requiring resuscitation. These hospitals would work in close co-operation with the special units—chest, plastic, neurosurgical—in the region, which should if possible be grouped together, and thus provide complete accident cover.

It has been suggested that the treatment of head injuries could be planned by opening up numerous "sub-areas" of neurosurgery, where a lone neurosurgeon with limited facilities would work. This suggestion still ignores the simple fact that accidents go to the nearest hospital, and, although it is essential that in the future this scheme should be streamlined further and hospitals be selected for major accident work, any organization must take into account the treatment of patients in hospitals to which they are first admitted. Specialist services, and particularly those of neurosurgery, are far better concentrated, and it is wiser to have several neurosurgeons in one centre with full ancillary services, providing between them an efficient 24-hour service, than to have individuals distributed throughout a region.

The peripheral hospitals, however, are not concerned only with the treatment and disposal of cases of major head injury. How essential is it to admit patients with minor degrees of concussion to hospital? And can beds be saved in this way? Our experience is that this work, although undramatic, is most valuable. To admit patients with minor degrees of concussion allows one to diagnose early the case that develops such a complication as an extradural clot, and this period of observation can also be properly used to determine the pattern and speed of the patient's subsequent progress. A short rehabilitation programme during the two or three days' observation is a major factor in the prevention of the post-concussional syndrome. It has for some years been our policy to follow up nearly all our patients until they have become symptom-free and have returned to work. It has been found that the number of cases of post-concussional syndrome arising among the in-patients is very small, but many more examples are seen in patients who, because of the mildness of their injury, have not been admitted to hospital. Quite apart from the surgical potentialities, the admission of these patients, short of wasting beds, results in a very considerable economic saving to the country in weeks off work and helps to restore them to health more quickly.

Finally, one should reiterate that head injuries constitute only one facet of the accident services of this country. A good deal has been heard lately of the need to plan for occasional national disasters such as railway accidents, but a more urgent problem is the treatment of the hundreds of daily accidents on our roads and in our homes.

Both problems can be solved if in every region an efficient accident service is planned, based on area hospitals, each having an accident service that serves also a group of smaller peripheral hospitals with which they work in concert. Special units would be grouped around a main accident service at the centre. Once such a system is established, it can deal with most situations. In the Radcliffe we had experience of this at the time of the Didcot rail disaster in 1955. It was found quite possible to provide additional beds in the hospital quickly by sending some patients home and transferring others. Thus it required no alteration in the system of the accident service to deal with the 54 casualties admitted. A week-end road disaster in which 10 or more people are involved and arrive in hospital together is unhappily a common occurrence in all major hospitals, and a plan that deals with this quickly and efficiently can be readily expanded. Better hospital facilities are required, but much can be done by harnessing our existing resources. A good deal of what is required at this stage can come from organization within the profession.

THE CARDIAC MURMUR IN RELATION TO SYMPTOMS IN THE NEWBORN

BY

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During the first few hours of life a cardiac murmur is often heard in the newborn baby. The murmur increases in intensity towards the end of systole, sometimes continuing through the second sound. It has been attributed to blood flow through a persistent ductus arteriosus (Burnard, 1958). This paper describes the relationship between the presence of this murmur, a previous history of asphyxia, and the maturity and the clinical condition of the baby. Both asphyxia and dyspnoea were associated with a higher incidence of the murmur, particularly in premature infants.

Materials and Methods

Murmurs were identified by auscultation and recorded with a phonocardiograph as previously described (Burnard, 1958). Asphyxia was regarded as being present when there was a delay of three minutes or more in establishing normal breathing after birth, or, in a few instances, when there was severe foetal distress (even though normal breathing began within three minutes of delivery).

The mature babies were nursed in cots at a room temperature of approximately 70° F. (21.1° C.); extra warmth was applied in a bassinet heated with electric light bulbs. Blood pressure was measured with a 1-in. (2.5-cm.) cuff, both by palpation and the flush method. The premature babies were nursed in incubators that were maintained at 85° F. (29.5° C.) for babies over 4½ lb. (2,040 g.) and 90° F. (32.2° C.) for those weighing less, and were supplied with oxygen in varying concentrations.

