CHANGES IN HEART SIZE IN THE DYSPNOEIC NEWBORN BABY

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In a previous paper (Burnard, 1959) the relationship was discussed between symptoms and the crescendo systolic murmur in the newborn baby. Changes in heart size are considered in the present paper, and an explanation is offered of the similarities in symptomatology and necropsy findings between the full-term baby who has suffered asphyxia at birth and the premature baby. Particular importance is attached to the association between murmur, breathlessness, and cardiac enlargement.

Method and Material

Forty-nine premature babies were radiographed on one or more occasions in the first seven days of life. Dyspnoea was taken to be present when there was an expiratory grunt or a rate of breathing above 50 a minute. The examinations were made according to the presence of this symptom and its progress, rather than at fixed intervals. Thirty-six of the babies survived and 13 died. All the babies received oxygen, the apparently healthy at concentrations of 30–35% for at least 12 hours, and the ill ones at higher concentrations for periods according to their need as judged clinically.

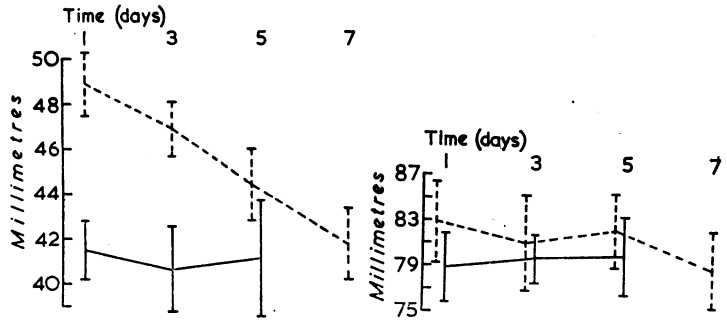

FIG. 1 FIG. 2

FIG. 1.—Mean transverse diameter of heart in 29 babies (weight between 3 lb. 8 oz. and 4 lb. 12 oz.; 1,585 and 2,155 g.). The vertical lines are the 95% confidence limits of each mean. The numbers of *x*-ray films taken in the dyspnoeic groups on days 1, 3, 5, and 7 were 13, 12, 12, and 12, and in the non-dyspnoeic group 12, 11, 9, and 3. ----, Dyspnoea (16 babies, mean weight 4 lb. 6 oz.; 1,985 g.). ——, No dyspnoea (13 babies, mean weight 4 lb. 3 cz.; 1,900 g.). FIG. 2.—Mean internal diameter of thorax (same babies and conventions as in Fig. 1).

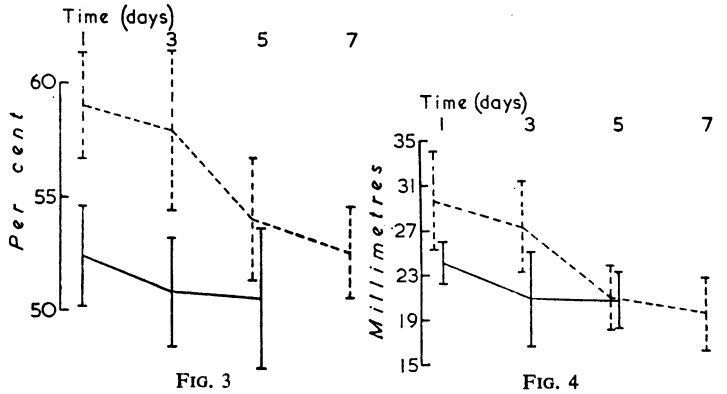

FIG. 3 FIG. 4

FIG. 3.—Mean cardiothoracic ratios expressed as a percentage (same babies and conventions as in Fig. 1). FIG. 4.—Mean width of mediastinum at third rib (same babies and conventions as in Fig. 1).

Radiographs were taken with the baby supine, the lower trunk being protected, at a tube-distance of 48 in. (122 cm.) and an exposure time of 1/50 second. Oxygen was supplied during the procedure and time was allowed for the infant to settle in this position, with the help of a dummy when necessary. In healthy babies the picture was taken either during momentary apnoea or on a shallow inspiration. In dyspnoeic babies a brief apnoeic pause could sometimes be chosen too, but more commonly the radiograph was taken on inspiration. A single film was usually enough; if it showed rotation of the thorax a second was taken. The number of exposures for each baby was between two and seven. I was present at all examinations.

