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Perinatal cardiac arrest

Quality of the survivors

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Steiner, H., and Neligan, G. (1975). *Archives of Disease in Childhood*, **50**, 696. **Perinatal cardiac arrest: quality of the survivors.** Twenty-two consecutive survivors of perinatal cardiac arrest have been followed to a mean age of 4¼ years, using methods of neurological and developmental assessment appropriate to their ages. 4 showed evidence of gross, diffuse brain-damage (2 of these died before the age of 3 years). These were the only 4 survivors of the first month of life who took more than 30 minutes to establish regular, active respiration after their heartbeat had been restored. The arrest in these cases had occurred during or within 15 minutes of delivery, and followed antepartum haemorrhage, breech delivery, or prolapsed cord. The remaining 18 were free of any evidence of brain damage. In the majority of these the arrest had occurred during shoulder dystocia or exchange transfusion, or was unexplained; the heartbeat had been restored within 5 minutes in most cases, and regular, active respiration had been established within 30 minutes thereafter in all cases.

To succeed in bringing a fellow human being back from the dead is a most exciting and gratifying clinical experience. But the immediate sense of achievement is often rapidly succeeded by a vague fear that the quality of the life which has been restored may have been irrevocably impaired by brain damage due either to the period of circulatory arrest which presumably accompanied the clinical cardiac arrest, or to the primary cause of the cardiac arrest itself. This emergency is particularly likely to occur during or soon after birth, and there are two good reasons for believing that the chances of completely successful treatment should be better in the newborn baby than in later life. Firstly, the mechanisms which enable the term fetus and newborn of many mammalian species to survive a much more prolonged period of asphyxia than the adult include the ability of the central nervous system to survive a more prolonged period of circulatory arrest (Dawes, 1968). Secondly, the chest wall of the newborn is so soft and pliable that external cardiac massage, when indicated, can be performed promptly and efficiently (Moya *et al.*, 1962). Furthermore, nearly all cases of perinatal cardiac arrest in this country now occur in a clinical context where optimal methods of resuscitation should be immediately available.

Sporadic reports of cases of successful external cardiac massage in the neonatal period, with some information concerning the subsequent progress of the babies concerned, have appeared during the past 12 years (Moya *et al.*, 1962; Surks and Ladner, 1962; Gallagher and Neligan, 1962; Mathews, Avery, and Jude, 1963; Hey and Kelly, 1968). But we have seen no report of any series of cases which makes it possible to assess the prognosis realistically.

We have followed 22 babies who had been successfully resuscitated after perinatal cardiac arrest, and had survived the neonatal period, to an age when their neurological and developmental status could be satisfactorily assessed. The findings and the conclusions we have drawn from them form the basis of this report.

Material and methods

During the 9 years between mid-1961 and mid-1970 a serious attempt was made to resuscitate 39 babies born in this hospital whose heart beat could not be heard by at least one experienced observer either immediately after birth (14 cases of fresh stillbirth), or within the first 15 minutes (11 cases of cardiac arrest in the labour

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ward), or later during the first week of life (14 cases of early neonatal cardiac arrest). All the babies were free of external evidence of congenital malformation, and most were at or beyond term. The immediate results of the resuscitation attempts are summarized in Table I. Those recorded as showing 'no response' did not regain an audible and effective heartbeat; those who died in the first week had regained an effective heartbeat and active respirations and death was attributed to adrenal or intracranial haemorrhage in 3 cases, with evidence of severe kidney damage in some cases. *Treatment* of the cardiac arrest was the responsibility of members of the hospital's paediatric staff, one or more of whom was present or immediately available in every case, and was carried out in accordance with an agreed procedure which did not vary in essentials, but did vary in detail, during the 9-year period.

Essentials. (1) Tracheal intubation using a moulded rubber tube of 2·5, 3·0 or 3·5 mm external diameter, as soon as clinical cardiac arrest was confirmed (in some of the cardiac arrests in the labour ward the baby had already been intubated before arrest occurred). Positive pressure ventilation using pure oxygen at a pressure of up to 30 cm water, by occluding the Y-piece of water manometer circuit of Resuscitaire trolley (or prototype) for a few puffs. Then (2) Sternal compression, towards vertebral column, at a rate of about 60/min (pausing every 10–15 s to allow a single inflation of the lungs, and every minute or so to allow auscultation of the heart). Cardiac massage was continued for 15 minutes routinely before accepting defeat. In 8 of the ultimate survivors no other form of treatment (see details below) was used: 7 are completely normal.

