# A SURVEY OF MECONIUM STAINING OF AMNIOTIC FLUID AND MECONIUM ASPIRATION SYNDROME IN KENYATTA NATIONAL HOSPITAL

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I CERTIFY THAT THIS DISSERTATION IS MY ORIGINAL WORK AND HAS NOT BEEN PRESENTED FOR A DEGREE IN ANY OTHER UNIVERSITY.

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# C O N T E N T S

## S U M M A R Y

A 3 - month survery on meconium staining of amniotic fluid and meconium aspiration syndrome, was done. 1103 infants were born, 140 (12.7%) had passed meconium in utero and 9(0.82%) developed meconium aspiration syndrome. 8(88.9%) of meconium aspiration syndrome babies had had tracheal suction. 4 babies died, giving a mortality rate of 44.4% of the infants who developed meconium aspiration syndrome. There was a higher incidence of abnormal foetal heart rates and low Apgar scores in the meconium stained infants than among the controls. The caesarean section rate was 2 times higher in the meconium group than among the controls.

# I N T R O D U C T I O N

## DEFINITION

Meconium aspiration syndrome is an aspiration pneumonia with progressive respiratory distress within the first 24 hours after birth, (1, 2, 3, 4.)

It has been recognized for a long time as an important cause of neonatal morbidity and mortality. The clinical picture varies from mild transient tachypnoea to severe respiratory distress (3-8). The diagnosis of meconium aspiration syndrome requires;

(i) presence of meconium-stained amniotic fluid and/or meconium-stained baby

(ii) development of clinical respiratory distress within 24 hours of life, and

(iii) chest x-rays consistent with aspiration pneumonitis (1,9,10).

The finding of meconium in the trachea alone does not constitute an aspiration syndrome, (3).

## PATHOPHYSIOLOGY OF MECONIUM ASPIRATION SYNDROME:

Investigations by earlier workers demonstrated chemical pneumonitis induced by meconium. Cruickshank (11) injecting human meconium into the tracheas of rabbits showed an abudant polymorphnuclear leucocyte infiltration and alveolar collapse. He also demonstrated that amniotic fluid alone produced little reaction. Tyler et al (8) showed that chemical pneumonitis was of late onset and insidious. They suggested that bile salts and proteolytic enzymes, both of which are components of meconium could possibly be responsible for the reaction. They

excluded a possibility of low pH or bacteria causing the pathophysiological changes in their study (12).

Clark et al (13) demonstrated that meconium could change the surface tension of lung extract in vitro. They did not, however, establish the chemicals involved.

However, mechanical airway obstruction by meconium particles plays the most important role in the pathophysiology of meconium aspiration syndrome. The changes that follow the obstruction depend on the amount of meconium aspirated and the site of obstruction. Large meconium plug is capable of blocking the large airways, leading to rapid death from asphyxia and acute cor pulmonale The transition to airbreathing may not be accomplished. Cor pulmonale may develop even before pulmonary changes occu (3,5,8,14). Smaller amounts of meconium particles move quickly to lung periphery resulting in obstruction of the distal airways. Gregory et al (3), using tentalum-labelled meconium demonstrated this migration which normally takes about one hour. The obstruction that follows may be complete, leading to atelectasis of distal alveoli. There is right-to-left intrapulmonary blood shunting and ventilation-perfusion abnormalities. Severe hypoxaemia results, demonstrated by a fall in arterial oxygen tension. If there is partial obstruction of the small airways, a "ball-valve" effect can result. Air can pass by the obstruction during inspiration since the airways widen. But during expiration the airways collapse around the obstruction leading to air trapping. As more air is trapped distally, the alveoli expand, eventually some may rupture resulting in pneumothorax or pneumomediastinum. The air trapping is demonstrated by a rise in functional residual capacity and emphysema seen on chest X-rays.

Other changes that occur as a result of meconium aspiration include:-

(i) a fall in lung compliance and an increase in a pulmonary airway resistance.

(ii) Increased pulmonary vascular resistance -

(iii) Decreased cardiac output and persistent foetal circulation, which aggravate the already altered ventilation - perfusion ratio.

THE PATHOPHYSIOLOGY OF MECONIUM PASSAGE IN UTERO:

The passage of meconium in utero and its significance has been a controversial subject. It is generally accepted that meconium passage in utero is a response to foetal hypoxia. The mechanism is that the hypoxia induces mesenteric vasoconstriction which, in turn, causes transient intestinal hyperperistalsis. Therefore meconium is seen more frequently in situations which may cause reduction in Oxygen supply to the foetus. Such situations include maternal hypertension, anaemia, toxaemia chronic pulmonary disease, prolonged pregnancy, cord accidents and difficult labour and delivery. Lucas et al (15) supported this when they found a higher level of motilin in cord blood of infants who had passed meconium in utero and also had abnormal foetal heart rate pattern i.e. foetal distress.

Fenton and Steer (16) contended that meconium passage is a normal physiologic function of a term and post-term foetus. Abramovici et al (17) and Miller et al (7) did not find any significant difference in foetal heart rate pattern and scalp blood pH between foetuses with meconium stained amniotic fluid and those without meconium. This was described as probably a temporary compensated foetal distress state, with a decompensated state following, if intervention is delayed. Despite these findings, the presence of meconium, an abnormal foetal heart rate pattern and PH changes, are associated with increased infant morbidity and mortality as shown by Krebs et al (18).

When meconium passage occurs in utero then the stage for meconium aspiration syndrome is set. Meconium aspiration may occur even before delivery in an asphyxiated foetus. A few cases of intrauterine meconium aspiration have been reported. The methods of resuscitation after delivery contribute very much to the development of symptomatic meconium aspiration syndrome. Other factors like method of delivery, amount of meconium in trachea, do influence the outcome of the meconium-stained pregnancy (15,19,20)

INCIDENCE

It is very important to know the incidence of a disease so as to take appropriate measures in its prevention and management. The incidence of meconium staining aspiration syndrome have been variable from one report to another (1,3,4,5,8.) However Bacsik (5) in a review article summed up the various figures to give the incidence of meconium staining as 8.29% of all deliveries and that of meconium aspiration syndrome as 1-3%.

There is no survey previously done in Kenyatta National Hospital on meconium staining and meconium aspiration syndrome. Looking at perinatal mortality figures compiled in monthly reports in the Departments of Obstetrics and Gynaecology and those from Paediatrics, one hardly ever finds a diagnosis of meconium aspiration syndrome. From postmortem reports, most perinatal deaths are attributed to intrapartum hypoxia and the significance of meconium is found in the airways may be overlooked. Whereas meconium is found during labour its effect on subsequent route of delivery has not been determined. The indications for caesarean sections, for example, may be reported as foetal distress without giving the criteria for diagnozing the distress.

The aims and objectives of this study are to carry out a survey on meconium staining with an intention of finding out:-

(i) the incidence of meconium staining of amniotic fluid, and the incidence of symptomatic meconium apiration syndrome, in Kenyatta National Hospital,

(ii) the effect of meconium staining of amniotic fluid on subsequent selection of route of delivery.

(iii) To compare the foetal heart rate and Apgar scores of meconium stained infants and those of infants without meconium staining.

## MATERIALS AND METHODS

This study was done in Kenyatta National Hospital Maternity Unit between 1st July, 1981 and 30th September, 1981. All the mothers who were admitted in labour ward for delivery were included in the study, except those admitted with intrauterine death. The latter were left out because they were not expected to be born alive and hence could not be included in those who would develop symptomatic meconium aspiration syndrome. Note that the referenced work excludes intrauterine deaths in their studies (3,5,7,17).