From the radiographs measurements were made as follows. The maximum transverse diameter of the heart (T.D.) was measured at right angles to the thorax, care being taken not to confuse the right border of the heart with the thymic margin, which often bulged further to the right but at a higher level. The internal diameter of the thorax (I.D.) was measured at the right diaphragmatic dome (Martin and Friedell, 1952; Caffey, 1956) and the width of the mediastinal shadow opposite the third rib posteriorly. The cardiothoracic ratio was calculated at T.D./I.D. and expressed as a percentage. The variability in the means of successive measurements by one observer on the same set of films was found to be 0.5 mm. for T.D., 3 mm. for I.D., and 1.4 mm. for mediastinal width. These did not affect the significant differences in these measurements between groups of babies when subdivided according to their symptoms (Figs. 1–4). A check was made in which films were presented at random to a second observer. The means of his measurements when plotted in the same way showed the same significant differences.

The same measurements were made from the radiographs of 11 seriously breathless mature babies.

The majority of necropsies were performed by Dr. R. R. Wilson at the Paddington General Hospital, and I am indebted to him for permission to quote from the reports.

Results in Premature Babies

A study of the serial changes in 29 surviving babies of similar size and maturity was made. Sixteen babies (mean weight 4 lb. 6 oz., S.E. 2.0 oz.; 1,985 g., S.E. 57 g.) who were dyspnoeic were compared with 13 (mean weight 4 lb. 3 oz., S.E. 2.2 oz.; 1,900 g., S.E. 62 g.) in whom respiratory distress was not observed, although in a few of the latter the nursing staff had reported slight grunting in the first hour after birth. The periods of gestation were from 33 to 37 weeks and weights between 3 lb. 8 oz. and 4 lb. 12 oz. (1,585 and 2,155 g.). From a consideration of the measurements illustrated in Figs. 1–4 the following conclusions were drawn.

First, the mean transverse diameter of the heart was significantly greater in the babies who were breathless, and declined as they recovered. The best agreement between two observers was obtained with this

measurement. The duration of the symptom varied; the majority of dyspnoeic babies were indistinguishable by the third day from the controls, who were never breathless, and all were well by the fifth day.

Secondly, there was no significant change in the mean internal diameter of the thorax with the passage of time in either group, nor was there a difference between the groups (Fig. 2). The mean cardiothoracic ratio would therefore be a valid estimate of heart size in the two groups (Fig. 3). The differences between the mean ratios were of the same order as for transverse diameter, being significant to the third day. The wider limits of confidence for the ratio were due to the greater range of values for the denominator, reflecting the variability in shape of the lower thorax in different babies, with crowding of the ribs in some and splaying in others.

Thirdly, there was a significant decline in mediastinal width in dyspnoeic babies between the first and the fifth day. Between the two groups the difference in mediastinal width had not the same order of significance. This measurement was the most difficult to make consistently from baby to baby, since the edge of the shadow was often ill-defined.

The changes are illustrated in Fig. 5, a typical series of radiographs in a dyspnoeic baby, and in the outline diagrams (Figs. 6 and 7).

A comparison of absolute measurements was thus made between 29 premature babies of approximately the same size who lived but could be distinguished by their symptoms. Since in them there was no significant change in mean thoracic diameter during the period of observation, nor a difference between means according to whether the babies were well or ill, the cardiothoracic ratio was used as a basis for comparing heart size in the whole group of 49 premature babies. Of the additional 20 babies, 16 weighed less than 3½ lb. (1,585 g.), and 9 of these died; the cardiothoracic ratios of 4 babies who weighed between 3½ and 5½ lb. (1,585 and 2,495 g.), but who died are also given. When the ratios are plotted (Fig. 8) the tendency towards greater heart size in ill babies is apparent.