Details. (1) Intracardiac injections were given by needle passed through an intercostal space to the left of the sternum into a cardiac cavity (checked by withdrawal of blood) if the heartbeat did not return within about 4 minutes. During the earlier years, 0·5–1·0 ml nikethamide was given, but latterly a similar volume of 8·4% sodium bicarbonate solution has been used instead.

(2) The umbilical vein was catheterized as soon as possible, depending upon the availability of an assistant, for administration of a manitenance infusion of 10% dextrose solution, in many cases. From about 1965 onwards the infusion has been preceded by injection of a bolus of 5–10 ml 8·4% sodium bicarbonate solution (the exact volume depending upon an estimate of the baby's weight, where this is not accurately known, and the duration of the arrest). The risks of this procedure appear to be justified by the severity of the problem (Behrman, 1966). After an interval the baby's blood has been checked to determine if the anticipated metabolic acidosis has been adequately corrected.

Follow-up of the 22 survivors of the first month of life has been the responsibility of one of us (H.S.). Information about their subsequent progress is available for all 22 children, 18 (82%) as a result of one or more personal examinations, to a mean age of 4¼ years (range 5–98 m) at the time of the latest examination. The examination included the Denver Developmental Screening test (Frankenburg and Dodds, 1967); a standard neurological examination; specific examination of the motor system described by Milani-Comparetti and Gidoni (1967); the Stycar test of hearing (Sheridan, 1958) and vision (Sheridan, 1960); the Draw-a Man IQ (Goodenough, 1926). All these tests were used as the children reached appropriate ages and 5 of the eldest were also assessed by a clinical psychologist, using the Wechsler Intelligence Scale for children (1949) or the Preschool and Primary Scale of Intelligence (1967). We feel that these methods of assessment should have detected all moderate and severe handicaps, but clearly a more prolonged period of observation will be necessary to look for minor neurological handicaps and learning difficulties (Steiner, 1974). In the 4 cases (18%) who were not accessible for these examinations, information was obtained from paediatricians who looked after them up to the time of their early death (2 cases), or in a residential institution (1 case), or following adoption (1 case).

Results

In Table II we have summarized the findings at follow-up, together with the clinical data, for the individual babies. They are not in chronological order but in groups corresponding to the clinical subdivisions used for presenting the immediate results, in terms of survival, in Table I. Table II

TABLE I

Immediate results of 39 consecutive attempts to resuscitate cases of perinatal cardiac arrest in Princess Mary Maternity Hospital, 1961–1970

Timing of cardiac arrest	Group	Total no.	Outcome		
			No response	Died first week	Survived
During delivery	A	14	4	4	6
First 15 min	B	11	–	3	8
During exchange transfusion	C	11	1	3	7
During 1st week, unexplained	D	3	1	1	1
Total no.		39	6 (15%)	11 (28%)	22 (56%)

TABLI

Summary of clinical data concerning 22 consecutive survivor

Case no.	Obstetric problems	Birthweight (kg) and/or gestational age (w)	Cardiac arrest (min)
Group A: fresh stillbirths			
1	Shoulder dystocia	3600 g	3+
2	Breech	3420 g 42 w	¾+
3	Prolapsed cord; caesarean section; fetal distress	43 w	6+
4	Shoulder dystocia	4650 g	3+
5	Shoulder dystocia	4700 g	15+
6	Shoulder dystocia	5050 g	10+
Group B: CA in labour ward			
7	Primigravida aged 44 yr; caesarean section		½
8	Shoulder dystocia	3800 g	2½
9	Breech; fetal distress; APH		¼
10	Severe fetal distress; failed forceps; caesarean section		1
11		2050 g 42 w	1
12	Severe APH	43 w	6½
13	IUT × 1; caesarean section for failed induction	33 w	3
14	Severe APH; fetal distress	1960 g 38 w	10
Group C: CA during exchange transfusion (ET)			
15	Cord Hb 108%		1
16	IUT × 4; cord Hb 85%		2
17	Cord Hb 87%		2
18	Severe APH; cord Hb 91%	1230 g	1
19	Cord Hb 70%	34 w	10
20	Cord Hb 43%	35 w	10
21	Cord Hb 78%	35 w	6
Group D: CA in first day: unexplained			
22	Severe APH; caesarean section; asphyxia (IPPV for 8 min)		2

*Following return of heart-beat.

APH, antepartum haemorrhage; IUT, intrauterine transfusion; NDN, normal developmer and neurology; WISC, Wechsler Intelligence Scale for Children; WPPSI, Wechsler Preschoo and Primary Scale of Intelligence; IPPV, intermittent positive pressure ventilation; Gooc enough Draw-a-Man Test.

gives a rough picture of the clinical background and outcome in each baby, and permits simple groupings which may be of interest. We hope to tabulate some such groupings from which we believe that practical conclusions can be drawn.