All mothers had an obstetrical examination on admission. Those who had membranes ruptured spontaneously or artificially, had the amniotic fluid inspected for meconium. At amniocentesis or at delivery, meconium was looked for in the fluid. The time when meconium was first seen was noted and the author or a paediatrician on duty were informed, if author was not present at the time. The foetal heart was monitored by intermittent auscultation, using a foetoscope, at intervals of 30 minutes. Any foetal heart rates that became less than 120 or more than 160 beats per minute or were irregular, were regarded as abnormal. For these foetal hearts auscultation was done every 15 minutes. A partogram was used to record all the observations and progress of labour (Appendix I). The decision of route and method of delivery was made by the obstetrician.

The indications for caesarean sections or vacuum extraction, time when decision was made and delivery accomplished, were recorded.

After delivery, the infants were immediately assessed by Apgar score method between 0 and 1 minute by the author or paediatrician on duty. In the majority of cases this was done while oral and nasopharyngeal suction was being done. In a few, assessment was done before suction was started. Infants who scored 6 or less at 1 minute or had thick meconium on the body and mouth, had tracheal suction. This was accomplished by using a neonatal laryngoscope for direct visualization of the cords and suction done with a polythlene tube. If there was no respiratory effort in some of these infants during tracheal suction, an endotracheal tube, 3mm diameter, was passed into the trachea immediately and followed by suction through this tube. Oxygen was then given by intermittent positive pressure ventilation, not exceeding 25cm$H_2O$, till spontaneous and regular respiration started or till the infant died. Intravenous bicarbonate and glucose were given to those with slow heart rate and cyanosis. Those infants who scored 7 or more at 1 minute and did not have thick meconium on the body and/or mouth, had nasopharyngeal and oral suction only. 5 minute Apgar scores were determined. The procedure for resuscitation was recorded on the protocol as provided (Appendix II & III).

After resuscitation the baby's condition was reassesed by using

(i) Motor activity
(ii) respiratory rate
(iii) Heart rate
(iv) Presence or absence of cyanosis.

Gestational maturity was estimated by using dates and external physical score of Dubowitz and Dubowitz[24]. The neurological score was inappropriate since some of the infants had central nervous system depression and would give a false score,( ). The babies were then weighed and categorised into

(a) appropriate for gestational age (AGA)
(b) small for gestational age (SGA)
(c) Large for gestational age (LGA) according to gestational age by dates, physical examination and weights.

The weights were grouped into 2000 gms or less, 2001 to 25000gms, 2501 to 3000 gms, and above 3000 gms.

All the infants who had meconium staining of amniotic fluid were admitted to nursery. The following signs were observed for:-

(a) rapid respiratory rate, of over 60 per minute
(b) chest retractions
(c) grunting
(d) cyanosis
(e) chest crepitations
(f) reduced air entry

Any infant who developed a rapid respiratory rate, chest retractions and crepitations was regarded as sick and more investigations were carried out. A chest X-ray, blood gas analysis and septic screen were done. Blood gases were analysed in intensive care unit using pH-Blood Gas Analyser-213 and pH-Blood Gas calculator by Instrumentation Laboratory inc... Management consisted of oxygen by mask, incubator care, intravenous infusion of 10% dextrose and antibiotics. These were Kanamycin and crystapen which were currently in use in the nursery in high risk babies while awaiting laboratory culture reports. When amniotic fluid is mixed with meconium it acts as a good media for bacterial growth, hence prophylactic antibiotics. The infants who did not develop any respiratory distress after 24 hours were discharged.

The infants who had meconium staining and failed to establish normal respiration during initial resuscitation were transfered to nursery from labour ward to continue with intermittent positive pressure ventilation, dextrose and antibiotics as stated above. No mechanical ventilator was used. Those who died had a postmortem examination.

The diagnosis of meconium aspiration syndrome was made in the infants using the following criteria:-

(i) Meconium staining

(ii) Clinical respiratory distress in the first 24 hours of life

(iii) Chest radiological changes consistent with aspiration (1, 22) or

(iv) An infant who failed to establish normal breathing after birth and when post mortem was done after death, meconium was found in the airways.

The controls were selected from among those with mothers who delivered, without meconium being detected in the amniotic fluid at any phase of labour, during the same period. Every 5th delivery without meconium was taken (systematic sampling), (23). The controls had the same observations as the meconium-stained deliveries.

N.B. Meconim aspiration syndrome in the subsequent pages refers to symptomatic meconium aspiration syndrome.

RESULTS

The total number of deliveries during the period of study was 1103.

TABLE I

Incidence of Meconium staining of amniotic fluid and that of symptomatic meconium aspiration syndrome.

| | No. | Percentage % | Incidence per 1,000 births |
|---|---|---|---|
| Meconium-stained amniotic fluid | 140 | 12.7 | 127 |
| Meconium aspiration syndrome | 9 | 0.82 | 8.2 |

From Table I, the incidence of meconium staining is 12.7% while that of meconium aspiration syndrome is 0.82%
It works out that meconium aspiration occured in 6.4% of those who had meconium staining.

TABLE II a

Distribution of 1-Minute Apgar Scores

| Apgar scores | with meconium staining | without meconium staining |
|---|---|---|
| 0-6 | 37 (26.4%) | 7 (5%) |
| 7-10 | 103 (73.6%) | 133 (95%) |
| Mean Apgar score | 7.8 ± 2.21 | 9.1±1.41 |

Table IIa shows the distribution of 1-minute Apgar scores. The difference between the means is significant; the difference between the two means being greater than twice the standard error (S.E.) of the difference- S.E.=0.22.

TABLE IIb

One minute Apgar scores of 6 or less compared between the meconium stained group and those without meconium staining.

| | with meconium staining | without meconium staining |
|---|---|---|
| No. (%) | 37 (26.4) | 7 (5%) |
| Apgar score range | 2 - 6 | 3 - 6 |
| Mean Apgar score<br>SD | 4.65<br>± 1.23 | 4.7<br>± 1.38 |

However, when Apgar scores of 6 or less are taken there is no significant difference between those with and those without meconium staining (SE = 0.313) as shown in table IIb.

TABLE IIIa

Distribution of 5-minute Apgar scores



| Apgar scores | with meconium staining | without meconium staining |
|---|---|---|
| 0-6 | 10 (7.1%) | 5(3.6%) |
| 7-10 | 130 (92.9%) | 135 (96.4%) |
| Mean Apgar Scores | 9.1± 1.65 | 9.7± 0.95 |

In table III a, there is a significant difference between these Apgar scores; the infants with meconium staining having a lower mean Apgar score. S.E. = 0.0136

TABLE III b.

The 5- minute Apgar Scores of 6 or less compared between the meconium group and the controls:

| | With Meconium staining | Without meconium staining |
|---|---|---|
| Number (%) | 10 (7.1) | 5 (3.6) |
| Apgar Score Range | 1 - 6 | 5 - 6 |
| Mean Apgar Score | 9.1±1.65 | 9.7±0.95 |

As Shown in Table III b, there is a significant difference between the mean Apgar scores of the meconium - stained babies and the controls (S.E. = 0.616). Note the much higher Apgar Scores among the controls.

