Haematological data during course of illness in relation to treatment.

Conversion: SI to traditional units—Blood counts: $1 \times 10^9/l = 1000/mm^3$.

1.5 mg/kg body weight, later increased to 4.5 mg/kg. Corticosteroid treatment was stopped after three weeks. The course of her subsequent illness and its treatment is summarised in the figure.

Her Hb and WBC having remained nearly normal for four months since the last blood transfusion, oxymetholone was discontinued. Five months later the original symptoms returned and her blood was again pancytopenic. She required transfusions, and oxymetholone was restarted. Again her condition remitted, but this time oxymetholone was continued, although in a decreasing dose. In July 1973 her Hb was 17.0 g/dl, WBC $4.10 \times 10^9/l$ (4100/mm³), and platelets $48.0 \times 10^9/l$ (48 000/mm³), and she continued on oxymetholone 0.7 mg/kg. She returned in April 1974 complaining of breathlessness, paroxysmal nocturnal dyspnoea, leg cramps, and shaking of the left arm. She had a plethoric face, ankle oedema, orthopnoea, and a systolic murmur. The spleen was not palpable. There was a Parkinson-like tremor of the left arm. Investigations showed Hb 18.1 g/dl, packed cell volume 56%, a ⁵¹Cr red cell mass of 33.4 ml/kg, 26% above the upper limit of normal. Total WBC was $5.30 \times 10^9/l$ (5300/mm³), with a normal differential count. The platelet count was $35.0 \times 10^9/l$ and the erythrocyte sedimentation rate 1 mm in 1 hour. Iliac crest biopsy showed a normocellular marrow with no evidence of myeloproliferation. Pulmonary function was normal and P_{O_2} 11.1 kPa (83 mm Hg). Liver scan and excretion urogram were normal and showed a spleen of normal size. α -Fetoprotein was undetectable. She was treated for heart failure. 500 ml of blood was withdrawn through an antecubital vein, and oxymetholone was again stopped. She improved with disappearance of breathlessness, night sweats, cramps, and tremor.

Five months after stopping oxymetholone she was again ill with Hb 8.1 g/dl, WBC $2.10 \times 10^9/l$ (2100/mm³) (neutrophils $0.4 \times 10^9/l$ (400/mm³)), and platelets $23 \times 10^9/l$ (23 000/mm³). Blood transfusions were again started, and so was oxymetholone 300 mg/day. Five months later Hb had risen again to 18.1 g/dl, WBC $4.10 \times 10^9/l$ (4100/mm³) (neutrophils $1.75 \times 10^9/l$ (1750/mm³)) (neutrophils $1.75 \times 10^9/l$ (1750/mm³)), and platelets $71.0 \times 10^9/l$ (71 000/mm³). Oxymetholone was reduced to 10 mg/day. After six months at this dose the blood remains normal, except for a platelet count of $100-400 \times 10^9/l$ (100 000-400 000/mm³) and the patient is feeling well, with no abnormal physical signs.

Discussion

The unusual feature of this case is the considerable sensitivity to oxymetholone, the blood picture changing from anaemia to polycythaemia and each causing distressing symptoms. Indeed, polycythaemia first occurred after nine months on only 0.7 mg/kg. (50 mg/day), whereas the recommended dose is about 2.5 mg kg⁻¹ day⁻¹ or more. Polycythaemia was probably not present at the outset, since she did not appear clinically polycythaemic in the summer of 1970, nor did her subsequent course or investigations favour a diagnosis of polycythaemia rubra vera. Oxymetholone was finally implicated in causing polycythaemia when anaemia recurred on withdrawing it and polycythaemia reappeared on its reintroduction. Further investigations and the subsequent history have not uncovered any other contributing factor.

The most important feature, however, is the proof that oxymetholone can stimulate aplastic marrow. Severe symptomatic pancytopenia regressed three times with oxymetholone, yet recurred twice within five months of stopping this drug. Drug-induced aplastic anaemia has a 5-year mortality rate of about 70% despite treatment, and the value of oxymetholone has been questioned.^{2,3,5} The evidence of this and other reported "oxymetholone-dependent" patients^{1,5} shows that a group of patients exists for whom oxymetholone can be life-saving—especially if death from haemorrhage, infection, and anaemia can be

prevented during the three to four months which it takes for the stimulation of bone-marrow function by oxymetholone to be apparent.

We thank Professor Alan Read for permission to report this case. Correspondence should be addressed to Dr Thomas S Low-Beer.

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Neonatal jaundice in association with oxytocin stimulation of labour and operative delivery

Most recent attempts¹⁻⁴ to explain the relationship between oxytocin stimulation of labour and the development of neonatal jaundice have not considered the potentially confounding effect of the delivery process. The exception is that of Campbell *et al*,⁵ which showed a temporal trend in neonatal jaundice equalling the increased use of oxytocin and epidural anaesthesia. Their findings interestingly revealed more abnormal deliveries (forceps, breech, and especially ventouse) in the jaundiced group. These procedures were more common in mothers who had been given epidural anaesthesia. The association between oxytocin and abnormal delivery was not studied, however.

Patients, methods, and results

Our review of the 13 166 term babies born from 1969 to the end of 1974 (exclusive of those with Rh and ABO incompatibility, and those weighing less than 2500 g) failed to show significant differences in the incidence of bilirubinaemia exceeding 171 μ mol/l (10 mg/100 ml) among infants delivered after unstimulated, oxytocin-induced, and oxytocin-augmented labours ($\chi^2=2.2$; DF=2; not significant). Analysis clearly showed notable differences according to the type of delivery procedure to which the fetus had been subjected ($\chi^2=21.1$; DF=4; $P<0.001$). This relationship applied to those born after induced and augmented labours as well as after unstimulated labours. A slightly greater frequency of bilirubinaemia was encountered after stimulated labours (as contrasted with unstimulated labours) among babies delivered through the vagina, but here the differences were negligible (see table).