Probable causes of cardiac arrest. We have attempted to determine the cause in each case in terms of the known antecedent clinical factors; and then to determine the prognosis for the survivors following each type of cause. We have summarized these results in Table III. Groups A and B have been combined because, whether the actual cardiac arrest occurred just before or just after delivery, the ultimate causes are likely to be the same (and Table II shows that there is about the same risk of severe brain damage in the 2 groups). The cause of cardiac arrest during exchange

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f first month, after perinatal cardiac arrest (CA)

Regular respirations (min)	Neonatal complications	Follow-up	
		Age (yr)	findings
17	—	6½	NDN
46	Apathy; fits; irritability	4	Quadriplegic; mentally subnormal
64	Irritability; fits	4	" "
7	Irritability; fits	4	NDN; WPPSI 78
15	Irritability; fits; tetany	2½	NDN
4	Irritability	1	NDN
1	—	8	NDN; WISC 103
2½	Erb's palsy	5	NDN; WPPSI 109
15	—	5	NDN
28	Irritable 12 h	3½	NDN
6	Irritable 24 h; fits; tetany	3	NDN
32	Fits; tetany	2	Died; quadriplegic defective
3	ET×3	1½	NDN
32+	Apathy; irritability; fits	½	Died; quadriplegic, defective
1	ET×1	8	NDN; WISC 106
3	ET×3	7	NDN
2	ET×2	6	NDN; Goodenough 95
28	ET×2	5	NDN; WPPSI 84
10	ET×4	3½	NDN; deafness
12	ET×2	3	NDN
4	ET×2	½	NDN
3	CA at 13 h	5	NDN; Goodenough 95

transfusion in the 7 babies who survived is not known with certainity. We have attributed it to '? biochemical or physical' causes because these were not particularly severe cases of haemolytic disease (in contrast to the 4 cases shown in Table I who failed to survive), and we suspect that the heart was affected either by reversible biochemical abnormalities (e.g. a high level of potassium or citrate), or a fall in temperature in the baby's blood (before we adopted the routine practice of passing the donor blood through a warming coil in a thermostatically controlled water bath). The harmful effects of using cold blood were described by Hey, Kohlinsky, and O'Connell (1963).

It is clear that all the instances of severe handicap followed a perinatal cardiac arrest, but that all 5 babies in whom this was attributed to shoulder dystocia escaped unscathed, showing normal neuro-

TABLE III

Probable causes of cardiac arrest, and quality of survivors

Group*	Probable cause of cardiac arrest	No. of survivors	Findings at follow-up		
			Normal	Quadriplegic	Deaf
A+B	Shoulder dystocia	5	5	–	–
	Antepartum haemorrhage	3	1	2	–
	Breech deliver	2	1	1	–
	Prolapsed cord	1	–	1	–
	Severe fetal distress	1	1	–	–
	None known	2	2	–	–
C	?Biochemical or physical	7	6	–	1
D	None known	1	1	–	–
Total		22	17 (77%)	4 (18%)	1 (5%)

*See Table I.

logy and development (at a mean age of 45 months). This finding is consistent with some published case reports (Epple and Sutherland, 1959; Leake, 1959; Mathews *et al.*, 1963), and with the expectation that the chances of recovery should be better after a 'catastrophic event' than when cardiac arrest has been preceded by a more or less prolonged period of progressive anoxia amd respiratory or metabolic acidosis (*Lancet*, 1972). The relatively high risk of brain damage in the children whose arrest was attributed to antepartum haemorrhage, breech delivery, and prolapsed cord is also consistent with this expectation.

The only neurological deficit found in the 8 cases where cardiac arrest occurred later than the first 15 minutes after delivery was deafness (high-tone) in a child whose arrest occurred during his first exchange transfusion, at the age of 45 minutes, and whose unconjugated plasma bilirubin level later rose to a level of 22 mg/100 ml at the age of 38 hours.

Duration of cardiac arrest. This was judged by the time which elapsed until the heartbeat could be heard. In group A cases this must have been appreciably longer than the time recorded, which was the elapsed time between completion of delivery and return of the heartbeat. In several instances where the heartbeat returned within 1 or 2 minutes of starting external massage it was noted as being initially very strong, so that it could easily be felt, or seen, as well as heard. The findings summarized in Table IV do suggest, as might be expected, that longer duration of arrest is associated with a higher incidence of brain damage (this was only too obvious in 3 of the 8 survivors of cardiac arrest which lasted more than 5 minutes). However, this relation does not appear to be strong enough to act as the basis of a clinical policy of withholding treatment, since even following an arrest of 10–15 minutes' duration, the majority of the survivors developed quite normally, and only one of the 5 was brain-damaged.