TABLE IV

Distribution of one-and five minute Apgar Scores in infants with meconium aspiration syndrome.

| Apgar Score | | No. | % | Mean Apgar Score |
|---|---|---|---|---|
| 1-minute | 0-6 | 8 | 88.8 | 3.8±1.69 |
| | 7-10 | 1 | 11.2 | |
| 5-minutes | 0-6 | 6 | 66.6 | 5.0±2.60 |
| | 7-10 | 3 | 33.4 | |

Table IV shows the distribution of Apgar scores of the nine infants who developed respiratory distress. Their Apgar scores both at 1-minute and 5 minutes were very low range being 2-6. The 1-minute Apgar score frequencies were as follows:-

TABLE IV (cont.)

2 infants had a score of 2,
3 infants had a score of 3,
2 infants had a score of 4,
1 infant had a score of 6, and
1 infant had a score of 7 .

TABLE V.

1-minute Apgar scores and tracheal suction

| Apgar Score | Total | Number Suctioned | Number not suctioned |
|---|---|---|---|
| 0-6 | 37 | 29(65.9%) | 8(34.1%) |
| 7-10 | 103 | 15(15.6%) | 88(84.4%) |
| Total | 140 | 44 | 96 |

There were 44 (30%) infants who had tracheal suction as seen in Table V and out of these, 23 (52.27%) infants had meconium suctioned out of the trachea. The remaining infants were either too vigorous to tolerate tracheal suction, or had no meconium on body, nose nor mouth.

TABLE VI

Tracheal suctioning and meconium aspiration

| | Total | Developed meconium aspiration | No meconium aspiration |
|---|---|---|---|
| Suctioned | 44 | 8 | 36 |
| Not suctioned | 96 | 1 | 95 |
| Total | 140 | 9 | 131 |

The suctioned babies had a significantly higher incidence of meconium aspiration than those who were not suctioned as seen in Table VI, $x^2 = 14.7$, $p<0.05$. The 8 infants who were suctioned and developed meconium aspiration syndrome were the same who had Apgar scores of 6 or less, shown in Table IV, and 7 of them had meconium in the trachea. These 7 infants make 30.4% of those who had meconium in the trachea.

TABLE VII

Distribution of Foetal Heart Rate recorded within one hour of delivery.

| Foetal heartrate and rhythm Patients | Total (%) | 120-160 Regular No.(%) | 120-160 Irregular No.(%) | >160 | <120 | Not recorded | total abnormal No.(%) |
|---|---|---|---|---|---|---|---|
| Without meconium staining | 140 (100) | 130 (92.86) | 1 (0.71) | 1 (0.71) | 3 (2.14) | 5 (3.58) | 5 (3.58) |
| With Meconium Staining | 140 (100) | 113 (80.71) | 10 (7.14) | 4 (2.86) | 9 (6.43) | 4 (2.86) | 23 (16.43) |
| With Meconium Aspiration syndrome | 9 (100) | 4 (44.44) | 1 (11.12) | 2 (22.22) | 2 (22.22) | 0 - | 5 (55.56) |

In table VII, showing the foetal heart rates recorded within an hour before delivery, the incidence of abnormal foetal heart rates in the meconium stained infants is higher (16.43%) than that in the controls (3.58%) $x^2 = 12.76$ $P<0.05$. Among the infants who developed meconium aspiration syndrome 5(55.56%) had abnormal foetal heart rates.

Those mothers who came in second stage of labour and delivered in the examination room could not have the foetal heart rate recorded.

TABLE VIII

Comparison of Methods of Delivery.

| Method of Delivery | with meconium | without meconium |
|---|---|---|
| Spontaneous Vertex | 73 (52.1%) | 110 (78.6%) |
| Caesarean Section | 56 (40%) | 25 (17.9%) |
| Vacum extraction | 7 (5%) | 2 (1.4%) |
| Breech | 4 (2.9%) | 3 (2.1%) |

Table VIII compares methods of delivery between the meconium stained pregnancies and those without meconium staining. There was significant difference between the methods of delivery selected in the two groups, ($x^2 = 22.26$, $P<0.01$). The main difference came from the spontaneous vertex deliveries and caesarean sections. Among the nine infants who had meconium aspiration, 4 were delivered by caesarean section, 4 by spontaneous vertex delivery and 1 by vacuum extraction.

TABLE IX

Indications for Caesarean section

| Indications | with meconium staining | without meconium staining |
|---|---|---|
| Abnormal Foetal Heart | 17 | 0 |
| Normal Foetal Heart | 16 | 0 |
| Previous scar | 8 | 10 |
| Breech in Primigravida | 4 | 1 |
| Postmaturity | 2 | 1 |
| C.P.D. andB.O.H. | 0 | 1 |
| C.P.D | 1 | 5 |
| Obstructed Labour | 7 | 3 |
| Transverse Lie | 0 | 1 |
| P.O.P | 0 | 1 |
| A.P.H. | 0 | 1 |
| Total: | 56 | 25 |

Key: A.P.H. = Antepartum Haemorrhage
C.P.D = Cephalopelvic Disproportion
B.O.H. = Bad Obstetrical History
P.O.P. = Persistent Occiput Posterior

Table IX compares the indications for caesarean section in the meconium stained group and the group without meconium staining. There were 33(53.5%) caesarean sections done in the meconium group, where the indications was the presence of meconium, with or without abnormal foetal heart rates. Other caesarean sections in this group were done because of other indications other than meconium stained amniotic fluid.

TABLE X

Comparison of Birth Weights.

| Birth Weights (gms) | with meconium | without meconium |
|---|---|---|
| 2000 or less | 3 (2%) | 7 (5%) |
| 2001-2500 | 14 (10) | 15 (11%) |
| 2501-3000 | 43 (31% | 45 (32%) |
| 3001 and above | 80 (57%) | 73 (52%) |
| Total | 140 | 140 |
| Mean Weights | 3084±500 | 2960±578 |

Table X compares the birth weights of infants who passed meconium in utero to those who did not. The mean weights are different as shown in the table but, using 't' test $P > 0.05$. The difference in the birth weights of the two groups was not significant.

The wider weight scatter, as seen by a higher standard deviation, for the infants without meconium staining was caused by preterm babies weighing as low as 900 gms.

TABLE XI

Comparison of gestational Age.

| Gestational age (weeks) | with meconium staining | without meconium staining |
|---|---|---|
| 36 or less | 9 (6.5%) | 24 (17%) |
| 37-42 | 108 (77%) | 107 (76.5%) |
| Above 43 | 9 (6.5%) | 7 (5%) |
| Not known | 14 (10%) | 2 (1.5%) |
| Total | 140 | 140 |

Table XI compares the gestational age by dates between infants with meconium passage in utero and those without. There is a higher percentage of infants with gestational age of 36 weeks or less in the group without meconnium staining than among those with meconium. A relatively high number of mothers did not know the time they had their last menstrual flow while others never had any menstruation after a preceding pregnancy.

TABLE XII

Comparison of Gestational age by physical examination with meconium staining

| Age in Weeks by physical assessment | with meconium staining No. (%) | without meconium staining No. (%) | with meconium Aspiration syndrome No. (%) |
|---|---|---|---|
| 36 or less | 10 (7.9) | 23 (16.7) | 1 (11.1) |
| 37 - 42 | 101 (80.2) | 109 (79.0) | 7 (77.8) |
| Above 42 | 15 (11.9) | 6 (4.3) | 1 (11.1) |
| TOTAL | 126 (100) | 138 (100) | 9 (100) |

Table XII, still shows a higher incidence of premature infants among the non-meconium stained babies than those with meconium staining, on physcial examination. Among the infants who developed meconium aspiration 7(77.8%) were term, and only I had features of post maturity and the other features of intra-uterine growth retardation.