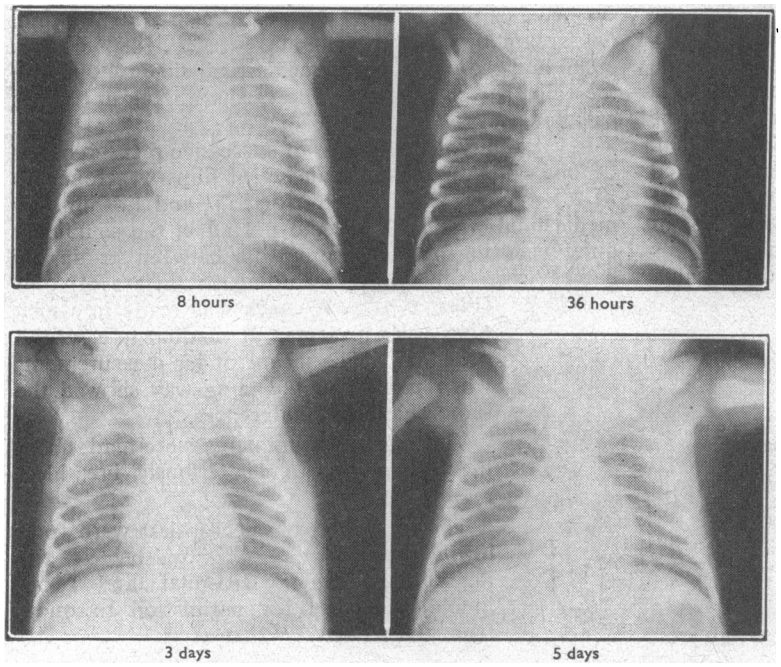

FIG. 5.—Baby H., 2 lb. 13 oz. (1,275 g.), showing diminishing heart size and mediastinal width during recovery from severe dyspnoea. Radiographs taken at 8 hours, 36 hours, 3 days, and 5 days.

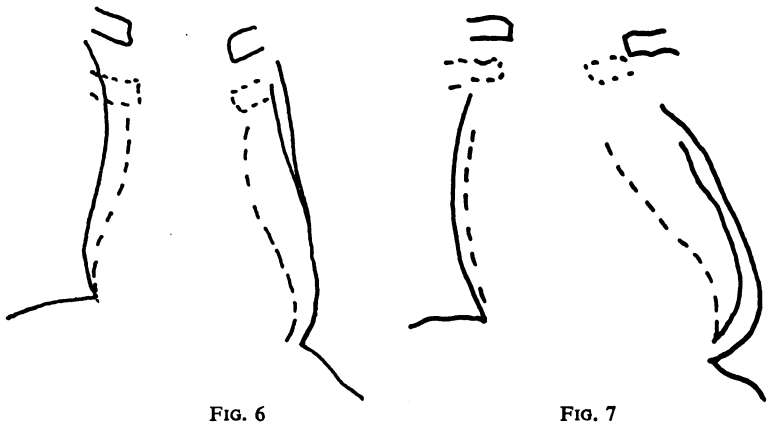

FIG. 6

FIG. 7

FIG. 6.—Outline of heart and mediastinum at 1 hour and 15 hours (continuous lines), and at 72 hours (broken line), during subsiding dyspnoea. Premature, weight 4½ lb. (2,040 g.). FIG. 7.—Outline of heart and mediastinum at 6 hours and 36 hours (continuous lines), and at 6 days (broken line), during subsiding dyspnoea. Premature, weight 3½ lb. (1,585 g.).

Certain factors that may bear on aetiology are given in Table I. Abnormal labour refers to complications of pregnancy that terminated in premature labour or caesarean section. Asphyxia means three minutes or more apnoea after birth, and the murmur was the crescendo systolic murmur that has already been described. Under the abbreviation "H.M." are given the numbers with a radiologist's diagnosis of "hyaline membrane disease"; in an additional three babies who died an equivocal opinion was held. Hyaline material in air passages, with capillary congestion, was reported at the necropsies of eight, fibrin and polymorphs in one, and gross pulmonary haemorrhage in one. The hearts were all normal in structure. Only one death occurred after the first 72 hours, and that baby, with a cardiothoracic ratio of 65 on the third day and 70 on the fifth, had lungs which were reported at necropsy as containing oedema, fibrin, and haemorrhage.