Dyspnoea here refers to rates above 40 per minute in full-term and 50 in premature babies who have been at rest for a minute or longer, as well as to grunting laboured breathing, even though the rate is no higher than 40. A respiratory rate of 40 a minute is normal for healthy infants (Smith, 1951); and Miller and Conklin (1955) point out that the same is true after the first hour in premature babies when they are well. There is a subjective element in the clinical assessment of dyspnoea, and it was found difficult to draw a sharp line between respiratory embarrassment and normal breathing as a baby recovered.

Mature and Healthy Babies

In 69 normal babies who were nursed in their cots, without extra warmth, the crescendo systolic murmur developed in 4 (6%) between two and six hours after birth. Twenty-two babies were kept warm as described

above, and eight (36%) of these developed the murmur within the same time—a significantly higher proportion. One of the latter, at a rectal temperature of 98° F. (36.7° C.), became very breathless; this baby improved over a three-hour period as its temperature fell to 94° F. (34.4° C.) and during this time the murmur disappeared. The temperatures reached by the babies in warmed cots varied considerably, partly because after the babies had spent a variable period in the labour room wrapped in a towel their temperatures were very different on arrival in the nursery. The distribution of rectal temperatures in warmed babies who had the murmur fell about the mean for all warmed babies (Fig. 1). As some reached temperatures well above the mean without developing the murmur, factors other than the temperature must also have determined the presence or absence of this crescendo systolic murmur.

Warming increased the incidence of all types of murmur in the newborn baby. Most warmed babies developed murmurs in the second to fourth hour after birth, a few in the fourth to eighth hour, and none after

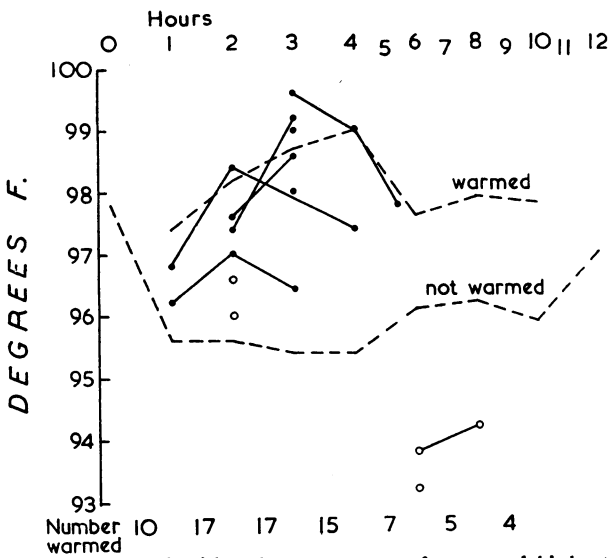

FIG. 1.—Effect of raising the temperature after normal birth at term. Broken lines: upper, mean temperature of 22 babies when warmed; lower, mean temperature of 69 babies, unwarmed and with no murmur. ●=Crescendo systolic murmur in 8 warmed babies. O=Crescendo systolic murmur in 4 unwarmed babies. Duration of murmur less than one hour except when continuous line joins successive observations on the same baby.

that (Table I). The ejection murmurs of early systole have not been included in the table. The pansystolic and diastolic murmurs have already been described (Burnard, 1958), and, while an exact basis for comparison is not available, these murmurs were certainly less common in unwarmed babies.

After asphyxia at birth the incidence of the crescendo systolic murmur in 41 babies was 25 (61%). They were nursed in their cots without extra warmth; though some were slightly cyanosed no oxygen was considered necessary in any of them, and all survived. Fig. 2 shows the mean temperatures at different intervals from birth in these babies. The difference between the means is significant ($P<0.05$) from the first to the sixth hour. Thus a fall in temperature favours the disappearance of the murmur when it develops after asphyxia at birth.