From Tables XI and XII, it can be seen that the number of premature babies differ considerably in the two groups - $x^2 = 7.7$ $P \leqslant 0.01$. So the difference in the incidence of prematurity between the meconium group and controls is statistically significant. It can also be deduced that the majority of infants who pass meconium in utero are above 36 weeks of gestation.

MORTALITY RATE

There were 4 deaths of the nine infants who developed meconium aspiration syndrome - a death rate of 44.4%. Three infants never established normal breathing, 2 dying within the first 15 minutes of life, while the third died at the age of 4 hours. The fourth infant died 4 days after birth and had developed bronchopneumonia. Postmortem examination done showed meconium in the airways of three infants and the fourth was found to have bilateral bronchopneumonia.

X-rays

Three infants died before chest x-rays could be taken. The six x-rays that were taken had the following findings:

(i) Three showed bilateral patchy coarse opacities

(ii) Two showed hemopacities consistent with lung collapse

(iii) The last one had normal x-rays. These were taken after 15 hours of age and the infant had shown clinical improvement.

CASE REPORTS

Four case reports are presented here to exemplify the clinical picture of the infants who had meconium aspiration syndrome.

CASE REPORTS

CASE 1

Mother W. M. N, a para $8^{+0}$, was referred from a District hospital with labour pains. On admission in that hospital a vaginal examination was done and the membranes were ruptured during the procedure. There was thick meconium in the amniotic fluid and a face was presenting. The foetal heart rate was normal.

On arrival at Kenyatta National Hospital, which was two hours after rupture of membranes, W.M.N. had strong and frequent contractions. The uterine size was appropriate for dates. The foetal heart rate was 142 per minute and regular. Cervix was fully dilated and thick meconium was draining. Five minutes later she had a spontaneous vaginal delivery.

The baby had an Apgar score of 7 at one minute and 9 at five minutes. He gasped before suction was done. He had only nasal, oral and oropharyngeal suction, from where little meconium-stained fluid was retrieved. No oxygen was given. He weighed 3320 gms and was appropriate for age by examination.

Thirty minutes later, the baby developed a rapid respiratory rate of 72 per minute with intercostal and subcostal retractions. There was no grunting nor cyanosis but air entry was reduced and the chest had bilateral crepitations.

He was started on oxygen by mask, nasogastric tube feeding and antibiotics (Crystalline penicillin and Kanamycin intramuscularly). Blood gas analysis done one hour after delivery showed slight hypercabia but good oxygenation -

| | | |
|---|---|---|
| Packed cell volume | = | 48.2% |
| pH | = | 7.37 |
| $pO_2$ | = | 80.mmHg |
| $pCO_2$ | = | 45.mmHg |
| $SO_2$ | = | 95.5% |
| $HCO_3$ | = | 25mEq/L |
| B.E. | = | 0 |

However oxygen therapy by mask was continued. Chest X-ray done at five hours of age showed a hemiopacity consistent with lung collapse -(see figure 1).

Day 2. Still had respiratory distress, with a respiratory rate of 80 per minute but intercostal and subcostal retractions were much less than previously. Bilateral crepitations were still present, more so on the left side. Air entry had improved. Oxygen by mask was continued and and intravenous drip of dextrose, 10%, started.

Day 3. Respiration had improved, respiratory rate was 64 per minute, no retractions, air entry good and no crepitations. But the temperature had risen to 38°C. Septic screening was done and antibiotics continued. Blood culture grew staphylococcus albus, considered a contaminant. Oxygen therapy was stopped.

Day 4. There were no signs of respiratory distress and no fever. Child was well and discharged to mother on Day 5.

N.B. (i) Serial X-rays were not done because of using a portable unit and the expense involved.

(ii) Repeat blood gases were the same as the first ones, on day 2.

(iii) Repeat blood culture showed no bacterial growth after 48 hours.

Comment:-

This infant had a moderate respiratory distress from which he recovered without requiring positive pressure ventilation.

CASE II

Mother R.M. was admitted in labour at the gestational age of 37 weeks by dates and uterine size. Foetal heart rate was 140 per minute and regular. Membranes were artificially ruptured then and the amniotic fluid was thinly meconium stained. Two hours and five minutes later, the foetal heart rate was still normal and R.M. had a spontaneous vaginal delivery. Thick meconium came out in the remaining amniotic fluid.

The infant was depressed with 1-minute Apgar score of 3. Oral and nasopharyngeal suctioning started immediately after delivery of the baby. As laryngoscopy was being applied the infant started gasping. Tracheal suction was done with meconium-stained fluid being extracted. Endotracheal tube of 3.0 mm diameter was inserted and more suction done after which oxygen was given intermittently under a pressure of 20 $cmH_2O$. He gradually improved, at five minutes he had an Apgar score of 7 and by thirty minutes was breathing spontaneously without oxygen.

Physical examination 30 minutes after birth revealed a postmature infant, weighing 3170 gms, with respiratory distress. Respiratory rate was 74 per minute, had intercostal and subcostal retractions and mild cyanosis peripherally. The chest had bilateral coarse crepitations and reduced air entry. Intravenous Kanamycin and crystalline penicillin were started, and he was also started on oxygen by mask and an intravenous infusion of 10% dextrose. Blood gases done at 1 hour of age were:

| | | |
|---|---|---|
| P.C.V | = | 60% |
| pH | = | 7.27 |
| $pO_2$ | = | 58 mmHg |
| $pCO_2$ | = | 38 mmHg |
| $HCO_3$ | = | 16.5 mEq/L |
| BE | = | -9 mEq/L |

Oxygen therapy and intravenous infusion was continued for twenty-six hours and stopped. A chest X-ray taken four hours after birth showed bilateral patchy opacities (see Figure 2).

The infant had a rapid recovery on oxygen. By eight hours respiratory rate was 56 per minute, retractions were minimal but the chest still had crepitations. By 24 hours there were no crepitations and no signs of respiratory distress. He was discharged on Day 3.

CASE III

Mother D.O., a primigravida, was admitted 14 days before her expected date of delivery with a history of labour pains lasting over seven hours. On admission, her blood pressure was 130/80 mmHg, she had weak contractions and foetal heart rate was 136 per minute, regular. The membranes were intact and cervix was 2 cm. dilated.

16½ hours later, she had not progressed satisfactorily; contractions were still weak, cervix was 4 cm. dilated and foetal heart rate was 160 per minute but regular. Artificial rupture of membranes was done and the amniotic fluid had thick meconium. There was no cord presenting. A diagnosis of foetal distress was made and the patient was prepared for caesarean section.

15 minutes later the foetal heart rate was down to 100 per minute and regular. After 45 minutes the baby was delivered by caesarean section. She was very depressed, with a 1-minute Apgar score of 2 and had meconium staining all over the body.

Resuscitation by immediate intubation and suction, with removal of thick meconium from the trachea, was done. Oxygen was given by intermittent pressure of 20 $cmH_2O$, and intravenous 50% dextrose (2mls diluted), intravenous sodium bicarbonate (4mls diluted) were given. The heart beat remained at about 60 per minute and cyanosis deepened.

There was no response to resuscitation. At 7 minutes, the heart stopped and external cardiac massage was done, with intracardiac adrenaline being given. However, there was no response and the infant died at the age of ten minutes.

Post mortem was done the same day - report:
Weight was 2700 gms. Infant was covered with thick meconium. Respiratory tract with meconium down to small airways. There was patchy aeration and no pneumothorax. There was air and blood in the pericardium, possibly as a result of resuscitation. Cause of death - intrapartum asphyxia with meconium aspiration.

