MECONIUM STAINED LIQUOR-REAPPRAISAL

by

T. KAUR,* M.B.,B.S., D.G.O.

and

A. K. DEBDAS,** M.D., M.R.C.O.G. F.R.C.S.

Introduction

Meconium is a hundred per cent foetal product and is not normally passed by the fetus during intrauterine life. However, the question arises when the fetus happens to pass it before birth in presentations other than breech and it is astonishing to find in the literature that the passage of meconium in the liquor has been described from as trivial as physiological defeacation in uterus (Fenton and Steer, 1962) to as serious as to cause 9% perinatal mortality (Walker, J. 1959).

The knowledge of existence of such magnitude of difference of opinion was the object behind taking up of the present study.

Material and Methods

The present study consists of retrospective analysis of 400 consecutive cases of meconium stained liquor delivered at Telco Maternity Hospital during the two years period from January 1976 to December 1977.

Excluded from the study were cases of breech presentation, multiple pregnancy, eclampsia, concealed accidental haemorrhage, gross congenital abnormality, birth weight under 1750 Grams, Rhesus isoimmunisation and also those of uncertain maturity. This was to assess the real impact of meconium staining alone on the state of baby at birth and its perinatal mortality uninfluenced by other adverse factors which can directly affect these.

In our unit meconium is classed as 'thick' only when copius particulate meconium is seen on the pad. All other cases of staining are classed as 'thin'.

All babies have Apgar scoring done at one minute after birth. Percentile weight of babies were calculated according to the chart prepared by Kavoor *et al* (1977).

Of the 400 cases, in 352 (88%) meconium was present at the time of rupture of membranes be it spontaneous or artificial and whether amniotomy was performed to induce or to expedite labour. In the rest 48 cases (12%) the liquor was recorded as clear at rupture but noted to be meconium stained later.

Besides above, 200 consecutive cases with clear liquor irrespective of clinical condition but with above exclusions delivered during part of the above period, were also collected from records to serve as control cases. The duration of labour of these patients were also comparable with those of meconium group, as roughly 90% patients of each group delivered within 12 hours and 98% within 18 hours.

*Senior House Officer,
Dept. of Obstetrics and Gynaecology, St. Mary's Hospital, Isle of Wight, England.
**In-charge,
Department of Obstetrics and Gynaecology, Telco Maternity Hospital, Jamshedpur-831 004.
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However, both caesarean section rate (6.25%) and the combined forceps and vacuum extraction rate (8.5%) were treble in meconium group in comparison to control group (2% and 3% respectively).

Results and Analysis of Data

Correlation of Meconium Staining with Apgar Score

A comparison has been made in Table I between the meconium and control group (with clear liquor) on the 'one minute' Apgar score. The most striking thing that noticeable in Table I is that as

TABLE I

Comparing the 'one minute' Apgar Score of Meconium group with that of control group

Apgar Score	Meconium group No. of cases (%)	Control group No. of cases (%)
7 to 10	359 (89.75%)	192 (96%)
4 to 6	26 (6.50%)	7 (3.50%)
0 to 3	15 (3.75%)	1 (0.50%)
	400 (100.00%)	200 (100.00%)

high as 89.75% of babies were born in excellent condition (Apgar 7-10) inspite of their passing meconium in utero. Only 6.5% of babies of meconium group scored 4-6 and mere 3.75% scored 0-3. Although, on its own this is very good result, it would not appear to be so if compared with those of control group, because, as is evident from the Table, babies with clear liquor fared even better -96% scoring 7 to 10 and only 0.5% scoring 0-3.

In Table II an attempt has been made to correlate the quantity of meconium passed by the fetus in the liquor with the Apgar score at one minute.

TABLE II

Correlation of Quantity of Meconium in Liquor with Apgar Score

Apgar Score	Thin meconium No. of cases (%)	Thick meconium No. of cases (%)
7 to 10	302 (95.87%)	57 (67.05%)
4 to 6	11 3.49%)	15 (17.64%)
0 to 3	2 (0.63%)	13 (15.29%)
	315 (99.99%)	85 (99.98%)

Out of 400 cases, liquor of 315 babies (78.75%) were thinly meconium stained and that of 85 babies (21.25%) were thickly stained.

It is evident from Table II that whereas with thin meconium staining as high as 95.87% of babies were born with Apgar 7-10, with thick meconium only 67.05% were born in such excellent condition. Table II also shows that while with thin meconium only 2 babies out of 315 (0.63%) were born with Apgar 0-3, the percentage of babies born in such severely depressed state in thick group was 24 times higher (15.29%). Even in Apgar 4-6 group there was much higher percentage (17.64%) of babies with thick meconium than those with thin meconium (3.49%). Hence the conclusion that can be drawn from Table II is that whereas just yellow staining of liquor without solid bits of meconium in it can be practically harmless, presence of this latter factor can make it extremely dangerous.

Correlation of Birth Weight with Passage of Meconium in Utero

Table III correlates the distribution of percentile birth weight of the babies with meconium stained liquor with that of the babies of control group with clear liquor.