Delay in establishing regular active respiration. After the return of the heartbeat (which resumably indicates that an effective circulation has been re-established), this could indicate relatively severe brain damage, specifically affecting the respiratory centre in the midbrain, but possibly more widespread. The relation between the duration of this delay, and the risk of severe, irreversible, and widespread brain damage are summarized in Table V. It is clear that this relation is very strong, and the fact that no baby who took more

TABLE IV

Duration of cardiac arrest, and quality of survivors

Duration of cardiac arrest (min)	No. of survivors	Findings at follow-up		
		Normal	Quadriplegic	Deaf
1 or less	7	6	1	–
2–5	7	7	–	–
6–9	3	1	2	–
10–15	5	3	1	1

TABLE V

Delay in establishing regular, active respiration, and quality of survivors

Delay in regular aspiration after return of heartbeat (min)	No. of survivors	Findings at follow-up		
		Normal	Quadriplegic	Deaf
1–15	20	19	–	1
16–28	3	3	–	–
32 or more	4	–	4	–

than 30 minutes to start regular breathing after the return of the heartbeat later developed normally could be regarded as a contraindication to further active resuscitation when this point has been reached. The dividing line may appear to be narrow, but the degree of handicap in the 4 survivors was so gross that we have felt justified in incorporating this contraindication into our routine policy of management, so answering the difficult question posed by Cockburn (1971), of 'when to stop ventilating the infant who fails to sustain regular respirations but who maintains a good colour and regular heartbeat . . .'. A further finding which strengthens the validity of this concept, that a delay of more than 30 minutes in establishing regular, active respiration after the return of the heartbeat is a grave prognostic sign, is that 5 of the 7 cases in groups A and B who failed to survive the first week of life had also experienced such a delay (range 42–69 min).

Discussion

The routine availability of more effective methods of resuscitation could be expected to save the lives of some newborn babies who would otherwise have died simply because they failed to make the dramatic physiological adjustments which are necessary after the birth of any mammal. This expectation appears to have been justified in the case of intermittent positive pressure ventilation (Neligan, Prudham, and Steiner, 1974,) which is the first requirement in treating cardiac arrest, since this is almost always preceded, and always accompanied, by respiratory arrest. The addition of an efficient and safe method of dealing with cardiac arrest, by closed-chest massage, presumably further increased the chance of saving lives, even though the absolute numbers involved might be quite small. In fact our 22 survivors of cardiac arrest are derived from a total population of 20 793 babies who were born in our hospital during the period covered by this report (a rate of little more than 1/1000 live births), and for much of this period there was a policy of selective admission of mothers with obstetric problems, and for all of it of mothers suffering from Rhesus isoimmunization. The latter fact partly explains the large number of 7 cases in which cardiac arrest occurred during exchange transfusion. This procedure was performed some 2009 times in our hospital during the 9 years.

On the other hand, the effectiveness of the more efficient methods does appear to introduce an increased risk that brain-damaged individuals will survive, who would otherwise have died quickly. The vague fear of this possibility was mentioned in our opening paragraph. We hope that our findings enable us to be more precise, not only about the magnitude and characteristics of this risk, but also the more positive aspects of the quality of the survivors.

It is true that the overall results summarized in Table III show that nearly 20% of the survivors were brain-damaged, and that the severity of the disability in the 4 children concerned was such that the early deaths of 2 of them could only be regarded as merciful, both for them and for their families. But the other side of the coin is represented by the fact that we found no evidence of any degree of brain damage, or of any attributable handicap, in the other 18 children (if we accept that the case of high-tone deafness was more likely attributable to the bilirubin level of 22 mg/100 ml). Clearly this optimistic finding needs to be confirmed by more prolonged and more searching assessment of these children at later ages, but the nature of the evidence we have already obtained seems to us to justify a positive approach to the treatment of this emergency in general, combined with a specific search for a clinical policy which will enable us to avoid survival of the brain-damaged minority.