Comments:

This child gives a good example of acceleration and then deceleration of the foetal heart rate in a foetus in distress, which is best seen when using continuous monitoring. It was still possible to detect this with intermittent auscultation, though this may have been late. The decision to deliver by caesarean section was taken following meconium staining and not an abnormal foetal heart rate.

This case was similar to the one reported by Frank Manning (19), where an infant with intrapartum asphyxia aspirated the meconium before delivery. In addition to intra-uterine aspiration, intermittent positive pressure applied before thorough cleaning of the trachea could push the meconium further down leading to more difficulties in its removal, Carson et al, (33).

CASE IV

Primigravida M.A was admitted at term with a history of fits, twice before arrival at casualty department. She had been attending ante-natal clinic outside Kenyatta National Hospital and did not have her record from that clinic with her.

On admission in labour ward, she was deeply sedated with valium, had pitting oedema of the legs, a blood pressure of 190/150 mmHg and $4^+$ of albumin in the urine. The foetal heart rate ranged between 136 and 158 per minute and regular (counted by four different people), with an average of 142 per minute.

A diagnosis of eclampsia was made and she was started on pethidine, hemineverin and valium. 5 hours after admission a caesarean section was done while the foetal heart rate was still normal, 142 per minute and regular. On rupture of membranes at operation, thick meconium was seen. The infant was very depressed with a 1-minute Apgar score of 4. The Baby was covered with meconium.

Immediately endotracheal intubation and suction were done. Very thick meconium was suctioned out of the nose but none was found in the trachea. Intermittent positive pressure ventilation was done with oxygen at 25 $cmH_2O$. Intravenous 50%dextrose, sodium bicarbonate and nalorphine, were given. By 5 minutes Apgar score was down to 3 and resuscitation continued. Spontaneous breathing started at 10 minutes but this was irregular gasps.

At 30 minutes of age the infant had regular sustained but shallow breathing movements. Oxygen flowing continuously at 2 litres per minute was started, together with a 10% dextrose infusion, in the nursery. Intravenous Gentamycin and Crystalline penicillin were also started. Blood gases done at 45 minutes after birth (blood taken from brachial artery) showed:

| | | |
|---|---|---|
| pH | = | 6.75 |
| $pO_2$ | = | 44 mmHg |
| $pCO_2$ | = | 89 mmHg |
| $SO_2$ | = | 44% |
| $HCO_3$ | = | 12 mEq/L |
| BE | = | -25 mEq/L |

There was severe acidosis and 10 mls of $NaHCO_3$ was given intravenously. At the age of three hours the infant started having apnoeic episodes and intermittent positive pressure ventilation was resumed, this time there was no pressure gauge except the flowmeter. This was continued for one hour and the baby died at the age of four hours. Chest X-ray could not be taken before death because of the short period the baby lived.

Post mortem done next day showed a term infant, weighing 3460 gms, with meconium staining of skin, umbilical cord stump and nails. The respiratory tree was full of meconium-stained fluid especially the bronchi and bronchioles. There was a left lung collapse and left tension pneumothorax. Histology showed meconium and squamous debris in the lungs.

Cause of death; intrapartum and postnatal hypoxia due to meconium aspiration syndrome.

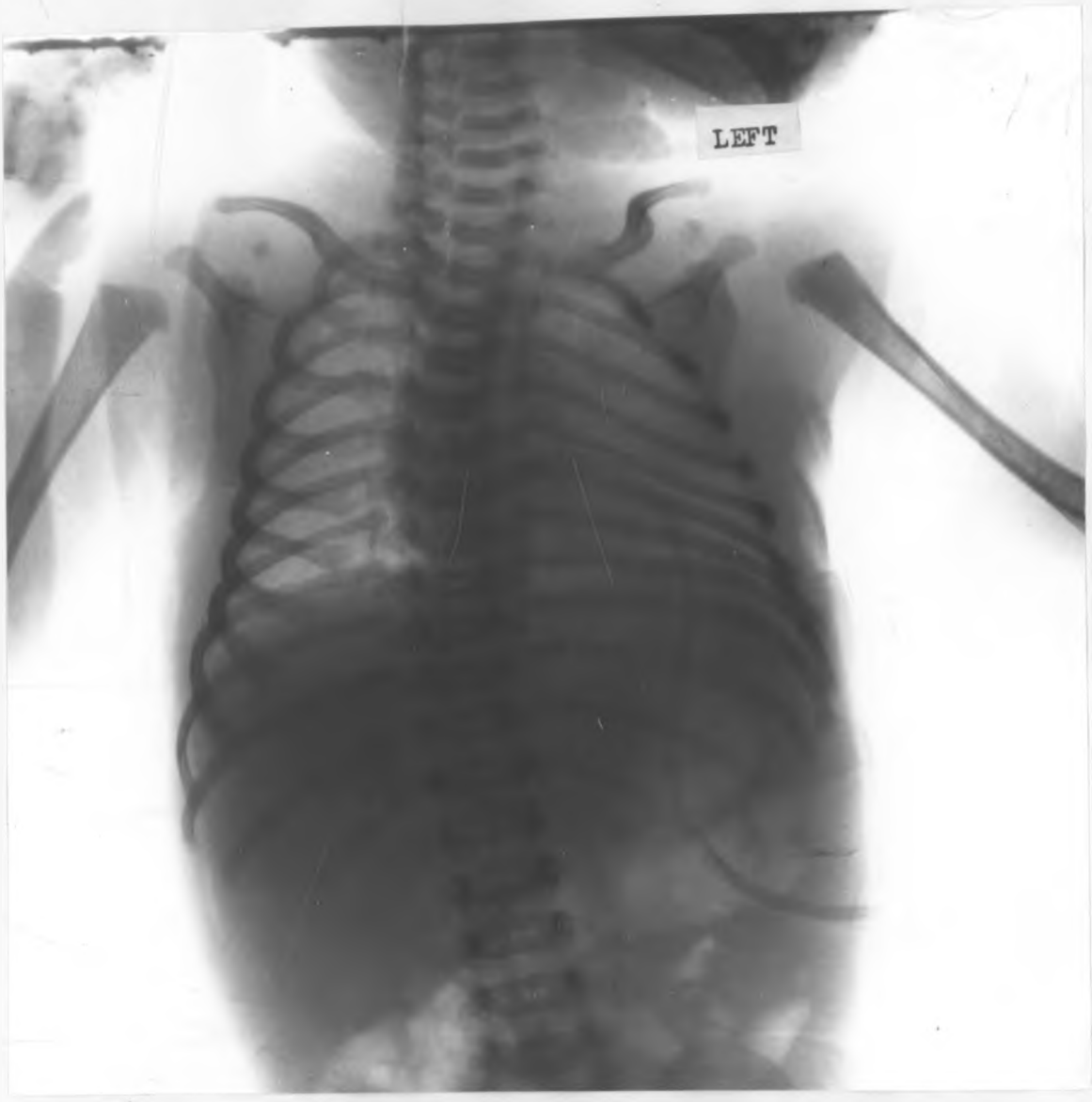
Comments:

The foetal distress was not obvious in this patient, but with eclampsia and heavy maternal sedation the risks were very high.

Suction before delivery of the chest was not possible due to the position of resuscitation equipment, which was outside theatre.