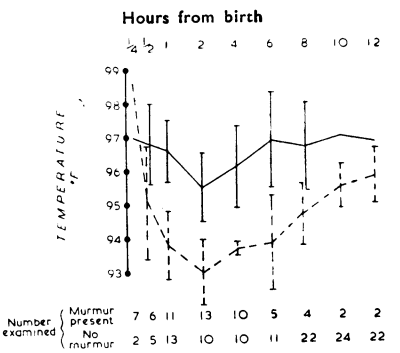

FIG. 2.—Relation of asphyxia and temperature to murmur. Mean temperatures at different intervals from birth in 41 mature babies who had suffered from asphyxia, and in 25 of whom a murmur was detected. Continuous line: murmur present. Broken line: murmur absent. The vertical lines are the 95% confidence limits of each mean.

Although the babies to whom these observations relate seemed to be well in natural conditions, apart from the brief administration of oxygen in the labour room, severe dyspnoea developed in a few babies after asphyxia at birth (see under next heading). Closer observation was therefore made on the breathing of 20 babies in whom there had been a three-minute delay before the establishment of normal respiration. In all of them the respiratory rate was found to exceed 40 a minute at the age of an hour, rising to between 60 and 90 in some, with grunting. The crescendo systolic murmur was heard in 15 (75%). None became seriously ill, but four were considered to need oxygen for 48 to 72 hours, and dyspnoea subsided slowly. The rest were well within 24 hours, and all survived.

Changes in blood pressure were not consistently related to the development or disappearance of the murmur in an individual baby, nor were there significant differences in pressure according to its presence or absence in different babies. It is, however, a difficult matter to measure the pressure accurately. The effect of late tying of the cord was examined in 10 babies who were kept for 10 minutes below the level of their mother's abdomen, but in only one did the murmur develop.

Breathless Full-term Babies

Mature babies sometimes die after a period of respiratory embarrassment, and then present the same necropsy findings of pulmonary haemorrhage, hyaline membrane formation, and, less commonly, frank pulmonary oedema, that are found in prematures. Observations have been made in 16 severely ill full-term babies. Physical signs changed with time and clinical condition, and murmurs came and went. Of these 16 babies, 11 (69%) had a crescendo systolic or a continuous murmur (Table II). Although all but three of them were severely asphyxiated at birth and were treated for this, the clinical illness in eight was an unexpected event

TABLE I.—*Total Incidence of Murmurs (Excluding Early or Mid-systolic) in 22 Warmed Babies*

Hours:	0–1	–2	–3	–4	–6	–8	–10
No. of babies warmed	10	17	17	15	7	5	4
Range of temperature °F.	95·2–101	96–102	96·6–102	96·4–102	95·2–99	96·2–101	97–99·2
Range of temperature °C.	35·1–38·3	35·6–38·9	35·9–38·9	35·8–38·9	35·1–37·2	35·7–38·3	36·1–37·3
Crescendo murmur (Fig. 1)	0	2	4	7	2	0	0
Diastolic murmur	0	6	5	1	0	1	0
Pansystolic ,,	0	1	3	2	1	0	0
Total murmurs		9	12	10	3	1	0

after a period during which they had been regarded as well. Thus one baby was sent to the nursery after an hour's treatment with oxygen in the labour room and was found in a collapsed state with a little blood at the mouth nine hours later.

TABLE II.—*Findings in 16 Breathless, Severely Ill Full-term Babies*

	Crescendo Systolic Murmur	Pansystolic Murmur	Continuous Murmur	No Murmur	Post-mortem Findings*
Died 24–72 hours (10 babies)	4	1	1	4	Atelectasis with congestion and areas of haemorrhage (3) Massive pulmonary haemorrhage (4) Pneumonia with gross congestion or haemorrhage (3)
Died 5–8 days (2 babies)			2		Cardiac hypertrophy,† pulmonary haemorrhage
Survived (4 babies)	2		2		

* Number of babies in parentheses.
† Heart weights 24 and 26 g.; birth weights, 5 lb. 14 oz. and 6 lb. (2,665 and 2,720 g.); expected heart weight for size of infant, 18 g. (Coppoletta and Wolbach, 1933.)