Observations were made on heart rate in relation to respiratory difficulty. After birth the rate was high, between 160 and 200 a minute. In some of the larger babies it remained comparatively fast. A characteristic finding at the height of respiratory distress, and within a few hours of the onset of the illness, was a fixed rate between 110 and 120 a minute that did not vary if the baby cried or was roused to physical activity. The rhythm had a tick-tack quality suggesting prolongation of systole. The babies' general condition was very grave at this stage. When they survived, early signs of improvement were a rise in the rate to 140 or 150 a minute, or a lability of the rate as shown by variations in it with exertion. Pulmonary crepitations which had developed then disappeared. The

TABLE I.—*Relations Between Dyspnoea in Premature Babies and Birth History, Crescendo Systolic Murmur, and Radiographic Changes in Lungs (See Text)*

	No. of Babies	Weight		Labour		Asphyxia	Murmur	"H.M."
		<3¼ lb. (1,585 g.)	3¼–5½ lb. (1,585–2,495 g.)	Normal	Abnormal			
Lived—No dyspnoea	16	3	13	11	5	5	1	0
Lived—Dyspnoea	20	4	16	11	9	13	15	4
Died—dyspnoea	13	9	4	7	6	7	9	6

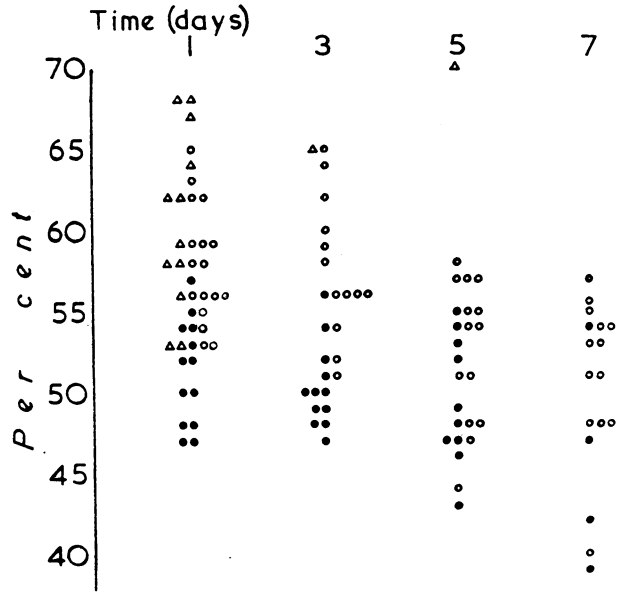

FIG. 8.—Cardiothoracic ratios in 49 babies (see text). The numbers of *x*-ray films taken in the dyspnoeic group, in addition to those illustrated in Figs. 1–4, were 16, 2, 3, and 2 on days 1, 3, 5, and 7. Twelve babies died between days 1 and 3, and 1 on day 6. Survived: ●, No dyspnoea. ○, Dyspnoea. Died: △, Dyspnoea.

treatment given was an adequate supply of oxygen and calories, and in some instances digitalis; it was of particular interest to notice the rising heart rate in the presence of full digitalization as bodily vigour improved and dyspnoea lessened.

Heart Size in Mature Dyspnoeic Babies

Of the 16 seriously ill full-term babies on whom observations were described in the preceding paper, 5 were radiographed once or more. The measurements, in these as well as in an additional 6 babies whose clinical condition was similar, are given in Table II. Since no control examinations were made of healthy mature babies the data of Martin and Friedell (1952) have been used for comparison. In the dyspnoeic babies who comprise the present material the transverse diameter and cardiothoracic ratio were significantly greater than in Martin and Friedell's healthy babies on the first day and closely comparable to their abnormal babies. The decline by the fifth day was steep, being similarly comparable, and quite different from the normal.

It is of interest to record the fact that in five mature babies who were adversely affected during an exchange transfusion the same symptomatology of dyspnoea and crescendo systolic murmur developed: no radiographs were taken.

TABLE II.—*Mean Transverse Diameters and Cardiothoracic Ratios (±S.E.) in Normal and Dyspnoeic Mature Babies*

	T.D. (mm.)		C.T. Ratio (%)	
	Day 1	Day 5	Day 1	Day 5
Dyspnoeic (present series)	57·9±1·1	50·3±2·7	60·3±1·5	52·0±1·6
,, (Martin and Friedell)	57·8±1·3	52·3±1·3	63·4±0·8	57·8±1·0
Normal (ibid.)	50·2±0·7	47·8±0·9	55·6±0·7	52·6±0·8

Present series: 12 radiographs on 11 babies in first 30 hours, 4 radiographs on 4 babies on day 5.
Means for normal and dyspnoeic babies taken from Martin and Friedell (1952) and standard errors calculated from their data. (They classify dyspnoeic babies as "cyanotic"; their second examination, listed under "day 3–6," is placed in the above table on day 5.)

