turing it with a fine needle introduced into the right atrium through the chest wall.

My thanks are due to Mr. David Watson who carried out the needling of the balloon in Case 1 and to Dr. Clive Bowkett who helped in the care of these infants.

References

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Department of Paediatric Cardiology, Leeds Regional Thoracic Surgical Centre.

Addendum
Since submission of this report, two further examples of the same complication have been described elsewhere (Ellison et al., 1970).

References

Association of Hypoglycaemia with Cardiac Enlargement and Heart Failure in Newborn Infants
A retrospective review of the chest x-rays of infants with hypoglycaemia seen in this hospital has shown that hypoglycaemia in the newborn is frequently associated with cardiac enlargement, sometimes with signs of heart failure.
**Short Reports**

**Material**

Forty-three newborn infants with hypoglycaemia (blood glucose level < 25 mg./100 ml.) were seen from November 1965 to October 1969. Infants of diabetic mothers were not included. 16 of the infants had undergone chest radiography. Heart failure was diagnosed in the presence of the following signs: (1) dyspnoea and respiratory rate above 50/minute; (2) radiological evidence of cardiac enlargement with pulmonary congestion; and (3) hepatomegaly. Cardiac enlargement was diagnosed when the cardiothoracic (CT) ratio was greater than 2 SD above the normal mean (0.50 ± SD 0.03) for newborn infants in this hospital.

**Results**

Twelve of the 16 x-rays showed cardiac enlargement. 4 of these infants fulfilled the criteria for clinical heart failure. The findings in these patients are summarized in the Table.

All infants were born at term. 6 had birthweights lower than 2 SD below the mean for their gestational ages (3 of these had heart failure). The infants who developed cardiac failure first became ill 12–55 hours after birth with dyspnoea and cyanosis and one became comatose. One of the 8 infants with cardiomegaly without failure was asymptomatic, another had apnoea at birth, the others became symptomatic between 5 hours and 3 days of age. 2 had apnoeic spells, 1 central cyanosis, 1 muscular tremors, and 5 had convulsions.

One infant with cardiac failure had heart catheterization studies at 2 weeks of age, which showed a small left-to-right shunt at the atrial level. It is unlikely that this was responsible for the cardiac enlargement and heart failure. Other cardiac findings were non-specific and included tachycardial, overactive precordium, and gallop rhythm. All 4 patients with heart failure and one without failure had one or more ECGs; all were abnormal with peaked P waves in lead I, R wave voltages in lead V1 greater than 20 mm., and greater than normal praeordial T wave inversions.

In all infants the hypoglycaemia was treated with intravenous glucose infusions. Of those with cardiac failure, 1 improved rapidly, while 3 showed little improvement. When the blood glucose levels of these patients remained low corticosteroids were given. The infants without heart failure showed a much more rapid clinical and biochemical response to glucose, and only 2 received corticosteroids.

One infant with cardiac failure died at 7 days of age from cerebral haemorrhage. The heart at necropsy weighed 26 g. (normal for age: 20 g.) and was anatomically normal. The other 3 infants with cardiac failure had normal hearts on follow-up examination (Fig.). 3 of the 8 infants without cardiac failure had follow-up x-rays and all showed normal heart size; 2 others had clinically normal hearts at follow-up.

**Discussion**

Benzing et al. (1969) described 27 infants with cardiac failure and hypoglycaemia; 26 had severe cardiac malformations and 22 died. The only infant without a malformation exhibited cardiac failure at 11 days of age, and by 18 months the heart was normal. Apart from this patient, 1 briefly described by Miller (1944), and 8 by Reilly et al.

