with Hb A1c. Patients with very high Hb A1c values possibly represent a subgroup of the diabetic population, outside the normal criterion of long-term control, during a phase of metabolic decompensation; and they may show features more typical of the lipid disturbances of ketosis than of long-term diabetic control.

Thus our main finding is related not to diabetic control but to the type of diabetes, in that patients with maturity-onset diabetes have abnormally low HDL cholesterol concentrations irrespective of vascular disease and, possibly, of mode of treatment, whereas patients with juvenile-onset diabetes have essentially normal concentrations of HDL cholesterol.

We thank Professor M G Nelson and Mr D W Neill, principal biochemist, for laboratory facilities, and Mrs Mildred Fry and Miss May Weller for their help.

References
2 Miller, G J, and Miller, N W, Lancet, 1975, i, 16.

SIDE EFFECTS OF DRUGS

Ritodrine-induced acidosis in pregnancy

Beta-sympathomimetic agents such as salbutamol, terbutaline, and ritodrine are commonly used to prevent premature delivery but may have metabolic side effects, particularly in the case of salbutamol. Enhanced lipolysis and glycolysis result in raised blood glucose, fatty acid, and lactate concentrations and a fall in serum bicarbonate. Severe metabolic acidosis, however, has been described only in pregnancy complicated by diabetes. We report a case of decompensated acidosis that occurred during an infusion of ritodrine.

Case report

An 18-year-old woman in the 32nd week of pregnancy was admitted to the department of obstetrics having been in labour for about two hours. The cervix admitted one finger and the membranes were intact. Ritodrine, diluted in 5% dextrose in water, was infused by a pump at 100 micrograms/minute to delay delivery. Hydrocortisone succinate 1 g four times daily was given intravenously to prevent neonatal lung prematurity. Twelve hours after admission abdominal tenderness unrelated to uterine contractions was noted. Fetal heart rate monitoring showed a persistent tachycardia of 170/min and some late decelerations. The mother also had tachycardia but was otherwise physically normal. Attention was drawn to a serum bicarbonate concentration of 8 mmol/l and a 10 mmol/l anion gap. Qualitative test for ketonuria was negative. Blood glucose concentration was 8.8 mmol/l (159 mg/100 ml) and pH 7.25. Ritodrine infusion was slowed to 30 micrograms/minute and sodium bicarbonate (40 mmol/hour) given. Three hours later the serum lactate concentration was 7 mmol/l (63 mg/100 ml) and the anion gap 8 mmol/l. Ritodrine was stopped, and within three hours the lactate and bicarbonate concentrations were 2.7 mmol/l (24.3 mg/100 ml) and 20 mmol/l respectively. Delivery occurred 24 hours after admission; mother and child did well.

Comment

Although we cannot exclude the possibility of mild gestational diabetics in this case, normal fasting and postprandial blood glucose concentrations the day after delivery strongly suggest that the patient had a normal glucose tolerance. We therefore conclude that ritodrine, associated with corticosteroids, induced transient severe acidosis with predominant hyperlactacidemia.

Blood electrolyte and glucose concentrations should be carefully monitored in non-diabetic patients given ritodrine for premature labour to detect potentially harmful metabolic acidosis.

Reversible oliguric renal failure associated with ibuprofen treatment

Nephrotoxicity has been associated with non-steroidal anti-inflammatory agents such as phenylbutazone, oxyphenbutazone, and aspirin. It has not been reported after use of ibuprofen, although similar propionic-acid derivatives—namely, fenoprofen and naproxen—have been associated with renal papillary necrosis. We report here a case of reversible oliguric renal failure, which occurred in a patient with osteoarthritis treated with normal doses of ibuprofen.

Case report

A 65-year-old man was admitted to this hospital with acute left-sided weakness. His medical history included atherosclerotic and hypertensive cardiovascular diseases treated with frusemide, spironolactone, and digoxin;
SHORT REPORTS

Heparin and infusion phlebitis

The problem of infusion phlebitis has produced a variety of explanations and advice on avoidance. The cause is not known—particularly, infection, reaction to the canula and to infusion fluids, or simple trauma are all possible but unproved causes. In some hospitals the policy is to change the site of drips every day. In a busy surgical unit there is not the time available for this and many patients do not have suitable veins. In practice, most drips are left in situ until early phlebitis occurs, they stop spontaneously, or are no longer required. Anecdotally, heparin is said to prolong the life of drips but the advice on this is conflicting. The present study was designed to test the hypothesis that small doses of heparin, either subcutaneously or added to the infusion, both prolong drip life and reduce infusion-related phlebitis.

Methods and results

A total of 60 consecutive patients entering a general surgical unit under the care of one particular consultant, having intravenous infusions expected to continue for more than 5 days, were entered for this trial. The included patients admitted for routine and emergency procedures but excluded those actively bleeding from any site. No other groups were excluded. Patients admitted for routine procedures at high risk from pulmonary embolism were excluded as routine practice. 5000 units of subcutaneous (sc) heparin three times a day. Patients were alternately allocated to a control group receiving no intravenous (iv) heparin or to a group receiving a total of 2000 units of heparin per day via the infusion. This was divided into four doses of 500 units given six hourly into the infusion bags and did not affect the partial thromboplastin time.

Drip life and reasons for discontinuation in control patients and those receiving subcutaneous (sc) or intravenous (iv) heparin

<table>
<thead>
<tr>
<th>Mean (± SD) drip life in days (n)</th>
<th>Controls</th>
<th>sc heparin</th>
<th>iv heparin</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>2.1 ± 1.0</td>
<td>3.4 ± 1.6</td>
<td>5.2 ± 3.2</td>
</tr>
<tr>
<td>P values</td>
<td>0.005</td>
<td>0.025</td>
<td>0.001</td>
</tr>
<tr>
<td>Reasons for discontinuation</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Phlebitis</td>
<td>6</td>
<td>4</td>
<td>5</td>
</tr>
<tr>
<td>Spontaneous stoppage</td>
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<td>4</td>
<td>1</td>
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<tr>
<td>Elective removal</td>
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<td>7</td>
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<tr>
<td>P values for spontaneous</td>
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<tr>
<td>thrombus versus elective removal</td>
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<td>0.025</td>
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of 500 units given six hourly into the infusion bags and did not affect the partial thromboplastin time.