Possible Errors in Method

In textbooks of diagnostic radiology the great variability in the appearances and measurements of the cardiac outline in infancy is stressed. It is worth remarking that a practical reason for such caution is lest the appearances be mistaken for a congenital deformity.

The present work was concerned with differences according to symptomatic state rather than individual diagnosis. The same person was present to ensure a uniform technique. The posture was the same in all babies. The bizarre effects of crying and of forced expiration were wholly excluded, the infants all being quiet when radiographs were taken.

Moderate inspiration might not have been the exact equivalent of a brief apnoeic pause so far as the position of the diaphragm was concerned (Bakwin and Bakwin, 1935), and pictures must also have been taken at different phases of the cardiac cycle. Despite these possible limitations in the technique, sufficient babies were examined to show differences in relation to symptomatology. The differences were beyond the range of observer error.

Present Findings

The first object of this paper has been to reopen the question of cardiac enlargement in the dyspnoeic newborn. Miller and Wilson (1943), dealing with babies of diabetic mothers, attributed their illness to heart failure, and considered the enlarged cardiac silhouette to be a valid criterion. In the present material the heart size in dyspnoeic premature babies was significantly greater for the first three days than in apparently healthy controls, and the decline in size by the fifth day was significant. In full-term babies heart size was significantly greater in breathless babies in the first 30 hours than in normal data cited from the literature.

Interpretation of the cardiac silhouette itself could not go beyond the statement that the chambers showed a general enlargement. The measurement of mediastinal widening at the third rib may have included the thymic shadow, the right lobe of which could sometimes be identified. However, the rapid reduction in width illustrated in Figs. 5–7, and the narrow pedicle that was frequently seen in babies when they were not breathless, indicate that the thymic breadth in the radiograph follows passively the form of distensible mediastinal structures, presumably the great veins. Further investigation is required to determine the physical conditions responsible for enlargement of the venous shadow in the presence of respiratory distress. There is known to be a greater negative intrathoracic pressure then (Karlberg *et al.*, 1954). The supine position tends to broaden the mediastinum (Caffey, 1956), but this would have been a constant effect and does not explain the rapid diminution in serial studies.

The thorax was well expanded in these babies, no significant difference being evident between dyspnoeic and healthy, nor in the dyspnoeic babies as they improved. This is noteworthy, despite the fairly wide range of the observations (Fig. 2), because it agrees with Steiner's (1954) finding that the lungs of premature babies appear very well inflated in radiographs taken within a few minutes of birth.

By calculation from radiographs in two dimensions, Kjellberg *et al.* (1954) demonstrated a significant reduction in heart size from the first to the second day in healthy mature babies; they were unable to reach a conclusion about prematures. It is therefore not surprising that in the healthy premature babies (Fig. 1) no significant decline appeared from consideration of films in the single antero-posterior dimension. Their purpose was to serve as controls for the dyspnoeic babies.

Necropsy Findings

In babies who die soon after birth the changes in the lungs have usually been thought to be the most important. The weight of the heart, however, was greater than normal in babies of diabetic mothers (Miller and Wilson, 1943; Winter and Gellis, 1954) and in two mature babies in whom death after severe birth asphyxia was deferred for a number of days (Burnard, 1959, Table II), during which time dyspnoea was continuously present.

The atelectasis which is found is generally accepted by pathologists as secondary or "resorption" in type (Potter, 1952; Claireaux, 1953). Radiographic changes in the lung fields have consequently been interpreted as the first evidence of alveolar collapse (Donald and Steiner, 1953; Ellis and Nadelhaft, 1957).