---

**TABLE**

**Summary of Findings in 12 Infants with Hypoglycaemia and Cardiac Enlargement**

<table>
<thead>
<tr>
<th>No.</th>
<th>Sex</th>
<th>Gest. (wk.) (mean, range)</th>
<th>Birthweight (kg.) (mean, range)</th>
<th>Age at Onset of Symptoms</th>
<th>Age of First Blood Glucose (hr.) (mean and range)</th>
<th>Greatest Cardiathoracic Ratio (mean and range)</th>
<th>Additional Findings</th>
<th>Outcome</th>
</tr>
</thead>
<tbody>
<tr>
<td>With cardiac failure</td>
<td>4 (3M, 1F)</td>
<td>40-5 (39-42)</td>
<td>2-55 (1-93-3-57)</td>
<td>12-55 hr.</td>
<td>44 (14-60)</td>
<td>0.71</td>
<td>Abnormal ECG; 4; hypocalcaemia, 1; small atrial septal defect, 1</td>
<td>1 died at 7 dy.; 1 mental retardation at 22 mth.; 1 mental retardation with convulsions at 3 yr.; 1 normal at 1 yr.</td>
</tr>
<tr>
<td>Without cardiac failure</td>
<td>8 (5M, 3F)</td>
<td>39-5 (37-40)</td>
<td>3-53 (1-92-5-53)</td>
<td>Birth—3 dy. (1 patient asymptomatic)</td>
<td>38 (5-96)</td>
<td>0.65</td>
<td>Abnormal ECG; 1; hypocalcaemia 3</td>
<td>All recovered; 1 mental retardation at 1 yr.; 1 recurrent hypoglycaemia; 3 normal at 6 mth., 1 and 2 yr.</td>
</tr>
</tbody>
</table>
(1969), the association of hypoglycaemia and heart failure in infants of non-diabetic mothers does not seem to have received attention.

There are many difficulties in assessing the heart size of newborn infants, and radiographic techniques vary from centre to centre. In the crying infant the maximum heart size occurs after inspiration, but this maximum is no different from the maximum at rest. During the crying effort transverse cardiac diameter may be reduced 12% (Burnard and James, 1961a). In our hospital, infants are made to cry in order to time inspiration, and it might be argued that infants who are able to cry vigorously will have smaller hearts than sick infants who cannot cry. However, the mean CT ratios of the hypoglycaemic infants with and without cardiac failure were 42% and 30%, respectively, above the normal mean, and it is unlikely that the crying factor accounted for these large differences in heart size.

Cardiac enlargement not due to primary heart disease or maternal diabetes may occur in the newborn with asphyxia (Burnard and James, 1961b), haemolytic disease of the newborn (Miller, 1944), and with polycythaemia (Gatti et al., 1966). Asphyxia might have been responsible for the abnormal heart size and cardiac failure in one of our patients, but not in the remainder. Two infants had Hb levels greater than 22 g./100 ml., but polycythaemia was not a factor in the other infants.

The fetus is thought to burn predominantly carbohydrate for energy, and the normal term infant has large carbohydrate stores in the liver and heart which are rapidly depleted after birth, especially in the presence of hypoxia (Shelley, 1964). The ability of the newborn animal to withstand anoxia is closely correlated with the initial cardiac glycogen content (Dawes, Mott, and Shelley, 1959). In contrast to adult hearts, hearts of some newborn animals have a limited capacity to oxidize long chain fatty acids and a greater capacity to oxidize glucose (Wittels and Bressler, 1965). If the same is true for the newborn human heart, deprivation of glucose might result in functional insufficiency.

These patients were selected retrospectively from a group of infants with hypoglycaemia. Cardiac failure was present in one-quarter and cardiac enlargement in three-quarters of the patients who had had chest x-rays. We do not wish to imply any cause and effect relation between the hypoglycaemia and the cardiac abnormalities, for both may have been secondary to another insult such as asphyxia. We suggest that cardiac enlargement and heart failure be looked for in any newborn infant with hypoglycaemia and vice versa. The presence of hypoglycaemia and heart failure does not, however, rule out serious congenital heart malformations.

