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From Paediatric Unit — Kandang Kerbau Hospital for Women.

Moderator: DOCTOR GWEN SMITH.

INTRACRANIAL BIRTH INJURY

Case Reports

3 cases presented by Doctor Chen Chi Wei.

CASE No. 1

Regd. No. 24256. C.A.H. Chinese. Aged 37 years. Married 14 years.

OBSTETRIC RECORD:

Previous History:

1st child 12 years ago, S.B. cause unknown.

2nd child 10 years ago, Normal delivery.

3rd child 8 years ago, Normal delivery.

4th child 7 years ago, S.B. cause unknown.

5th child 6 years ago, S.B., Prolapsed cord.

6th child 2 years ago, L.S.C.S., Toxaemia and Disproportion, weighing 7 lbs. 5 ozs.

Present Pregnancy: L.M.P.: 13.3.56. E.D.D.: 20.12.56. Booked. Health satisfactory. K.T. not taken. Foetus tending to be breech/oblique. X'ray pelviradiography done. Routine laboratory investigations normal. For elective caesarean section at term.

Labour Ward Notes: 16.12.56.

Labour commenced. 2 hours 20 minutes later on arrival at labour ward I. membranes ruptured and foot presented at vulva—L.S.A. Before L.S.C.S. could be performed, patient bore down and delivered breech up to umbilicus. the cord beating feebly. Kielland's forceps were applied to the after coming head half an hour after the umbilicus had appeared.

Infant Apgar I, feeble heart beat, resuscitated with oxygen, intubation and Nikethamide 1 ml.

INFANT'S RECORD:

Male. B.W. 5 lbs. 4 ozs.. Length 18 inches, Apgar I at birth. Flaccid and unable to suck.

Management:

1. Resuscitation (see above).
2. Nursed flat in cot.
3. Aspiration of mucus.
4. Syrup Chloral gr. 1 x 6 hourly for 24 hours.
5. Penidure 50,000 units x 3 hourly 3 days.

Spoonfed with E.B.M. followed by Lactogen mixture.

11th day: Developed fever and thrush. Treated with:

1. Glycerine Borax—3 hourly.
2. Gentian Violet 1% t.d.s. — 48 hours.
3. Penicillin 50,000 units I/M.— 6 hourly, and Streptomycin 50 mg. b.d.

25th day: Discharged. Weight 6 lbs. 2 ozs., baby was still hypotonic and lethargic. He was spoonfed on medium strength Lactogen 2½ ozs. x 7 feeds.

CASE No. 2

Regd. No. 10059. Bengali Aged 27 years.

OBSTETRIC RECORD:

Previous History:

1st child 14 years ago, Normal delivery.

2nd child 4 years ago, Normal delivery (died at 3 years, cause unknown).

3rd child 1¼ years ago, Normal delivery, A.P.H.

Present Pregnancy: Gestation unknown.

Unbooked.

Admitted 17.5.57. Said to have bled 35 ounces in single episode at home.

Physical examination revealed the patient to be in shock.

Presentation L.O.A. Central placenta praevia.

An E.U.A. was done in theatre: Os 1 finger dilated, cervix long and tubular, foetal heart very faint, foetus about 36 weeks. L.S.C.S. was done 1 hour 55 minutes after admission. Infant Apgar 4 and was given Nikethamide ½ ml and oxygen. Mother received blood transfusion of 3 pints.

INFANT'S RECORD:

Male, B.W. 5 lbs. 4 ozs., Length 18 inches. Apgar 4 at birth Cerebral grunt, sternal retraction, poor air entry especially left lung. Flaccid, gross overriding of sutures.

Management:

1. Nursed with head of cot raised.
2. Syrup Chloral gr. 1 x 6 hourly for 2 days.
3. Oxygen.

Feeding started after 36 hours. Vomiting was treated by a stomach washout and regrading of feeds to dextrose saline for 24 hours. Crepitations were heard in both lungs. A course of penicillin 50,000 units 6 hourly I/M. was given.

Aged 1 week the baby was less irritable and respirations were regular.

9th day: Discharged. Weight 4 lbs. 12 ozs. feeding on ¾ strength Lactogen 2½ ozs. x 8 feeds.

CASE No. 3.

Regd. No. 6557. L.P.G. Chinese. Aged 40 years.

OBSTETRIC RECORD:

Previous History:

Para 13. No other history available.

Present Pregnancy:

Gestation unknown. Unbooked. Gravida 14.

Labour Notes:

N.D. 2.4.57 — 10.05 p.m. — Labour 16 hours. Infant delivered in feeble state, blue, cried 2 minutes after birth.

INFANT'S RECORD:

Male, B.W. 4 lbs. 6 ozs., Length 15½ inches, condition as above.

Management:

1. No feeds first 36 hours.
2. Inj. Vit. K 1 mg. and Nikethamide ½ ml. stat.
3. Oxygen.

The baby died 16 hours 45 minutes after delivery.

AUTOPSY: "Intracranial haemorrhage with atelectasis of lungs.

Discussion

Doctor Field opened the discussion. She said that she divided these infants into 2 groups.

(1) Infants showing signs of cerebral irritation during the first 48 hours.

(2) Infants presenting on the 3rd day.

The first group was more likely to follow a rapid delivery rather than a difficult delivery. She compared the rapid compression and relaxation in the absence of proper moulding, to concussion. The premature infant was particularly susceptible to cerebral damage, as the delivery was usually rapid and the tissues, particularly blood vessels, were very fragile. Other lesions in which signs developed during the first 24 hours were tentorial tears and severe anoxia.