Dyspnoea was obvious in these babies and intense in several of them. The upper part of the sternum bulged forwards. Physical signs were absent from the lungs at first, and the air entry was good, but crepitations developed terminally. The heart rate was rapid and the second sound was usually loud; when it was soft there was no murmur except for a very short ejection noise. The liver was distinctly enlarged in five babies.

There was thus a close association between birth asphyxia and severe or fatal illness in 16 babies, with dyspnoea as the outstanding symptom and the crescendo systolic murmur as a concomitant finding.

Premature Babies

The most common murmur was the rough crescendo in late systole, which was seldom detected beyond the second sound. When it was heard after the second sound (Fig. 3) the resulting murmur was sometimes continuous on auscultation, although phonocardiographic records in the premature infant never showed a murmur continuous throughout the cardiac cycle like that of the adult persistent ductus arteriosus. Early and mid-systolic murmurs were also detected, but they are not discussed in this paper.

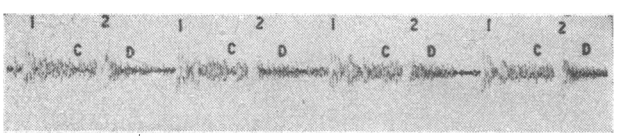

FIG. 3.—Premature, 8 hours of age. The murmur sounded continuous through both systole and diastole to the ear, but tracing fails to show it at the end of diastole. C=Crescendo systolic. D=Diastolic; 1 and 2, first and second heart sounds.

Incidence and Clinical Features

The crescendo systolic murmur was heard in 55 of 96 consecutive premature babies (57%). It was thus more often detected than in a similar series of full-term babies, in whom there was a total incidence of only 37%, although examination was more frequent (Burnard, 1958); it also lasted a longer time in premature babies. Among surviving premature babies it was found in 29 of 58 (50%), and among those who died in 26 of 38 (68%). The higher mortality in babies with the murmur is displayed when incidence is plotted according to age (Figs. 4 and 5). The incidence in surviving babies is probably in excess of that to be expected in a truly representative sample of prematures, since the distribution by weight of the babies studied (Table III) shows that the 4½–5½ lb. (2,040–2,495 g.) group was relatively small, as many babies of this weight were admitted direct to the general nursery, needing no special care.

Some features of the crescendo systolic murmur were more obvious in premature than in mature babies. Thus during the expiratory grunt that is common in dyspnoeic infants, but before actual phonation occurred, the murmur sometimes disappeared (Fig. 6). The effect of crying was the same, in that the murmur sometimes disappeared during the expiratory effort before the cry. In other babies the murmur was still heard before grunting or crying. Breathing also influenced the murmur. For example, in Fig. 7 the murmur is present after each inspiration, but absent during the second cardiac cycle after the last breath.

The murmur was hard to distinguish from the breath sounds during severe dyspnoea, though it could usually be made out by careful listening. Fig. 8 illustrates the murmur in a very breathless baby and also shows the relationship to respiration just mentioned, the amplitude being smaller after expiration.

The relationship to the supply of oxygen that has been noted by others (Zetterström, 1955) was clearly observed in two babies, the murmur returning with a reduction in the concentration and disappearing within a few hours of an increase.

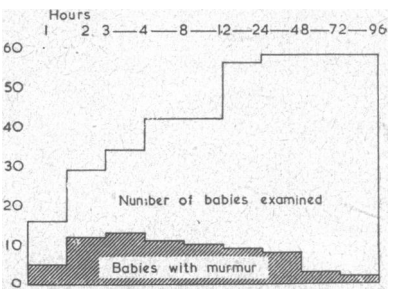

FIG. 4.—Incidence of murmur by age in 58 surviving babies.

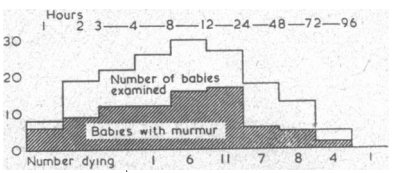

FIG. 5.—Incidence of murmur by age in 38 premature babies who died.

Among the babies who died a murmur which had disappeared as they improved not uncommonly returned as their condition became worse. It was also detected for the first time in some babies as they deteriorated.