Summary

A retrospective review of chest x-rays of 16 infants with neonatal hypoglycaemia, not due to maternal diabetes, showed cardiomegaly in 12. 4 also had cardiac failure. None of the infants had cardiac malformations which could have accounted for the cardiac findings. One infant died from cerebral haemorrhage. 8 infants were followed up, and in all of them the heart was normal. It is concluded that hypoglycaemia in the newborn is frequently associated with cardiac enlargement and heart failure.
Fasting Growth Hormone in Treated Diabetic Children

Since growth hormone administration in diabetic patients causes deterioration of their diabetic state (Luft, 1965), the poor control seen in some diabetic children might be the result of increased growth hormone secretion. Diabetic control and growth hormone levels have been correlated in a series of diabetic children.

Patients and Methods

Twenty-four diabetic children who were treated with insulin for 1 to 10 years were investigated. 10 were girls and 14 were boys. Their ages ranged from 5 years to 15 years 9 months. In half, the diabetic control was considered good, while in the remaining half the control was poor. The criteria for assessment of diabetic control are shown in Table I.

Control data were obtained from 13 non-obese metabolically and endocrinologically normal children with no family history of diabetes or coronary disease. 6 were girls and 7 were boys. Their ages were similar to the diabetic children ranging from 4 years 8 months to 15 years 2 months.

<table>
<thead>
<tr>
<th>Control No.</th>
<th>Age (yr.)</th>
<th>Sex</th>
<th>Fasting Blood Glucose (mg./100 ml.)</th>
<th>Fasting HGH (ng./ml.)</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>4 8/12</td>
<td>M</td>
<td>64</td>
<td>&lt;1</td>
</tr>
<tr>
<td>2</td>
<td>5</td>
<td>M</td>
<td>64</td>
<td>17</td>
</tr>
<tr>
<td>3</td>
<td>5 4/12</td>
<td>M</td>
<td>66</td>
<td>&lt;1</td>
</tr>
<tr>
<td>4</td>
<td>5 6/12</td>
<td>F</td>
<td>62</td>
<td>2</td>
</tr>
<tr>
<td>5</td>
<td>7 3/12</td>
<td>F</td>
<td>64</td>
<td>5</td>
</tr>
<tr>
<td>6</td>
<td>7 11/12</td>
<td>F</td>
<td>66</td>
<td>&lt;1</td>
</tr>
<tr>
<td>7</td>
<td>8 4/12</td>
<td>M</td>
<td>63</td>
<td>&lt;1</td>
</tr>
<tr>
<td>8</td>
<td>9 1/12</td>
<td>M</td>
<td>68</td>
<td>&lt;1</td>
</tr>
<tr>
<td>9</td>
<td>9 5/12</td>
<td>F</td>
<td>58</td>
<td>&lt;1</td>
</tr>
<tr>
<td>10</td>
<td>13</td>
<td>M</td>
<td>59</td>
<td>&lt;1</td>
</tr>
<tr>
<td>11</td>
<td>13 6/12</td>
<td>M</td>
<td>70</td>
<td>2</td>
</tr>
<tr>
<td>12</td>
<td>14 7/12</td>
<td>F</td>
<td>69</td>
<td>6</td>
</tr>
<tr>
<td>13</td>
<td>15 2/12</td>
<td>M</td>
<td>72</td>
<td>1</td>
</tr>
</tbody>
</table>

Mean          | 7 8/12    | 65-6 | 3-0
Range         | 15 2/12   | 58-79| <1-17

All patients were fasted overnight. None of the diabetic children had received insulin before blood collection. All diabetic children were treated with a mixture of semi-lente and ultra-lente insulin. Venous blood was collected from peripheral veins with minimal stasis. Aliquots were transferred to fluoride-oxalate tubes for glucose analysis and to heparin tubes for growth hormone assays. Plasma was stored at -20 °C. until assayed. Blood glucose was determined by an automated glucose-oxidase method described by Discombe (1963); plasma growth hormone was measured by the double antibody radioimmunoassay method of Hartog et al. (1964).

Results

The fasting blood glucose and plasma growth hormone results from the control children and from the diabetic children are shown in Tables II and III.
Association of hypoglycaemia with cardiac enlargement and heart failure in newborn infants.
O Amatayakul, G R Cumming and J C Haworth

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