substances from impacted neutrophils in the lungs. Since polyacrylonitrile membrane dialysers also activate complement but do not cause appreciable leucopenia,[3] it is more likely that the mechanism implicated in our patient was pulmonary leucostasis.

There are several other unexplained complications of dialysis which might be related to complement activation or leucostasis or both. They include arthralgia, abdominal discomfort, migraine, and ascites. Trials of polyacrylonitrile membrane (avoiding neutropenia) or polycarbonate membrane (avoiding complement activation) would seem worthwhile in these conditions.

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Osmotic fragility of erythrocytes in Duchenne muscular dystrophy

Duchenne muscular dystrophy is an X-linked recessive disorder affecting young boys. It is one of the more severe forms of muscular dystrophy and is transmitted by healthy female carriers. Onset is usually at 3-5 years and affected individuals become chairbound by the end of the first decade. Death occurs in the late teens or early twenties from cardiac failure or pneumonia. The basic defect in this disorder is as yet unknown but several recent studies[1-4] have suggested that there may be a generalised membrane abnormality affecting erythrocytes as well as muscle. Fisher *et al*[4] have reported an increase in the osmotic fragility in cases of what they refer to as "pseudohypertrophic muscular dystrophy." We report here our findings in a series of 10 patients with confirmed Duchenne muscular dystrophy and four definite carriers. (A definite carrier is defined as the mother of an affected boy with another affected male relative. The four carriers studied here had serum creatinine kinase levels of 83, 132, 213, and 344 IU/l (normal upper limit 85 IU/l).)

Patients, methods, and results

Heparinised samples of venous blood were obtained from patients (aged 1-18), their unaffected brothers (aged 1-16), and female carriers (aged 23-56) when the families attended the muscular dystrophy clinic in the department. Control samples were obtained from young boys (aged 4-15) before orthopaedic operations for disorders unrelated to neuromuscular abnormalities and healthy women volunteers (aged 17-67). The amount of lysis at various concentrations of sodium chloride was determined by the method of Dacie and Lewis.[5] The concentrations of sodium chloride resulting in 50% lysis of each sample were derived graphically.

Per cent lysis (mean ± SD) in male controls, boys with Duchenne muscular dystrophy (DMD), their unaffected brothers, female controls, and definite carriers

Sodium chloride concentration (g/l)	Male controls (n = 10)	DMD patients (n = 10)	Brothers (n = 5)	Female controls (n = 36)	Definite carriers (n = 4)
1·0	100	100	100	100	100
3·6	95·1 ± 2·9	94·4 ± 5·9	93·4 ± 6·0	95·5 ± 3·6	91·6 ± 2·8
3·8	90·7 ± 3·0	91·2 ± 7·2	83·4 ± 13·2	89·9 ± 8·5	86·5 ± 3·5
4·0	79·4 ± 12·5	83·4 ± 13·6	63·6 ± 18·3	78·8 ± 14·4	75·7 ± 11·7
4·2	50·7 ± 17·6	66·7 ± 18·3	38·5 ± 11·9	64·4 ± 18·1	49·5 ± 20·1
4·4	13·9 ± 9·9	34·0 ± 19·6	9·5 ± 2·1	40·4 ± 17·7	18·1 ± 14·9
4·6	4·5 ± 4·2	16·3 ± 13·0	4·6 ± 2·1	18·6 ± 11·3	5·8 ± 7·1
4·8	1·7 ± 1·8	6·9 ± 7·0	1·3 ± 0·7	6·8 ± 6·7	2·1 ± 3·3
5·0	0·0	1·3 ± 1·5	0·0	1·2 ± 1·9	0·0

Signs of lysis first became apparent in affected boys at a concentration of 5·0 g/l but in the controls and normal brothers at a concentration of 4·8 g/l. At all concentrations of sodium chloride from 4·4 to 5·0 g/l (see table) there was significantly greater lysis in affected boys than in either their unaffected brothers or controls ($P < 0.05$). The concentration (mean ± SD) of sodium chloride which resulted in 50% lysis was 4·18 ± 0·10 g/l for male controls, 4·30 ± 0·13 g/l for affected boys, and 4·08 ± 0·12 g/l for their unaffected brothers, the difference between affected boys and either their unaffected brothers or controls being statistically significant ($P < 0.05$). There was no difference in erythrocyte osmotic fragility between definite carriers and the control series of normal women.

Comment

These results provide additional evidence that there is a probable defect in the erythrocyte membrane in patients with Duchenne muscular dystrophy.

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Endoscopic removal of a swallowed ball bearing from stomach of a 4-year-old child

Foreign bodies commonly pass uneventfully through the gastrointestinal tract to be voided with the faeces. Thus patience and expectant treatment are usually recommended unless the size of the foreign body or its nature makes obstruction or perforation likely.[1] Hitherto in these circumstances removal by rigid endoscope or operation has been undertaken, but recently reports have described removal of various objects using fibreoptic endoscopes.[2] Grasping forceps and snares have been developed and ingenious techniques devised to retrieve objects with perforations or a waist—and even razor blades.[3] We were presented with a boy who had swallowed a ball bearing, and, as a suitable device was not available, we evolved a new method.

Case history

A fit West Indian boy aged 4 swallowed a steel ball bearing. Although well, he was brought to hospital by anxious parents. The results of examination were normal but radiographs of chest and abdomen showed a dense sphere 17 mm in diameter in the stomach. During the next six weeks he took a normal diet and did not vomit, yet serial radiographs showed the ball still in the stomach. As there was no sign of passage beyond the pylorus surgical removal was contemplated, but before laparotomy endoscopic removal was attempted.

A standard latex contraceptive condom was attached by cotton thread to the distal end of an Olympus GIFD2 endoscope. Three equally spaced ties were passed through the condom around the rolled rim and the free ends threaded through the lumen of the rubber hood detached from the