Summary

A computer system has been used for the recording and display of data from 277 infants with respiratory illnesses consecutively admitted to a neonatal intensive care unit during a period of 25 months. Improved efficiency of data handling was demonstrated, and the system proved valuable for teaching and research.

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Effect of phototherapy on thermal environment of the newborn

The aim of this study was to investigate the effects of the radiant energy emitted by phototherapy lights on the thermal environment of incubator- and cot-nursed babies, and to determine whether there were situations in which babies might be exposed to adverse thermal conditions.

Materials and method

The 40 babies included in this study were undergoing phototherapy for hyperbilirubinaemia in the neonatal unit of the City Hospital, Nottingham. Incubator-nursed babies had a mean birthweight of 1·94 kg (±0·15 SEM) and were studied at an average age of 4 days. Cot-nursed babies were heavier (mean birthweight 3·01±0·12 kg) and were studied at an average age of 6 days. The jaundice was considered to be physiological in all cases, but aggravated by prematurity in 13. In a further 3 babies bruising or cephalhaematoma was present. Infants with Rhesus or ABO incompatibility were not included.

Phototherapy was administered with either the Vickers unit, containing four 58 cm 40 watt tubes, or the Air Shields unit, containing eight 58 cm 20 watt tubes. Both units were used either 60 cm above the mattress of an open cot, or 40 cm above the mattress of an incubator.

Ambient temperatures were measured with thermocouples and recorded by a Cambridge multichannel recorder. The room temperature, mattress surface temperature, and air temperature 2 cm above the mattress were measured continuously. The suitability of the operative environmental temperature was assessed by measuring the baby's rate of oxygen consumption. An open circuit oxygen consumption apparatus has been developed that does not require moving the baby to a metabolic chamber (Smales, 1978).

All measurements were made 1½ hours after a feed. Incubator air temperature was recorded from the thermometer in the canopy of the incubator and an allowance made for radiant heat loss according to the temperature of the room air (Hey and Mount, 1967). In 10 babies, nursed naked in incubators, the rate of O_2 consumption was measured after an hour's phototherapy. After the following feed phototherapy was discontinued and a second measurement of O_2 consumption was made an hour later. Incubator temperature varied by less than 0·8°C for each pair of measurements.

In another 20 babies the rate of O_2 consumption was measured after an hour's phototherapy over an

open cot. Half the babies were treated with the Vickers phototherapy unit and half with the Air Shields unit. After the following feed phototherapy was discontinued and the babies were dressed in a nappy and nightshirt and covered with a blanket. The rate of O_2 consumption was measured again an hour later.

In a further 10 babies the effect of covering a cot during phototherapy was studied. O_2 consumption was first recorded after an hour's phototherapy without the perspex cover in position, and after the next feed phototherapy was continued with the cover in place and oxygen consumption measured an hour later. Both phototherapy units were included in the group.

Results

The Vickers 59 and 79 incubators have a temperature gradient of 0·8°C between the warmest point recorded by the thermometer in the canopy and the coolest point at the surface of the mattress. Both phototherapy units removed or reversed this gradient and within 2 hours they had also caused a rise of an average 1·8°C in the room temperature. The use of the phototherapy lamps reduced the rate of O_2 consumption of the infants nursed in incubators from $7{\cdot}8 \pm 1{\cdot}0$ ml O_2/kg per min (mean $\pm$ SE) to $6{\cdot}9 \pm 0{\cdot}8$ ml O_2/kg per min. Using Wilcoxon's Matched Pair Signed Ranks test, (Wilcoxon, 1945) this was significant at the $P<0{\cdot}005$ level.

The use of the Air Shields phototherapy unit over a cot raised the temperature of the surface of the mattress in 2 hours by an average of 4·6°C. The air temperature 2 cm above the mattress rose on average 2·5°C. The temperature changes produced by the Vickers unit were in the same direction but slightly smaller.

The rate of O_2 consumption of babies dressed and covered by a blanket in the cot was $7{\cdot}1 \pm 0{\cdot}8$ ml O_2/kg per min. When they were placed naked under the Air Shields phototherapy lamp, their rates of O_2 consumption increased to $8{\cdot}0 \pm 1{\cdot}2$ ml O_2/kg per min ($P<0{\cdot}01$). The rise was also significant when the Vickers unit was used ($P<0{\cdot}005$). This effect was reduced by using a transparent perspex cot cover. The rate of O_2 consumption of babies nursed under the cover was $7{\cdot}8 \pm 0{\cdot}7$ ml O_2/kg per min, which was significantly less than that found when the cover was not used ($8{\cdot}5 \pm 1{\cdot}4$ ml O_2/kg per min; $P<0{\cdot}01$).

Discussion

The fluorescent tubes used in phototherapy units emit a wide spectrum of radiant energy. The photo-oxidation of bilirubin involves energy in the 440–470 nm waveband with the peak effect in the blue part of the visual spectrum. Although this is transmitted freely by the perspex of incubators, wavelengths greater than 650 nm, in the infrared spectrum, are partly absorbed and result in an increase in surface temperature. Some energy is transmitted and increases the baby's surface temperature (Wu and Berdahl, 1974). An increase in skin blood flow under these conditions has been considered to be a response to facilitate evaporative heat loss (Oh *et al.*, 1973). This, in association with an increase in respiratory rate, is the basis of the high insensible water loss noted in babies receiving phototherapy in incubators (Oh and Karecki, 1972).