The second group, where symptoms were delayed, followed mild anoxia or a slow leaking haemorrhage and commonly followed breech delivery when the after coming head was delayed.

The characteristic signs of intracranial damage were:

1. Bulging fontanelle.
2. Refusal of feeds or poor feeding.
3. Vomiting.
4. Alteration in muscle tone. Flaccidity or spacticity could occur, the latter being more common.

Doctor Y. Khong then discussed the management of infants with intracranial damage. She stressed that the most important aspect of treatment was to reduce handling of the infant to a minimum. The head should be raised to reduce venous pressure. Oxygen should be given to all severe cases to reduce the risk of damage to cerebral tissues during the acute stage of anoxia. She said that blood or plasma transfusion of 5 c.c. per pound body weight was sometimes used to combat shock. Fluid intake was usually poor as these infants were unable to feed well. The mild dehydration which normally occurred dur-

ing the first few days was exaggerated. This was of value in reducing intracranial pressure provided that the dehydration was mild and was not progressive. Tube feeding was often used to maintain the necessary minimal intake.

Sedatives should be given if the baby were restless and if twitchings or convulsions occurred.

Vitamin K should be given to all cases where intracranial damage was likely, in order to prevent extension of bleeding due to hypothrombinaemia.

Lumbar puncture was advocated by some authorities to relieve intracranial pressure and remove blood from the C.S.F. to minimise residual fibrosis.

Doctor Smith outlined the basic treatment in current use in this hospital. The first 48 hours were allowed to elapse without giving feeds and subsequently a sufficient amount was given to maintain hydration. She did not consider hypertonic rectal saline a useful method of therapy.

Blood transfusion was contraindicated unless there was good evidence that the baby was suffering from haemorrhagic disease of the newborn, where Vit. K had been given without effect. Cerebral shock due to extensive hamorrhage was fatal. The rise in blood pressure following transfusion was potentially dangerous and may extend the haemorrhage. Sedative therapy was necessary where there was evidence of cortical irritation e.g., restlessnes, or fits. Chloral was the most useful drug either alone or combined with luminal. Cases where there was damage to the vital centres or cyanotic attacks, did not require sedation. Lumbar puncture was only of use as a diagnostic measure particularly to exclude meningitis.

Subdural taps were indicated where there was evidence of progressive increase of intracranial pressure. She quoted a case where hemiplegia increased and paralysis spread to the other side. Subdural tap revealed a large subdural haemorrhage.

Doctor Field emphasised the value of the prophylactic use of Vit. K in the prevention of extension of small haemorrhages due to hypoprothrombinaemia. She considered lumbar puncture was contraindicated as it would increase the size of any haemorrhage present.

Doctor Smith then discussed the prognosis for cases of intracranial birth injury. There were three methods of studying this problem.

1. Retrospective method. The birth histories of children suffering from cerebral palsy and mental defect were studied.
2. Follow-up study of infants following operative delivery.
3. Detailed follow-up study of infants showing signs of intracranial injury in the neonatal period.

She then quoted an example of each type of study:

Retrospective studies were common and were part of the work that had been done and was still in progress on groups of cerebral palsied children. This method was the least satisfactory as a history taken from a mother with an abnormal child was likely to be inaccurate. She would unconsciously exaggerate minor difficulties during labour and the neonatal period to explain her child's affliction. Gustavson quoted 185 cases. Mature infants 62% had spontaneous delivery. Premature infants 84% had spontaneous delivery.

There was a predominance among primiparae which was not true for congenital abnormalities as a whole. This evidence suggested that intracranial injury at birth was an important factor in the aetiology of cerebral palsy.

The incidence of spastic children was high—about .75 per thousand in England. The figure for Singapore was unknown; a register was at present being compiled. The cases referred to paediatric outpatients suggested that the incidence here was as high as in cities in other parts of the world.

She quoted a series of 190 cases of cerebral palsy reported by Bobath (1956). Of these, 36 had a history of asphyxia at birth, 37 were delivered by forceps and 43 showed other complications during the neonatal period.

These studies suggested that cerebral injury in the newborn was an important

aetiological factor in the development of cerebral palsy.

The second method of investigation was adopted by Krukenberg. He analysed 1.300 cases delivered at term by forceps and followed them up to the age of 2 years. The incidence of cerebral damage was

2% affected following high forceps.
1.5% „ „ mid forceps.
0.5% „ „ low forceps.
4% „ „ breech extraction.

The most valuable study published recently was that of Craig (1950). He studied 593 infants in whom he had diagnosed intracranial injury during the neonatal period. 472 were seen aged 1 year and 306 aged 5 years or more. Permanent sequelae were as follows:—

Physical disability	19
Physical disability with mental handicap	22
Mental disability alone	9
Convulsions after the age of 3 weeks with no other handicap	4

He pointed out that the prognosis for the individual case was much better than usually assumed. It was the exception and not the rule for an infant on whom there were no signs of residual disability during the neonatal period to develop permanent sequelae. There was no direct correlation between the severity of symptoms at birth and the eventual prognosis.

Doctor Field agreed that Professor Craig's work was a most valuable study. She hoped he would follow his cases further, through school life where less severe mental change was often diagnosed.

Doctor Saunders quoted a case of a difficult forceps delivery where there were definite signs of caudal and intracranial irritation. Burr holes were made and the haematoma syringed out.

Doctor Smith said that subdural haematoma were usually satisfactorliy treated by aspiration, during the acute phase. Surgical drainage was performed later if a membrane found.

Professor Sheares quoted a case where spasticity had followed a low forceps delivery with a long period of anoxia neonatorum. He emphasized the importance of good obstetrics in the prevention of intracranial injury.

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