Dyspnoea

Uncertainty of the origins of dyspnoea in the newborn is reflected in terminology. Thus we have congestive pulmonary failure (Potter, 1953), pulmonary syndrome of the newborn (Bound *et al.*, 1956), and hyaline membrane syndrome (Parmelee, 1952; Arey and Dent, 1953), or disease (Berfenstam *et al.*, 1958). Premature babies have usually been considered separately from those born at term. The absence of good correlation in clinical, radiographic, and necropsy findings has often been emphasized.

Dyspnoeic babies, both premature and full-term, can now, however, be shown to have in common, and at an early stage in their illness, enlargement of the heart, a characteristic murmur, and anomalous behaviour of the heart rate. The thorax and lungs are well expanded in early radiographs (Steiner, 1954; Lendrum, 1955). The abnormal findings vary from baby to baby, depending partly on the passage of time and also on the frequency of examination. The association is, however, common enough, having been identified in more than half the ill babies described in this and the preceding paper, to indicate cardiovascular behaviour of a different order from those who are well. It is therefore suggested that cardiac insufficiency is the basis of the disorder, and that the increase in lung markings described as the first identifiable change by Bauman and Nadelhaft (1958) and the more familiar fine granularity (Feinberg and Goldberg, 1957) represent the development of vascular congestion and oedema. No doubt atelectasis may follow.

The pattern of pathological change in the lungs can be very well interpreted on the basis of vascular congestion. Capillary congestion (Potter, 1952) and oedema (Claireaux, 1958) accompany resorption atelectasis in the premature. Alveolar haemorrhage confined to small areas of the lung or on a massive scale is found in both premature and mature babies (Claireaux, 1958). The hyaline membrane which lines alveolar ducts has a variable incidence in the premature (Latham *et al.*, 1955; Briggs and Hogg, 1958). The same post-mortem appearance has been described in mature babies (Blystad *et al.*, 1951; Potter, 1953). Hyaline is now known to be fibrin (Gitlin, 1957), and to arise endogenously (Duran-Jorda *et al.*, 1956). Alveolar haemorrhage and hyaline may coexist in the same specimen (Ahvenainen, 1958; Briggs and Hogg, 1958). The overlapping incidence in these items of descriptive morphology suggests a common origin rather than separate disease entities.

The occurrence of infection would be expected to complicate the histological picture. At the same time attention may be drawn to the finding that infiltration with polymorphs and macrophages follows hyaline accumulation (Van Breemen *et al.*, 1957).

Radiographic Interpretation

From the evidence that has been cited it seems unlikely that there is failure of primary expansion of the lungs when a baby who has evinced signs of life goes on to develop dyspnoea, or that obstruction of major air passages is important in its causation. Aspiration of infected uterine contents, or of meconium on a considerable scale (Emery, 1956), may give rise to a disseminated pneumonia with recognizable features (Peterson and Pendleton, 1955). In my experience such instances are not common and can usually be correlated with evidence from the birth history. The relationship of events at birth to the fine mottling that is characteristic of the dyspnoeic premature baby is a different matter. Although aspiration of amniotic liquor has been widely held responsible for asphyxial symptoms after birth and for changes in the lung, its place would seem less important now that the lung fields have been shown clear in the radiograph for a time preceding later changes (Steiner, 1954; Bauman and Nadelhaft, 1958), as well as for the reasons discussed above.

Obstruction to the circulation in unexpanded lungs was proposed to explain cardiac enlargement after birth asphyxia (Martin and Friedell, 1952; Lind and Wegelius, 1954). This, too, becomes untenable once the equivocal evidence for atelectasis as the primary event is recognized.

Aetiology

If mature and premature babies are now considered together, there are some indications of the manner of the cardiac enlargement and of its meaning. First, it was associated with dyspnoea and the occurrence, for a greater or less time, of the crescendo systolic murmur (Table I); the murmur was sometimes pansystolic or continuous (Burnard, 1959, Tables I and II). Enlargement was present from the earliest time at which radiographs were taken, and declined with improvement in dyspnoea. In the premature babies asphyxia at birth was common but not invariable (Table I); in the baby born at term it is likely that, with the possible exception of overload during exchange transfusion, difficult

labour or apnoea after birth was a necessary precursor (Martin and Friedell, 1952; Burnard, 1959).