In premature babies who suffered from dyspnoea the heart rate was raised and the second sound exaggerated and slapping in quality during the early phase of the illness, but there was no correlation between the presence of the crescendo systolic murmur and the quality of the second heart sound.

When the crescendo systolic murmur was first heard the lips and mouth were often somewhat dusky, and the hands and feet were a little blue for varying lengths of

TABLE III.—*Weights of Premature Babies*

Weight lb.	Weight g.	Living	Died	Total
<2½	<1,135	3	17	20
2½–	1,135–	13	13	26
3½–	1,587–	28	6	34
4½–	2,040–	12	1	13
>5½	2,495	2	1	3
Total		58	38	96

time. The murmur lasted as long as four days, however, in babies who were pink all the time. When it reappeared in two babies as the oxygen concentration was lowered, their symptomatic change was an aggravation of dyspnoea rather than a change of colour.

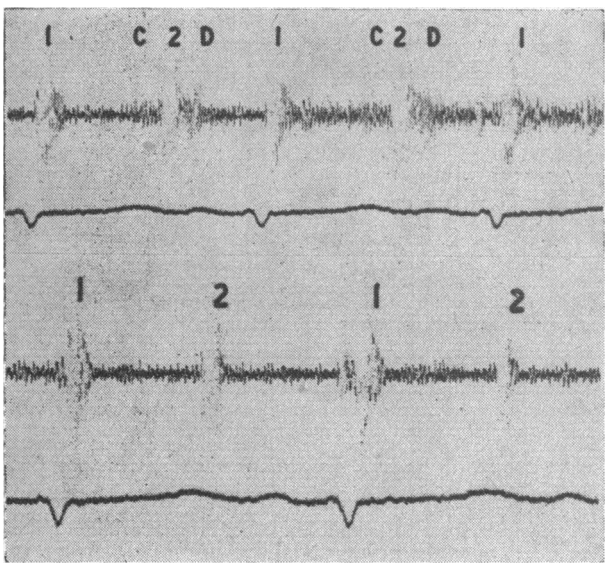

FIG. 6.—Relation of murmur to expiration with glottis closed. Premature, 36 hours of age. Symbols as in Fig. 3. E.C.G., lead I. Above: Murmur heard in late systole and early diastole. Below: Murmur absent during expiration with glottis closed, just before noisy grunt.

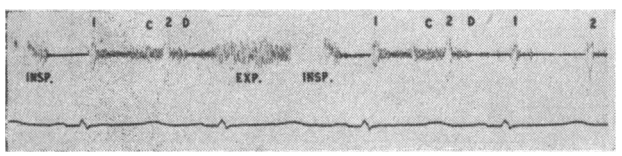

FIG. 7.—Relation to inspiration. Premature, 40 hours of age. Murmur heard intermittently. Tracing shows that it is well-marked in first heart beat after inspiration and absent in the second. Symbols as in Fig. 3. E.C.G., lead II.

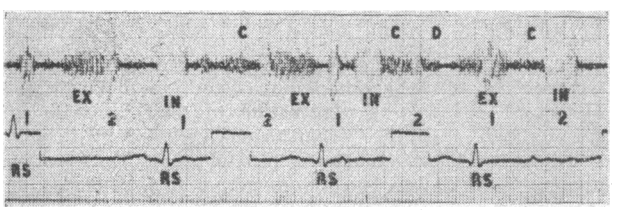

FIG. 8.—Premature, 18 hours of age. Crescendo systolic murmur could be heard despite noisy tachypnoea (respirations, 110; heart, 150 a minute). Tracing shows the murmur, which goes through the second sound in the third heart beat. Its amplitude is greater after inspiration (second and third beats) than after expiration (fourth beat). Symbols as in Fig. 3. E.C.G., lead II; each jump in the E.C.G. baseline signals inspiration in the phonocardiogram (IN).

Relation to Symptoms and Temperature

The murmur was frequently present in premature babies who were breathless, whether or not they had suffered asphyxia for three minutes or more at birth (Table IV).