The rise in temperature recorded at the mattress surface in both incubators and cots confirms that phototherapy units do emit significant radiant heat. Although incubator air temperature remains stable, the additional radiant heat increases the 'operative' environmental temperature. The fall in O_2 consumption recorded during phototherapy suggests that the original incubator temperature was below the thermoneutral temperature. If higher incubator temperature is used, phototherapy may result in the baby becoming overheated. A reduction in incubator air temperature should be made if operative environmental temperature is to remain constant.

When a phototherapy unit is used over an open cot the radiant heat output only partly compensates for the loss of insulation possessed by normally wrapped babies. The rise in O_2 consumption recorded with both phototherapy units indicates a small but significant metabolic response to cooling. If phototherapy is carried out at room temperatures below 25–28°C a larger response would be expected and this may have adverse clinical implications. The value of a perspex cover during phototherapy has been clearly shown.

Summary

Oxygen consumption was studied in 40 newborn babies undergoing phototherapy for hyperbilirubinaemia, both when enclosed within an incubator or nursed in an open cot. Exposure to phototherapy caused a rise in temperature at the mattress surface. This could result in overheating a baby nursed in an incubator, but by contrast is likely only partially to compensate for the loss of insulation of an unclothed baby in an open cot.

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Severe arrhythmias in Coxsackievirus B3 myopericarditis

Proven viral myopericarditis only rarely presents with life-threatening arrhythmias. This report describes the first such case with serological evidence of recent Coxsackievirus B3 infection.

Case report

A previously healthy 12-year-old girl was admitted to hospital on 6 September 1976 with a 4-day history of sore throat, shivering, malaise, occasional vomiting, and with 'fainting attacks' on the 2 preceding days. On examination she was apyrexial, blood pressure 100/90 mmHg, pulse 80 beats/min. There were no cardiac murmurs, no pericardial rub, and no signs of cardiac failure. She had slight neck stiffness. A few hours after admission she had a cardiac arrest. After resuscitation, chest *x*-ray showed marked cardiomegaly. Subsequently, multifocal ventricular ectopic beats occurred which were resistant to lignocaine and to mexiletine.

During the next 36 hours ventricular fibrillation occurred on 14 occasions. Each time it was terminated by DC defibrillation (100-200 J) but was followed by severe bradycardia ($\leqslant$30 beats/min) which only partially responded to atropine 0·6 mg IV. On 9 September she became breathless and was oliguric, blood urea had risen to 23 mmol/l (138·6 mg/100 ml; see Table), and chest *x*-ray showed pulmonary oedema. At this stage she was transferred to this hospital. On examination blood pressure was 100/60 mmHg, pulse 60 beats/min (see ECG, Fig.), jugular venous pressure raised 4 cm, liver enlarged 3 cm. A bipolar endocardial pacing catheter was introduced via the left antecubital fossa and 'on demand' pacing was started at 90 beats/min. She was given digoxin 0·125 mg/day, phenytoin 50 mg three times a day, prednisolone 20 mg per day, and frusemide 40 mg IV immediately. A urinary catheter was inserted.

Table *Results of clinical chemistry*

Investigation (normal max)	*Date: September* 6	7	9	10	14	23
Blood urea (2·7–7·5 mmol/l)	13·2	18·6	23·0	11·2	9·5	3·9
Alananine aminotransferase (100–500 nkat/l)	2562	4987	7185			
Aspartate aminotransferase (75–400 nkat/l)	8000	7935	6000			
Glutamin oxalotransferase (9–19 IU/l)				226	68	10
Glutamic phosphorotransferase (5–17 IU/l)				687	207	16
Creatine kinase (0–117 IU/l)				133	11	12
Lactic dehydrogenase (115–457 IU/l)				1368	691	217
Alkaline phosphatase (25–103 IU/l)		75			100	117
Bilirubin (0–22 μmol/l)		7			13	18

Conversion: SI to traditional units—Blood urea: 1 mmol/l $\approx$ 6·02 mg/100 ml. Bilirubin: 1 μmol/l $\approx$ 0·06 mg/100 ml.

Progress. Ventricular fibrillation did not recur and urinary output was satisfactory. By 14 September lung fields were radiologically normal and cardiac size was decreasing. On the 17th, paroxysmal atrial fibrillation occurred with a ventricular rate of 130/min. Phenytoin was stopped and Kinidin durules 1 bd started. Pulse rate was easily controlled and she was gradually mobilised. The pacemaker electrode was removed on the 23rd. She was discharged on 4 October taking digoxin 0·125 mg/day and Kinidin durules 1 bd. Digoxin was stopped one month later. She was seen in the outpatient clinic on 3 December; there were no abnormal physical signs but she complained of breathlessness, for example, when climbing stairs. Chest *x*-ray was normal; ECG is shown in the Fig.

Investigations. Results of clinical chemistry are shown in the Table and ECGs in the Fig. Hb 12 g/dl; white cell count 15·6 $\times 10^9$/l. ESR 4 mm in the first hour (Westergren). On 20 October echocardiography showed a small pericardial effusion.

Viral studies. On 9 September Coxsackievirus B3 neutralisation titre 32, and on the 21st, 512. Attempts to isolate the virus from stools and throat washings were unsuccessful.