Secondly, Cross and Dawes (1959) have shown in the newborn lamb that the cardiac output is virtually fixed, and does not rise in response to stresses that in older animals evoke a fourfold increase. The observations described on the heart rate of dyspnoeic premature babies are compatible with the idea that the heart at this time has difficulty in meeting additional stress. The falling off in rate despite the large demands made by increased work in distressed babies (Karlberg *et al.*, 1954) was a comparatively early feature of the illness.

Mayer (1953) has pointed out that the signs by which distress is estimated in the foetus during delivery are those of a pre-terminal condition. It is therefore not unreasonable to suppose that after the stresses of an asphyxial birth the heart may be unable to cope with the circulatory requirements of the newborn state. With the development of pulmonary congestion and interference with ventilation the asphyxial state would, of course, be perpetuated.

The fact that in the premature baby symptoms develop after normal labour and birth remains to be explained. The identity of symptomatology between premature and asphyxiated full-term babies has been emphasized throughout this and the preceding paper. A deficiency of respiration in the widest sense may therefore be inferred in the premature baby, resulting in an asphyxial condition sufficient to set off the same process as does asphyxial birth in the full-term baby. The lapse of time before the development of symptoms in the premature baby after normal birth is in favour of such an explanation. The handicaps of the premature baby may include deficiencies in respiratory enzyme function (Himwich, 1953), greater initial stiffness of the lung than in the full-term baby (Born *et al.*, 1955), and, using an anatomical yardstick, reduction in the area of lung available for gas exchange (Farber and Wilson, 1933; Norris *et al.*, 1941). According to the ideas which have been developed in this paper these are not the factors responsible for the typical respiratory distress of the premature baby so much as the pulmonary vascular congestion to which they give rise through the medium of heart failure.

Differences in the clinical course of premature babies of similar maturity may depend partly on birth conditions and partly on the early supply of oxygen both to minimize an inherent tendency to hypoxia and to assist uptake in the lung should congestion have already developed. The oxygen that is given in a routine manner after birth has thus to be taken into account before ill premature babies can be said to make a spontaneous recovery (Bauman and Nadelhaft, 1958); in the present series all the healthy premature babies were given oxygen in the first 24 hours.

Mechanism

Insufficiency of the heart must occur first on the left to bring about pulmonary congestion. Lendrum (1955) suggested left ventricular failure; mechanisms as yet unknown may be at work. Bonham Carter (1957) suggested that a raised venous pressure was perhaps beneficial. This could not be so if the heart were failing. The suggestion was supported by the views of Jäykkä (1957) that capillary filling from the arterial side is a prerequisite for alveolar expansion. Jäykkä (1958) has admitted the difficulty of explaining morbid capillary congestion on this basis.

The short-lived crescendo systolic murmur was related to the signs of cardiac failure as a clinical phenomenon. It was attributed to increased flow through the ductus arteriosus (Burnard, 1959), and this would certainly increase the demands on the left ventricle from the greater output required. Further investigation is needed to clarify the haemodynamic conditions underlying these signs.

Management

Hypoxia is the one element of the asphyxial state which can be directly relieved. If the above hypothesis is correct, then the earlier and more adequate the supply of oxygen the sooner will the effects of pulmonary congestion be limited. Retinal damage follows an abnormally high oxygen tension in the arterial blood in babies below 36 weeks in gestation. The problem of the babies in question, however, is to attain a normal tension. Blystad (1956) produced evidence of arterial desaturation in dyspnoeic babies, and there is urgent need for more information on these interrelationships. As an indication for early treatment in seriously ill babies dyspnoea rather than cyanosis has proved the better guide. The latter may be absent despite arterial desaturation (James and Rowe, 1957) and is always difficult to estimate (Swyer, 1958). The spells of apnoea and cyanosis which may punctuate their later course, when the babies seem relatively well in the intervals, are not under consideration here.

The posture most suited to the dyspnoea of pulmonary congestion is with the head raised. When there is a need in babies to deal with accumulated pharyngeal secretion the optimum position may seem in doubt. It is, however, worth pointing out that asphyxia is a cause of these excessive secretions (Zetterström, 1955). Consequently, adoption of the head-down position with the idea of assisting drainage might in fact aggravate the basic disorder. The eight mature babies who unexpectedly deteriorated after birth asphyxia (Burnard, 1959) had all been nursed during the latent interval with the head low, as is frequently conventional if there has been difficulty in establishing breathing at birth.