Certain antecedent factors appear to have a good prognosis: in particular, the abrupt physiological or physical insults which presumably cause cardiac arrest in cases of shoulder dystocia or during exchange transfusion. Even when the antecedent factor is less favourable, as in the case of antepartum, haemorrhage, prolapsed cord, or breech delivery, it is worthwhile acting as rapidly and efficiently as

possible, once the occurrence of cardiac arrest has been established by an experienced observer. If the heartbeat is back within 5 minutes, and the baby is breathing regularly and spontaneously within 30 minutes thereafter, the prognosis still appears to be uniformly good. In our experience at least, the only situation which need cause real concern about the possibility of permanent brain damage, is when active, regular respiration has not been established within 30 minutes after the return of the heartbeat (which is unlikely unless the cardiac arrest has lasted more than 5 minutes). In that case, as we mentioned in our results, we no longer regard it as justifiable to persist with resuscitative measures, in view of the uniformly very bad prognosis. Of 9 such babies who responded initially, 5 died before the age of 1 week and the 4 who survived the neonatal period were all quadriplegic and grossly mentally defective. We believe it is right to describe this fate as 'worse than death' (*Lancet*, 1974).

Our findings appear to confirm that the treatment of cardiac arrest is particularly worthwhile during the early neonatal period, and that by using certain simple clinical criteria for withholding further treatment in a few cases, it may be possible to reduce the proportion of brain-damaged survivors to well below the figure of nearly 20% which we have reported.

We are grateful to many colleagues on the paediatric staff of this hospital whose prompt action and accurate records have made this report possible.

References

Behrman, R. E. (1966). Alkali therapy in the delivery room. *Journal of Pediatrics*, **69**, 173.

Cockburn, F. (1971). Resuscitation of the newborn. *British Journal of Anaesthesia*, **43**, 886.

Dawes, G. S. (1968). Birth asphyxia, resuscitation, and brain damage. *Fetal and Neonatal Physiology*, p. 141. Year Book Medical Publishers, Chicago.

Epple, H. H., and Sutherland, J. M. (1959). Resuscitation of stillborn infants. *Obstetrics and Gynecology*, **13**, 259.

Frankenburg, W. K., and Dodds, J. B. (1967). The Denver developmental screening test. *Journal of Pediatrics*, **71**, 181.

Gallagher, B., and Neligan, G. (1962). Resuscitation of stillborn infant. *British Medical Journal*, **1**, 400.

Goodenough, F. L. (1926). *Measurement of Intelligence by Drawings*. World Books, New York.

Hey, E., and Kelly, J. (1968). Gaseous exchange during endotracheal ventilation for asphyxia at birth. *Journal of Obstetrics and Gynaecology of the British Commonwealth*, **75**, 414.

Hey, E., Kohlinsky, S., and O'Connell, B. (1963). Heat losses from babies during exchange transfusion. *Lancet*, **1**, 335.

Lancet (1972). (Leading article.) Limitations of resuscitation, **1**, 1169.

Lancet (1974). (Leading article.) Coma after cardiac arrest, **2**, 1302.

Leake, F. (1959). Case report quoted. (Resuscitation of stillborn infants. H. H. Eppell and J. M. Sutherland.) *Obstetrical and Gynecological Survey*, **14**, 366.

Mathews, D. H., Avery, M. E., and Jude, J. R. (1963). Closed-chest cardiac massage in the newborn infant. *Journal of the American Medical Association*, **183**, 964.

Milani-Comparetti, A., and Gidoni, E. A. (1967). Routine developmental examination of normal and retarded children. *Developmental Medicine and Child Neurology*, **9**, 631.

Moya, F., James, L. S., Burnard, E. D., and Hanks, E. C. (1962). Cardiac massage in the newborn infant through the intact chest. *American Journal of Obstetrics and Gynecology*, **84**, 798.

Neligan, G., Prudham, D., and Steiner, H. (1974). *The Formative Years: Birth, Family and Development in Newcastle upon Tyne*, Chap. 2. Oxford University Press for Nuffield Provincial Hospitals Trust, London.

Sheridan M. D. (1958). *The Stycar Hearing Test*. National Foundation for Educational Research in England and Wales, Windsor.

Sheridan, M. D. (1960). *The Stycar Vision Test*. National Foundation for Educational Research in England and Wales.

Steiner, H. (1974). A developmental and neurological follow-up study of children who survived severe perinatal problems. M. D.Thesis, University of Bristol.

Surks, S. N., and Ladner, W. (1962). Closed-chest cardiac massage in the stillborn. *Journal of the American Medical Association*, **180**, 328.

Wechsler, D. (1949). *Wechsler Intelligence Scale for Children*. Psychological Corporation, New York.

Wechsler, D. (1967). *Wechsler Pre-school and Primary Scale of Intelligence*. Psychological Corporation, New York.

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