TABLE IV.—*Incidence of the Crescendo Systolic Murmur in Premature Babies Related to Birth Asphyxia and to Clinical State*

	Asphyxia	No Asphyxia
Total babies	44	52
No. with dyspnoea	42	42
,, ,, ,, and murmur	28	27
Proportion of dyspnoeic babies with murmur	67%	64%
No. with no dyspnoea and no murmur	2	10
Proportion of total with murmur	64%	54%

Fig. 9 shows the relation between the murmur, temperature, and dyspnoea in 53 surviving babies. During the first 24 hours after birth many babies had a rectal temperature of less than 95° F. (35° C.), and yet a large proportion were breathless and had a murmur. The duration of this murmur was often brief, even though dyspnoea persisted, and in some breathless babies no murmur was found despite quite frequent examination. The fall in temperature may have accounted for the diminishing incidence of the murmur with the passage of time, or for its absence in some of these babies, as in mature babies with lower temperatures after birth asphyxia.

The effect of a rise in temperature on the incidence of the murmur in this group of surviving premature babies can be illustrated by the following example. Twins weighing 4 lb. and 3 lb. 10 oz. (1,815 and 1,645 g.) were placed in the same incubator at the age of 4 hours, their temperatures being 92 and 91.6° F. (33.3

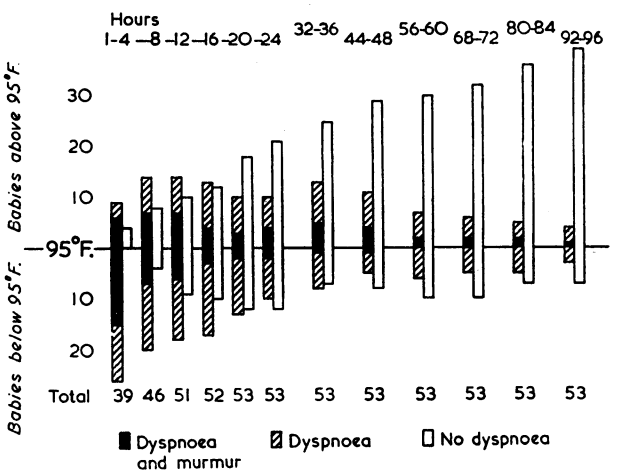

FIG. 9.—Incidence of murmur and dyspnoea related to rise in temperature and passage of time in 53 surviving premature babies. (The selection of 95° (35° C.) was arbitrary.)

and 33.1° C.) and respiratory rates 70 and 50 a minute. Three hours later the temperature of both babies was 98° F. (36.7° C.) and the respiratory rates 100 and 72. The first baby had no murmur; in the second there was a crescendo systolic murmur continuous through the second sound (Fig. 3). Ten hours later the second baby's temperature was 92° F. (33.3° C.), breathing was peaceful and remained so, and the murmur had gone. the first baby's temperature did not fall below 95.4° F. (35.2° C.), a crescendo systolic murmur developed, and there was considerable dyspnoea for 36 hours.

Body size had an important influence on temperature, and it was the smaller babies who had a temperature below 95° F. (35° C.) at the close of the observations (Fig. 9). In the first 24 hours, on the other hand, even the large and comparatively mature babies were mostly below 95° F. (35° C.) if they were breathless, and smaller ones were above 95° F. (35° C.) if they were well. In extremely dyspnoeic babies the work of breathing may have contributed to the exceptional instances of a relatively high temperature in the first 24 hours in the presence of dyspnoea. If this allowance is made it seems possible that symptomatic improvement, and not just the passage of time, determined the rise in temperature.

Discussion

The reasons for believing that the crescendo systolic murmur, whether continuous through the second sound or not, arises from left-to-right flow through a partly contracted ductus arteriosus have been given previously (Burnard, 1958). This murmur rarely occurred in normal babies (6% unwarmed; 36% warmed), but was often heard after birth asphyxia in mature babies (61%), and in premature babies it was found whether they had been asphyxiated at birth (64%) or not (54%).