Incidence of neonatal jaundice as related to labour stimulation and delivery method

Delivery	Unstimulated labour		Oxytocin-induced		Oxytocin-augmented		Total	
	No	%	No	%	No	%	No	%
Spontaneous ..	4997	7.0	565	7.4	1123	7.8	6685	7.2
Low forceps ..	2535	8.2	305	9.8	1030	8.4	3870	8.4
Midforceps ..	441	10.7	61	11.5	332	11.7	834	11.2
Cesarean section ..	1269	7.3	41	2.4	260	6.9	1570	7.1
Assisted breech ..	176	11.9	8	0.0	23	13.0	207	11.6
Total	9418	7.7	980	8.2	2768	8.5	13166	7.9

Moreover, no synergistic effect was demonstrable by which oxytocin stimulation in any way enhanced the association between the delivery procedure and the development of neonatal bilirubinaemia, even among infants delivered by the potentially more traumatic methods of mid-forceps or assisted breech. The increased incidence of bilirubinaemia in unstimulated and stimulated labours was entirely comparable for infants delivered vaginally

in vertex presentation (rate ratio was 1.1 for spontaneous, low forceps, and mid-forceps deliveries, but 0.9 for caesarean sections and 0.8 for breech deliveries).

Comment

It is recognised that demonstrable trauma associated with difficult operative vaginal delivery, resulting in bruising, leads to neonatal jaundice. When such clinically apparent cases were excluded by Campbell *et al.*,⁵ however, they still found more instrumental procedures in the jaundiced group. We would suggest that operative instrumental delivery may cause focal haemorrhages that are not readily detectable in the infant, but may nevertheless subsequently be manifested by the appearance of jaundice.

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Successful treatment of pneumatosis coli with oxygen

Pneumatosis coli is a condition of multiple gas-filled cysts in the subserosal and submucosal layers of the large bowel. Forgacs *et al.*¹ first described successful treatment with high concentrations of oxygen. Since then there have been only a few similar reports.

Case report

A 69-year-old woman presented with a 12-month history of excessive flatus and a 6-month history of passing up to 10 small motions daily. She had suffered from chronic obstructive airways disease for more than 40 years. She had never smoked and the plasma α -1-antitrypsin was normal at 2.4 g/l (0.024 mg/100 ml). She also had mild angina of effort and had had three minor cerebral thromboses with residual right-sided weakness and nominal

dysphasia. She had taken corticosteroids for five years for her chest disease and had developed diabetes mellitus, which was controlled by diet and glibenclamide.

She had a distended, tympanic abdomen. A polypoid mobile mass was palpable in the rectum. Sigmoidoscopy, abdominal x-ray examination, barium enema, colonoscopy, and rectal biopsy showed features of pneumatosis coli (fig 1). She was treated with high concentration oxygen for 20 hours daily for five days. An arterial oxygen tension of 40.5 kPa (304 mm Hg) was easily achieved with a 60% oxygen face mask and nasal cannulae at 4 l/min. The response to treatment was dramatic. Within 48 hours the frequent bowel movements ceased and the bowel habit was still normal after five months. The rectal mass disappeared rapidly and a repeat colonoscopy (one week) and barium enema (one month) showed a normal colon.

Discussion

The patient's symptoms and the presence of a rectal mass suggested a diagnosis of rectal carcinoma but the simple investigations of plain abdominal x-ray examination and sigmoidoscopy were diagnostic of pneumatosis coli, and major surgery in a high-risk patient was avoided. The case exemplifies the association between pneumatosis of the bowel and obstructive airways disease,² and this should be remembered when patients with lung disease present with diarrhoea or a change in bowel habit. Rectal bleeding and prolapse may also occur.^{3 4}

High concentration oxygen treatment is a cheap and simple form of treatment for pneumatosis coli. Although the cause of the condition is unknown, Forgacs *et al.*¹ correctly predicted that displacing nitrogen with oxygen in the lungs, and thus reducing the total partial pressures of gases in the venous blood, would deflate the cysts. They emphasised that oxygen administration must be continuous, but Dean and Castleden⁵ found intermittent therapy effective and allowed the patient more freedom.

I thank Dr Lindsay Davidson for his helpful criticism and permission to report this case.

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Barium enema before treatment showing multiple cystic filling defects.

Ocular toxicity due to rifampicin

Rifampicin is widely used as one of the first-line drugs in the chemotherapy of tuberculosis. Though oculotoxic effects such as loss of acuity of vision and colour blindness have been reported in a case treated with both rifampicin and ethambutol,¹ rifampicin alone has not yet been reported to be oculotoxic. We report below a case of reversible rifampicin oculotoxicity.

Case report

A 34-year-old man suffering from pulmonary tuberculosis (AFB-positive) was given daily the triple oral regimen of isoniazid (300 mg), rifampicin (450 mg), and ethambutol (1200 mg). After a week both eyes were painful, tender, red, and congested with thick white secretions. The drugs were stopped and the eyes cleared up within 72 hours. Treatment was then changed to isoniazid, PAS (10 g daily), and streptomycin (0.75 g intramuscularly daily for five days a week). He developed nausea and vomiting, probably due to PAS, and the drugs were stopped. He was then kept on isoniazid (300 mg daily) only for a week. There were no adverse reactions. Rifampicin (450 mg daily) was then added, and within 48 hours the same type of ocular reactions again developed. He was not taking any other drug