The administration of digitalis is a logical step if the myocardium is failing. Without adequate oxygen, however, digitalization, in dosage recommended by Nadas (1947), did not preserve the life of some dyspnoeic but viable babies. Since the work of breathing is greatly increased, an early supply of calories is desirable. The fall in neonatal temperature appeared to play some part in the natural history both of dyspnoea and of the crescendo systolic murmur, as described in the preceding paper; beyond the observations that a limited fall was not harmful, and that rapid warming on occasion precipitated symptoms, no conclusions have been reached on the optimal temperature for continuing care.

Summary

The heart was larger in the radiographs of premature babies suffering dyspnoea than in similar babies without dyspnoea. The heart size diminished as the symptoms improved.

In mature babies suffering dyspnoea the heart size was greater than in normal babies cited from the literature.

The association for a greater or less time of the crescendo systolic murmur with cardiac enlargement was

noted. The association between murmur and dyspnoea had been shown previously.

The triad of murmur, dyspnoea, and enlargement occurred in full-term babies after birth asphyxia and in premature babies whether they had been asphyxiated at birth or not. Both enlargement and the murmur were early phenomena in babies with dyspnoea.

This association, coupled with the apparent paradox of a slow heart rate in the presence of severe dyspnoea, and related to evidence of a limited ability in the newborn to increase cardiac output, led to the suggestion that cardiac insufficiency was present from an early stage.

Necropsy findings in the lung could be interpreted on the basis of vascular congestion. The early radiological appearances could mean that congestion and oedema were developing.

The suggestion was therefore made that insufficiency on the left side of the heart was the cause of pulmonary congestion and the subsequent symptoms. Asphyxia during birth brought this about in the full-term baby. In the premature child born without asphyxia a defective respiratory mechanism had to be presupposed.

The implications of these views for management were mentioned.

I am indebted to the obstetric staffs of the Paddington General and St. Mary's Hospitals for permission to make observations, and to the midwives and radiographers of both hospitals for help in carrying them out; to Dr. E. Rohan Williams and Dr. R. E. Lawrence for the use of their radiological reports; to Dr. David Symers for the random check in measurements; and to Dr. R. Lightwood, who is in charge of the Paediatric Unit, St. Mary's Hospital Medical School.

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ECHO-ENCEPHALOGRAPHY*

ULTRASONIC RAYS IN DIAGNOSTIC RADIOLOGY

BY

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Galton described his ultrasonic whistle in 1883, but, apart from signalling to dogs, no practical use seems to have been made of ultrasonic rays until the war of 1914–18. At that time pulses of ultrasonic rays were transmitted through water as a means of detecting and locating submarines. Since then it has become standard practice to employ ultrasonic echoes for measuring the ocean depth and to detect icebergs.

Since the second world war the same principles have been applied in the industrial field, particularly for the detection of flaws in large masses of metal. Dussik (1948) published a paper on the medical use of ultrasonic rays, but using continuous radiation. The patient's head was immersed in a water-tank and the absorption of transmitted radiation was measured. This had the obvious disadvantage of the tank and the danger of continuous radiation.

The earliest published work using pulses and echoes in medicine is that of Wild (1950). French, Wild, and Neal (1951) used apparatus developed by the United States Navy for service purposes and called the ultrasonic trainer. Owing to the high frequency of 15 million cycles per second, this apparatus could penetrate only 3 cm. into soft tissue. In consequence it was used mainly to differentiate carcinoma from benign conditions of the breast. Its use on the brain was restricted to patients at craniotomy where the probe could be applied direct to the cortex.

In early 1954 the physics department of the Royal Marsden Hospital demonstrated at the Physical Society's Exhibition a modification of the Kelvin and Hughes flaw-detector, mark IIB, which gave echoes from front to back of the intact skull, using pulses of frequency $1\frac{1}{4}$ Mc. per second.

In 1956 Professor Leksell, of Lund, published his results with the Kelvin and Hughes flaw-detector mark V, which he had used since 1953. Professor Donald, of Glasgow, has been using a specially modified flaw-detector for the differentiation of large abdominal tumours. He

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