Zetterström (1955) has suggested that the association of the murmur with asphyxia at birth means that asphyxia prevents the ductus closing. However, animal experiments suggest that under certain circumstances severe asphyxia, as well as oxygen, may actually cause the ductus to constrict (Born *et al.*, 1956). Moreover, Zetterström's hypothesis implies that the ductus arteriosus normally closes rapidly and completely in newborn babies. We now know that it may in fact remain partly open for many hours or days after birth, both in animals (Amoroso *et al.*, 1958) and in babies (James and Rowe, 1957; Adams and Lind, 1957).

Another possibility should therefore be considered—namely, that the presence or absence of a crescendo systolic murmur in the newborn baby is dependent upon the relative vascular resistances of the greater and lesser circulations. If the systemic vascular resistance increases, then, other things being equal, blood flow through the ductus arteriosus will increase: the faster the flow, the greater the likelihood of detecting a murmur. This idea is supported by the fluctuation in the murmur that was found in some premature babies in whom it faded on expiratory straining (Fig. 6), and was accentuated during inspiration (Figs. 7 and 8). Flow through the ductus arteriosus should diminish during straining with the glottis closed, as the rise in intrathoracic pressure tends to equalize pulmonary arterial and aortic pressures, while it should be increased by the greater ventricular output during inspiration.

The production of the murmur by extra warmth and its disappearance with the natural fall of temperature after asphyxia at birth (Burnard and Cross, 1958) are complementary observations, and would also fit the hypothesis that there may be changes in blood flow through the great vessels, consequent on temperature changes, which influence the genesis of the murmur. It appears that the most likely time for warming to show this effect is after the first hour (Table I), which is consistent with the need for a reduction in diameter of the ductus before a murmur is heard (Dawes *et al.*, 1955). The respiratory distress, as well as the effect on the murmur, that warming three babies produced should also be noted.

In relation to dyspnoea the clinical associations of the murmur were as follows. In the premature babies who recovered, dyspnoea was worse when the murmur was present. The murmur never occurred unless the clinical condition of the baby was serious. It was not always found in premature babies who died (Fig. 5), however, possibly because their temperatures were always low. The same generalizations were true of mature babies. Apart from the 16 who were gravely ill, however, dyspnoea in them was not so severe as in the prematures, and was indeed often overlooked until the 20 mature babies who had suffered asphyxia were watched more carefully.

Whether an increased blood flow from aorta to pulmonary artery through the ductus arteriosus affects the baby favourably or otherwise has yet to be shown. In lambs suffering asphyxia the increased shunt appears to be beneficial to the animal in permitting re-circulation through the lungs, with a rise in oxygen saturation of arterial blood, even though the greater amount of blood in the lungs diminishes pulmonary compliance and ventilation (Dawes, *et al.*, 1955). In asphyxiated babies respiratory difficulty is an outstanding phenomenon. The murmur is an indication that disturbed haemodynamic conditions are present when dyspnoea is evident.

Summary

In mature babies the incidence of the murmur that probably arises from left-to-right flow through the ductus arteriosus had a direct relationship to rectal temperature and to asphyxia at birth.

In premature babies the incidence was approximately the same as in mature babies after birth asphyxia. Prematurity of itself must have accounted for the similarity.

The murmur was an accompaniment of dyspnoea in premature babies and in severely breathless mature babies. Dyspnoea outlasted the murmur in premature babies. In some prematures the murmur returned if their clinical condition relapsed.

When mature babies had suffered birth asphyxia a raised respiratory rate was evident though they appeared healthy. The murmur was present in 75%.

A reduction in rectal temperature seemed to result in amelioration of dyspnoea and disappearance of the murmur. Conversely, warming three babies shortly after birth brought about dyspnoea as well as the murmur.

The suggestion is made that the murmur probably means an increase in flow through the ductus arteriosus above the rate present after normal birth.

The means whereby asphyxia causes an increase in flow through the ductus, and whether this in itself is harmful or not, are at present unknown.

I thank the obstetricians in charge of the maternity departments of the Paddington General and St. Mary's Hospitals for allowing observation of their patients; the nursing staff of the two departments; the Nuffield Institute of Medical Research for the loan of the phonocardiograph; and Dr. G. S. Dawes for help in preparing the text.

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