS. Antinuclear antibodies. *Textbook of rheumatology*. Philadelphia: Saunders, 1981:691-709.
[2] Condemni JJ, Barnett EV, Atwater EC, Jacox RF, Morgan ES, Vaughan JH. The significance of anti-nuclear factors in rheumatoid arthritis. *Arthritis Rheum* 1965;**8**:1080-93.
[3] Arthritis Foundation. Primer on the rheumatic diseases. *JAMA* 1973;**224**: suppl 5.
[4] Ward DJ, Johnson GD, Holborow EJ. Antinuclear factor in rheumatoid arthritis: its incidence and clinical significance. *Ann Rheum Dis* 1964; **23**:306-10.

(*Accepted 7 March 1984*)

**Centre for Rheumatic Diseases, University Department of Medicine, Glasgow G4 0EH**
M M STEVEN, MRCP, senior registrar
L G TEH, MB, CHB, senior house officer
L S TEH, medical student
T PULLAR, MRCP, registrar
J J F BELCH, MRCP, lecturer
D LEWIS, FRACP, research registrar
J PARKER, MRCP, registrar
A ZOMA, MRCP, senior registrar

Correspondence to: Dr T Pullar.

# Artefactual fetal heart trace in an undiagnosed dead fetus

Electronic monitoring of the fetal heart rate during labour is an established practice in most obstetric units. We describe an unusual case in which a high quality fetal heart trace was obtained from a scalp electrode attached to a macerated dead fetus, probably as a result of electrolytic voltage production in the scalp electrode.

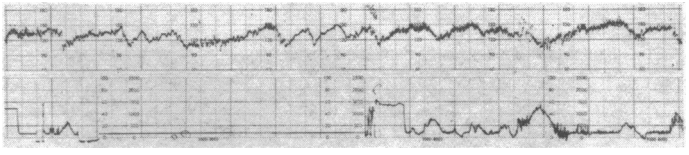
## Case report

The patient, a 35 year old primigravida, was admitted at term with a history of absent fetal movements for two days, during which she had experienced intermittent uterine contractions. No fetal heart was heard on auscultation with a Pinnard stethoscope or obtained using an external ultrasound transducer. On vaginal examination old, thick meconium was draining and the cervix was dilated 4 cm. A disposable Copeland fetal scalp electrode (A W Showell Ltd) was applied and connected to a Sonicaid FM 3 fetal monitor. Surprisingly a very clear fetal heart recording was obtained (see figure). A maternal tachycardia of 90 beats/min was present, reaching a maximum of 110 beats/min on occasions. In view of the irregularity of the fetal heart trace, we tried to obtain a sample of fetal blood. On direct vision through an amnioscope maceration of the fetal head was apparent, and intrauterine death was diagnosed. Labour proceeded and a macerated stillborn girl was delivered, weighing 3620 g.

## Comment

Electronic monitoring of the fetal heart by a scalp electrode is usually a reliable guide to the condition of the fetus during labour, but one must be aware of the possibility of recording the maternal heart rate through a dead fetus.[1 2] Conversely, fetal death has been diagnosed because of suspected transfetal recording of the maternal pulse when the fetus was in fact alive[3]—the "Lazarus syndrome." The death of a first twin has been missed because the second twin's heart rate was recorded by a scalp electrode attached to the first.[4]

In our case we discounted transmission of the maternal pulse through the dead fetus because of the discrepancy between the maternal pulse rate and the fetal heart rate when recorded simultaneously. Indeed, had we suspected intrauterine death initially we could have confirmed it by real time ultrasonography. In seeking an explanation for the trace we examined the scalp electrode and found that amniotic fluid had tracked up between the inner core and outer spiral of the



Fetal heart trace. Paper speed 1 cm/min.

electrode. This caused electrolytic activity between the differing metals of the electrode, which produced a steady voltage of around 200 mV. This steady voltage would not normally have triggered off the fetal monitor, but we found that any movement of the electrode caused a fluctuation in voltage which the monitor could interpret as a "fetal heart rate." This case illustrates a potential problem when monitoring the fetal heart rate with the type of scalp electrode described.

We thank Mr A Iles of the department of medical physics at Southmead Hospital for the electronic testing of the scalp electrode.

[1] Brandt H, Fischer WM. *Kardiotokographic*. Stuttgart: Georg Thieme Verlag, 1976.
[2] Paul RH, Petri EH. *Fetal intensive care: current concepts*. University of Southern California School of Medicine, 1973.
[3] Perkins RP, Soules MR. Misdiagnosis of fetal heart rate: an unusual case of mistaken identity. *Rocky Mountain Medical Journal* 1977;**22**: 207-9.
[4] Fehrmann H. Misdiagnosis of fetal heart rate during a twin labour. *Br J Obstet Gynaecol* 1980;**87**:1174-7.

(*Accepted 7 March 1984*)

**Department of Obstetrics and Gynaecology, Southmead Hospital, Bristol BS10 5NB**
FRASER N McLEOD, MRCOG, senior registrar
DAVID McCOY, FRCS, FRCOG, consultant obstetrician and gynaecologist

# Local skin depigmentation due to corticosteroid injection

An increasingly large number of localised cutaneous and musculoskeletal disorders are being treated by the intralesional instillation of corticosteroids (often mixed with local anaesthetic). These conditions include humeral epicondylitis, tenosynovitis, and bursitis as well as cystic acne vulgaris and hypertrophic keloid. This form of treatment is remarkably free of local and systemic complications but I report here local skin depigmentation that occurred at the site of injection—an unusual side effect that is particularly disconcerting to patients with deeply coloured skin.

## Case 1

A 22 year old African woman complained of locally painful swellings related to both tibial tuberosities. Radiographs indicated the cause to be small, ununited ossicles in the ligamentum patellae, and courses of short wave diathermy and ultrasound were prescribed without effect. The more painful lesion on the right was therefore injected with 40 mg methylprednisolone (Depomedrone). This relieved her pain and the procedure was repeated two months later on the left side with the same degree of success. Eight weeks after the second injection discrete areas of almost complete depigmentation, each measuring about 15×10 mm, appeared at both injection sites (see figure). There was no associated atrophy of the subcutaneous tissues. Normal pigmentation of the skin gradually returned, but it took two months on the right side and 11 months on the left.