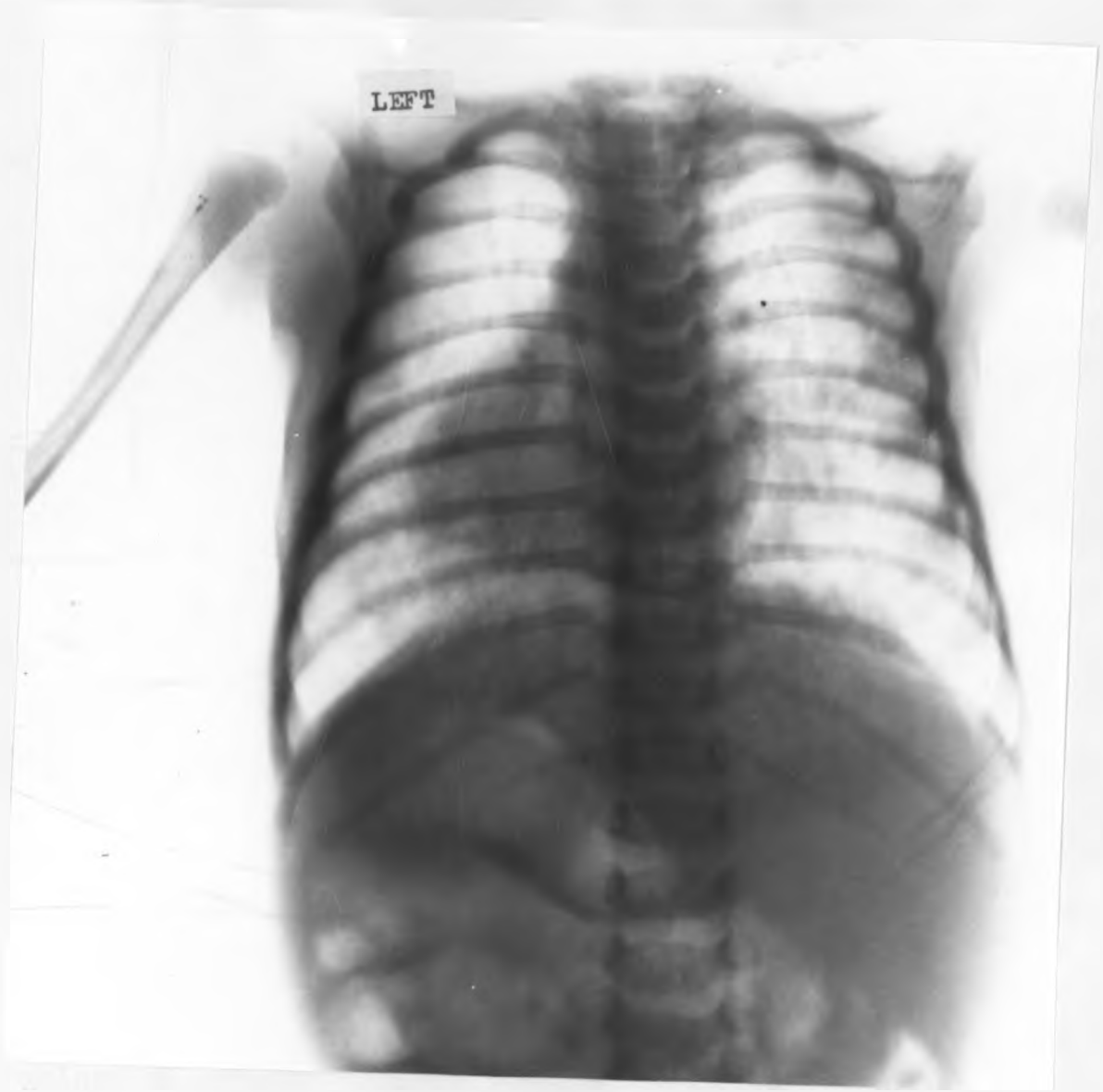
The child was severely depressed. This could be due to the maternal medication or foetal hypoxia or both. The resuscitation which was carried out was fairly vigorous as required but there was no pressure gauge on which to monitor the pressure of oxygen given apart from the rate of flow. Hence the pneumothorax could have resulted from over-inflation of the lungs and aggravated the respiratory failure.

FIGURE I




Chest X-ray showing a left lung opacity. There is hardly any area of aeration on the left. Note the overcrowding of ribs on same side.

FIGURE II




A chest X-ray showing bilateral patchy coarse opacities.

## D I S C U S S I O N

The incidence of meconium stained amniotic fluid in this study was found to be 12.7% as shown in Table I, and the incidence of meconium aspiration syndrome was 0.82% of deliveries. These figures fall withing the range given in previous reports by different workers. Gregory et al (3), in a prospective study, found an incidence of 8.8% for meconium staining and 1.6% for meconium spiration syndrome; Carson et al (4), in a retrospective study, found the incidences of 12.5% and 1.9% for meconium staining and meconium aspiration syndrome, respectively. The lowest figures previously quoted were by Teh et al (9) who gave 0.6% as the incidence of meconium aspiration syndrome. Still more variations are found in other reports but they all fall in the range given before of 8-29% for meconium staining of amniotic fluid and 1-3% for meconium aspiration sydrome, respectively, (1,5,8,24).

This is a high incidence of the condition and deserves preventive measures to be taken so as to reduce the morbidity and mortality. There can be much done to reduce the incidence of meconium aspiration syndrome as shown by Ting and Brady (24).

Meconium passage in utero has, traditionally, been regarded as a sign of foetal distress (1.2.3.5.7.25.26). However, Abramovici et al (17) and Krebs et al (18), disputed this when they did not find a significant difference between the scalp blood pH of infants with meconium staining from that of infants without meconium. Actually some 98% of infants with meconium stained amniotic fluid may have normal scalp blood pH (27,28).

Gregory et al (3) and Miller et al (7) correlated the Apgar score with meconium passage in utero. They found a significantly lower mean Apgar score in the infants who had passed meconium in utero than the controls. Table II and III show similar findings in this study; the infants who have passed meconium utero had significantly lower mean Apgar score than those who had not passed meconium. The non-

Meconium stained infants recovered much more quickly as seen in Table IIIb. Note that 88.8% of those who developed meconium aspiration had an Apgar score of 6 or less at 1-minute as seen in Table IV. This low Apgar score indicated intrapartum compromise (3,5). Meconium staining should therefore be regarded as a warning sign, but there is need for more parameters to exclude or confirm foetal distress.

Foetal heart rate pattern is an important parameter for the assessment of the foetal well being. Studies have been done to correlate foetal heart rate, pH of scalp blood and Apgar score at birth (7,18,28,29,30). Table VII, shows a significantly higher incidence of abnormal foetal heart rates in the meconium stained group than in those without meconium staining, $X^2 = 12.76$ $P < 0.05$. This strengthens the fact that infants with meconium passage in utero may be suffering from hypoxia. In this study intermittent auscultation was used to monitor the foetal heart rate. This does not give as good results as continuous foetal heart rate monitoring since early deceleration patterns may be missed. The diagnosis of foetal distress may be delayed (29). The combination of meconium passage and an abnormal foetal heart require an immediate review of the patient. If scalp blood pH can be done then it should be helpful in decision making as to what method of delivery should be selected (28,30,31,32,33)

Prevention of meconium aspiration syndrome is very important since it is associated with a high mortality rate (4). The measures taken have been quite variable and, so were their effectiveness. These evolve around foetal monitoring and removal of meconium from sites on the infants where the meconium can easily be aspirated. Fox et al (6) recommend direct visualization of the trachea, with a laryngoscope, and suction of all infants with meconium stained amniotic fluid, irrespective of the consistency of meconium and its presence or absence at the vocal cords. This is then followed by Oxygen either by mask or by positive pressure ventilation.

The infant is then put in a Trendelenberg position and percussion of the chest done with intermittent oral and nasal suction. In their prospective study using the same method above on 35 infants whom they diagnosed as having meconium aspiration syndrome, the mortality rate was reduced to 0, while the morbidity was markedly reduced. They however, do not give the incidence of meconium aspiration nor the total number of infants who had meconium-stained fluid.

Other workers, (2,4,5,24) however, recommend suctioning of the nose, mouth, and nasopharynx before and after delivery of the chest. This is followed by direct visualization of the vocal cords and, if meconium is seen, the trachea is suctioned. This method was found more feasible than that of Fox et al (6).

In this study, tracheal suction was done in 44 (30%) of all infants who had meconium stained amniotic fluid, as shown in Table V. Out of these 23 (52.27%) had meconium in the trachea. This figure agrees with the findings of Gregory et al (3) who found that 56% of those infants with tracheal suction had meconium in the trachea. In the same prospective study they found that 20% of the suctioned infants developed meconium aspiration syndrome. In this study a comparative figure of 18.1% was found, Table VI. These figures indicate that even with tracheal suction meconium aspiration syndrome may not be completely eliminated. As shown by other workers (4,5,5,24), suction considerably reduces the incidence and severity of meconium aspiration. In some instances, the mortality has been reduced to zero (4,6) by suction of the trachea.

Looking at the results in another way, it is seen in Table VI that 8 out of 9 (88.9%) infants who developed meconium aspiration syndrome had tracheal suction. For this occurrence Ting et al (24) found 100% of the infants who developed meconium aspiration had tracheal suction. As seen in Table VI

there is a significantly higher incidence of aspiration in the suctioned infants than those not suctioned. This contrary finding could have arisen as a result of:

(a) Intrauterine compromise leading to a low Agpar score and requiring vigorous resuscitation - Note that 88.8% had Apgar scores of 6 or less at 1 minute. Gregory et al found 13 out of 16 (81.25%) infants who developed meconium aspiration syndrome had Apgar scores of 6 or less at 1 minute.

(b) 5 infants gasped before suctioning was started and this could have rendered subsequent suctioning ineffective.

(c) Intermittent positive pressure was done, in some of the infants, before the tracheas were thoroughly cleaned, probably pushing the meconium into deeper airways.

(d) Some of the severely depressed infants could have aspirated in utero, (19,33). It is worthwhile noting in Table VII the high incidence of abnormal foetal hearts in the meconium stained group and those who developed aspiration (7). It was impossible in this study to do endotracheal intubation or nasopharyngeal and oral suction before delivery of the thorax. It is also very difficult to carry out routine tracheal suction in every infant with meconium staining, as was done by Fox et al (6), in a situation where there are very few experienced clinicians who can intubate neonates.

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Foetal distress is a very common indication for caesarean section in Kenyatta National Hospital. Tables VIII and IX, compare the methods of delivery and indications for caesarean section, between the meconium stained group and those without meconium staining. There was a significantly higher rate of caesarean sections in the meconium stained

group than in the non-stained group (P<0.05). 53.5% of these caesarean sections had indications of meconium staining, with abnormal foetal heart rates (23.5%), and normal foetal heart rate (30%). So 30%, of caesarean sections in the meconium staining group were done without any other indication of foetal distress other than presence of meconium. This number, and even those done with abnormal foetal heart rates, could be reduced if continuous foetal heart rate monitoring and scalp blood pH were done (27-32). The number with meconium aspiration syndrome was too small to work out the influence of method of delivery to development of meconium aspiration.

In Table X, it is seen that there was a difference in the mean weight between the infants who passed meconium in utero and those infants who did not pass any meconium in utero. Those who passed meconium had a higher mean birth weight of 3084±500 gms while the mean weight for those without meconium staining was 2960±578 gms. However, this difference was not statistically significant. But looking at Table XI, it is found that there were far more infants who were 36 weeks or less in the group without meconium than those who had passed meconium. Similar results are shown in Table XII, which shows gestational age by physical examination. There were 10 pre term infants among the meconium stained group as compared to 23 infants in the group without meconium staining, giving a statistically significant difference - ($X^2$=7.7 P<0.01). These findings agree with previous workers who described the meconium stained infant as usually a term or postterm baby. 93.5% of the infants were of getational ages of 37 weeks or more. This is similar to the findings of Mathews and Warshow (34) of 98.4% of the infants with meconium staining were 37 weeks or more. There was a higher incidence of postmaturity in the meconium stained infants (13.6%)as compared to 4.3% among those without meconium staining. The passage of meconium in utero has been associated with term or postterm infants by many workers and it is stated that passage of meconium very rarely occurs

before the gestational age of 34 weeks, (3,5,15,34,36).

Meconium aspiration had a very high mortality rate of 44.4%, as shown earlier. This is much higher than the rate of 28% reported in earlier studies (4,6,25). But when there were signs of respiratory failure the death rate increased to 62% (5,25). Three of the infants, in this study, who died, had respiratory failure due to airway obstruction. This high mortality rate in Kenyatta National Hospital should be viewed with great concern at the time when other studies have shown the possibility of reducing this to zero (3,6,24). It should, however, be noted that the actual number of babies who died is small.

## C O N C L U S I O N

Meconium staining of amniotic fluid during labour is a warning sign of foetal distress. This puts the infant into a high risk group and requires more intensive monitoring. Preferred methods of monitoring are continuous foetal heart rate pattern recording and scalp blood pH, when there is an alteration in the foetal heart rate pattern.

The incidence of meconium staining in Kenyatta National Hospital is the same as that reported by other workers elsewhere, but the symptomatic meconium aspiration syndrome found in this study was lower than previously seen in other areas. It is possible, however, that one or two infants did not exhibit any clinical signs of respiratory distress which would have prompted the investigator to do a chest X-ray. In some studies chest X-rays have been taken for all infants who had meconium staining irrespective of the clinical state of the infant (3,10). This could not be afforded.

The tracheal suction done was not preventive, seeing that a large number who developed aspiration syndrome had been suctioned. The explanation was that, most had severe intrauterine asphyxia, gasped before tracheal suction was done and there was a delay in initiating the tracheal suction in some. The resuscitation equipment is kept outside theatre and so suction could not be done immediately in the babies delivered by caesarean section.

There was a fairly significant correlation between meconium passage, abnormal foetal heart rate and low Apgar scores. The foetal heart abnormality in a meconium stained pregnancy should be used as a sign of foetal distress. However, there were very many caesarean sections done because of meconium staining and abnormal foetal heart rate. This situation needs review using continous foetal heart rate monitoring and/or scalp blood pH.

## RECOMMENDATIONS

(1) All infants who have passed meconium in utero should have:-

(i) oral and nasopharyngeal suction before chest delivery;

(ii) direct visualization of the vocal cords and suction

(iii) Tracheal suction if meconium is seen at the cords,

(iv) Paediatric follow up for at least 24 hours.

(2) Resuscitation equipment should be very near the place of delivery and both the Obstetrician and the Paediatrician should be involved in resuscitation.

(3) Mechanical ventilators and pressure gauges on oxygen flow route should be handy, for use in those infants who develop respiratory failure.

(4) Continous foetal heart rate monitoring should be initiated in Kenyatta National Hospital, at least for the infants at high risk.

(5) Another study to follow up radiological changes in infants with meconium staining could be carried out. This should involve serial chest X-rays on all infants with meconium staining and can uncover those with asymptomatic meconium aspiration.

## A C K N O W L E D G E M E N T S

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APPENDIX III

PARTOGRAM.

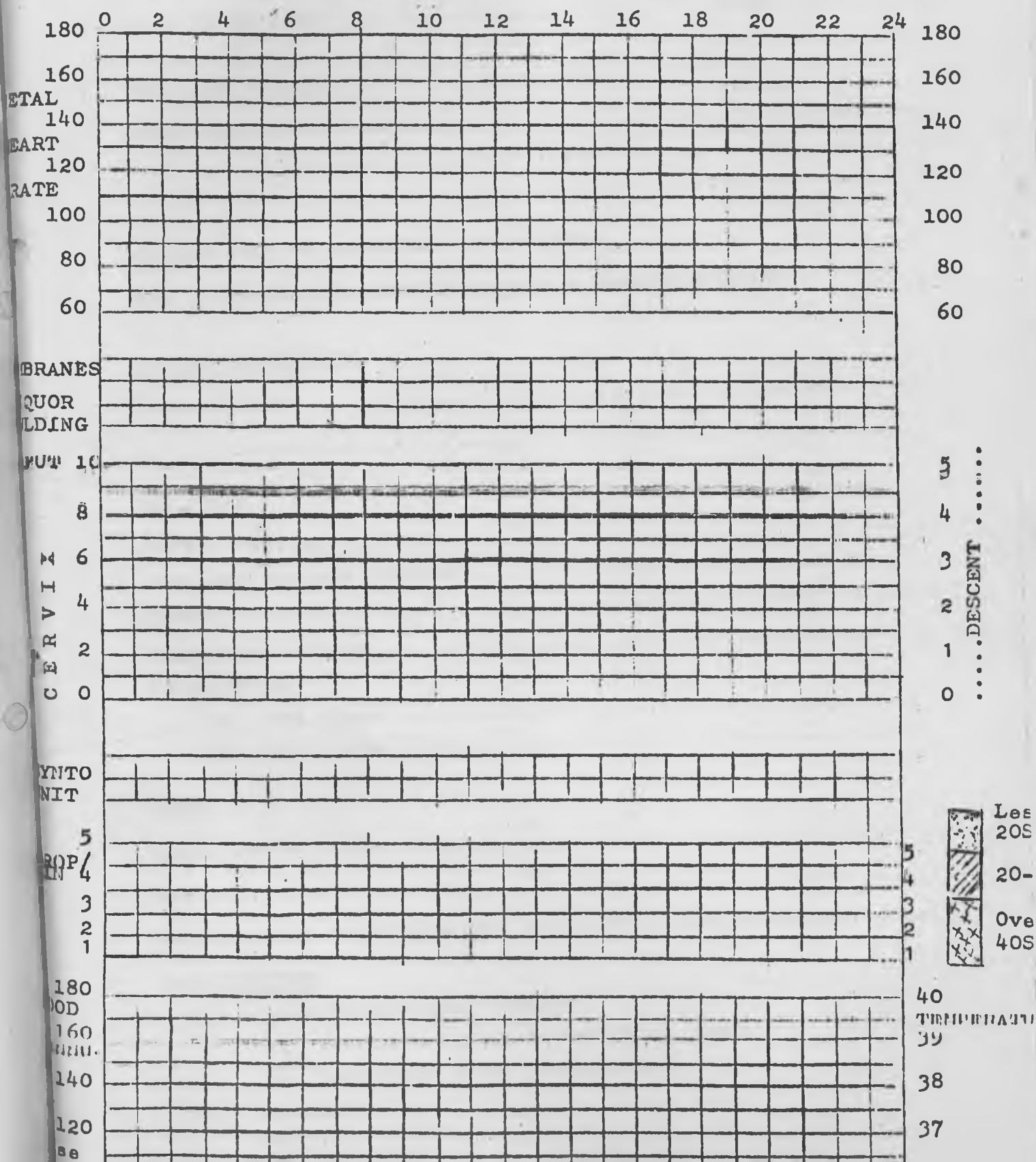
OBSTETRIC UNIT

DR. R. MAKOMBA

OBS. NO.....NAME..... LABOUR STARTED ON..

TIME ADMITTED IN LABOUR (ZERO HR).....MEMBRANES RUPTURED AT.....

AM/PM. ON.....



Appendix II

Dr. R. Makomba

## MECONIUM STAINING

Name of Mother.....Age.....

L.M.P.....E.D.D.....PARITY.....

A.N.C. - YES/NO Abnormalities .....

Date of Delivery .....Time.....

Meconium noticed

Hours before delivery.....

.....

.....

Consistency of meconium - thick/thin

Any signs of infection in mother - fever/discharge

FOETAL MONITORING

Foetal heart .....

.....

DELIVERY

Normal vaginal .....

Assisted vaginal (vacuum).....

Breech .....

Caesarean section.....

Duration of:-

(a) 1st stage .....

(b) 2nd stage .....

BABY

Apgar Score .....

Weight.....

Meconim seen on skin/nails, umbilicus, nose/pharynx/trachea.

RESUCITATION

(i) Mucus Extraction - YES/NO

(a) before or after delivery of chest

(b) before or after first breath

(ii) Intubation

(a) for suction

(b) for oxygen

(c) before or after first breath

(iii) Positive Pressure respiration - YES/NO

Duration .....

CLINICAL ASSESSMENT

wt. .....gm

Head circumference .....

Chest circumference.....

Maturity - by dates .....weeks

by examination .....weeks

Respiration:-

(a) asymptomatic meconium staining YES/NO

(b) symptomatic meconium staining

(i) Clinical signs:-

Respiratory Rate - Temperature.....

Retractions -

Grunting -

Gyanosis -

Crepitations -

(ii) Onset after delivery -

(iii) Duration of signs -

(iv) Related or complicating factors:-.....

.....

Investigations:-

Chest X-rays .....

.....

PH.....

$pO_2$.....

$pCO_2$.....

$SO_2$.....

B.E.....

P.C.V.....%.....

MANAGEMENT

.....
.....
.....
.....
.....

CLINICAL COURSE

.....
.....
.....
.....
.....

Died:- .....

(i) Clinical cause

(ii) Postmortem report.....

.....

.....

.....

.....

.....

COMMENTS:

APPENDIX III

MECONIUM ASPIRATION SYNDROME STUDY

Labour Ward Staff

(1) Please inform Dr. Makomba or the Paediatrician on call as soon as a mother with meconium stained amniotic fluid is admitted or seen anytime the membranes rupture.

(2) Foetal heart auscultation to be done every 30 minutes and recorded on the partogram.

(3) If foetal heart rate is abnormal record every 15 minutes, (Abnormal FHR is the one less than 120 or more than 160 per minute or irregular beats).

(4) Paediatrician to be present during delivery and resuscitation.

(5) Apgar Scoring and resuscitation to be done by Paediatriacian.

(6) Paediatrician:-

(a) All infants with Apgar Score of 6 or less or with thick meconium on body should have tracheal suction.

(b) Those who are not having spontaneous respiration or not making an effort to breathe, should have endotracheal intubation immediately, suction and oxygen. If oxygen is given by Intermittent Positive Pressure Ventilation (I.P.P.V.) do not exceed pressure of 25cm $H_2O$. Remove the tube when the baby is breathing spontaneously.

(c) Other infants do nasopharyngeal and oral suction only.

(d) Record all the procedures and observations taken and time when they were done or observed.