It is apparent from the Table that there was practically no difference in the percentile weight distribution between 10th

TABLE III

Percentile Weight Distribution of Meconium and Control Group

Percentile weight	Meconium group No. of cases (%)	Control group No. of cases (%)
Above 90	40 (10.00%)	28 (14%)
61-90	119 (29.75%)	56 (28%)
41-60	52 (13.00%)	26 (13%)
10-40	134 (33.50%)	74 (37%)
Below 10	55 (13.75%)	16 (8%)
	400 (100%)	200 (100%)

and 90th percentile among the meconium and control group —76.25% of former and 78% of the latter group falling in this range. However, there was much higher proportion (13.7% as opposed to 8%) of small for date babies (weight below 10th percentile) in the meconium group comparison to control group. The lightest baby of the present series was 1.9 Kg.

Meconium Staining and perinatal mortality

There were only 12 perinatal deaths amongst 400 cases with meconium staining of liquor—a perinatal mortality rate of 3%.

Of the 12 deaths, only 1 was associated with thin meconium and all the rest with thick meconium. All the 12 babies were born with very poor (under 4) Apgar score and 1, in fact, was stillborn (Table IV).

TABLE IV

Apgar Score at One Minute and the Intensity of Meconium Staining of Liquor in the Twelve Cases who Suffered Perinatal Death

Apgar score	Thin meconium	Thick meconium	Total
0	nil	1 (stillborn)	1
1	nil	nil	nil
2	nil	6	6
3	1	4	5
	1	11	12

Nine out of the 12 cases (75%) of the perinatal death developed heart signs (on ordinary auscultation) in addition to meconium staining of which 7 had bradycardia with irregularity and 2 had persistent profound tachycardia.

The cause of death of the baby who was stillborn was acute intra-uterine hypoxia in labour as evidenced by its deep aspiration of thick meconium. The baby was not light for date and was otherwise healthy.

The cause of death in all the 11 babies who died was meconium aspiration syndrome in perfectly healthy babies except 1 who was also light for date (below 10th per centile). The point of significance is that all the 12 fatal cases aspirated meconium and in all but 1 the factor aspiration directly contributed to the perinatal death.

Discussion

The suggested causes of meconium passage from review of literature have been shown in 'The chart' (see 'The chart). It is evident from the chart that meconium may be passed by the fetus not only as a result intra-uterine hypoxia but also due to various non-hypoxic causes. We have attempted to reappraise the whole situation in four broad headings.

Meconium and Various Obstetric Conditions

While Walker (1959) and many other workers found rising incidence of meconium staining with post-dated pregnancy, antepartum haemorrhage, pre-eclampsia supporting the hypoxic theory, there are others who did not have quite the same experience (Wood and Pinkerton, 1961). This inconsistancy is further strengthen-

ed by the finding of only 25% (1 in 4) meconium staining rate amongst anoxic stillbirth cases (Wood and Pinkerton, 1961).

Although posmaturity has much closer association with meconium passage (Bacsik, 1977), it is still to be established whether the factor behind is hypoxia or attainment of fetal gut maturity (see chart). The findings of the present series that 90% of our meconium stained babies were born in excellent condition and that only 3% of all suffered perinatal death, although as high as 57% of them were post dated, would support more the maturity theory than hypoxic theory of meconium passage.

We found growth retardation to be an uncommon association of meconium passage being present only in 13.75% cases. This is supported by the finding of Gregory *et al* (1974) who found the average birth weight of meconium stained babies to be 2911 Gm.

Meconium and Heart Signs of Fetal Distress

Stage of meconium passage has been described as a state of 'compensated fetal distress' during which the homeostasis of fetus is maintained by the 'diving reflex' as explained in the chart and, it has also been suggested that, only if stressed beyond its ability to compensate, the heart rate abnormalities appear (Abramovici *et al*, 1974). The fact that nearly 60% of those babies who suffered perinatal death in our series showed gross heart signs of fetal distress can be taken as a proof for the above theory.

Meconium and Neonatal Asphyxia

Whereas neonatal asphyxia rate (Apgar 6 or less) with meconium stained liquor in the present series was 10%, Abramovici *et al* (1974) found no correlation between the two (provided the labour was not prolonged). Bacsik (1977) summarised the respiratory distress rate of meconium stained babies and found it to range between 10 to 30 per cent.

It is relevant to mention here that whereas the neonatal asphyxia rate in our series for thin meconium was only 5% it was as high as 33% with thick meconium —a finding which is in agreement with the finding of White (1955) who found the latter figure to be 42%.

Meconium and Perinatal Mortality

While Walker (1959) recorded a perinatal mortality of 9% with meconium stained liquor this was only 3% in our series although, admittedly, our exclusions (see before), our short labours and smaller proportion (20%) of thick meconium cases contributed towards it. It is noteworthy, however, that for babies with Apgar 3 or less our perinatal mortality was 40 times higher with thick meconium in comparison to thin meconium and 11 out of our 12 babies lost passed thick meconium. This finding have the support of the observations of Bacsik (1977) who pointed out that the mechanical airway obstruction by meconium was the most important factor in bringing harm to the baby.

Conclusion

Association of meconium passage with chronic intrauterine hypoxic or hyponutritional state is neither constant nor frequent signifying that meconium in large majority of these cases, specially around term, is probably passed as a result of attainment of fetal gut maturity rather than due to above factors.

When, however, hypoxia is the cause of meconium passage, this being pathological hence a stronger stimulus, lot of meconium is passed and as these cases are often associated with scanty liquor the mixture becomes really thick.

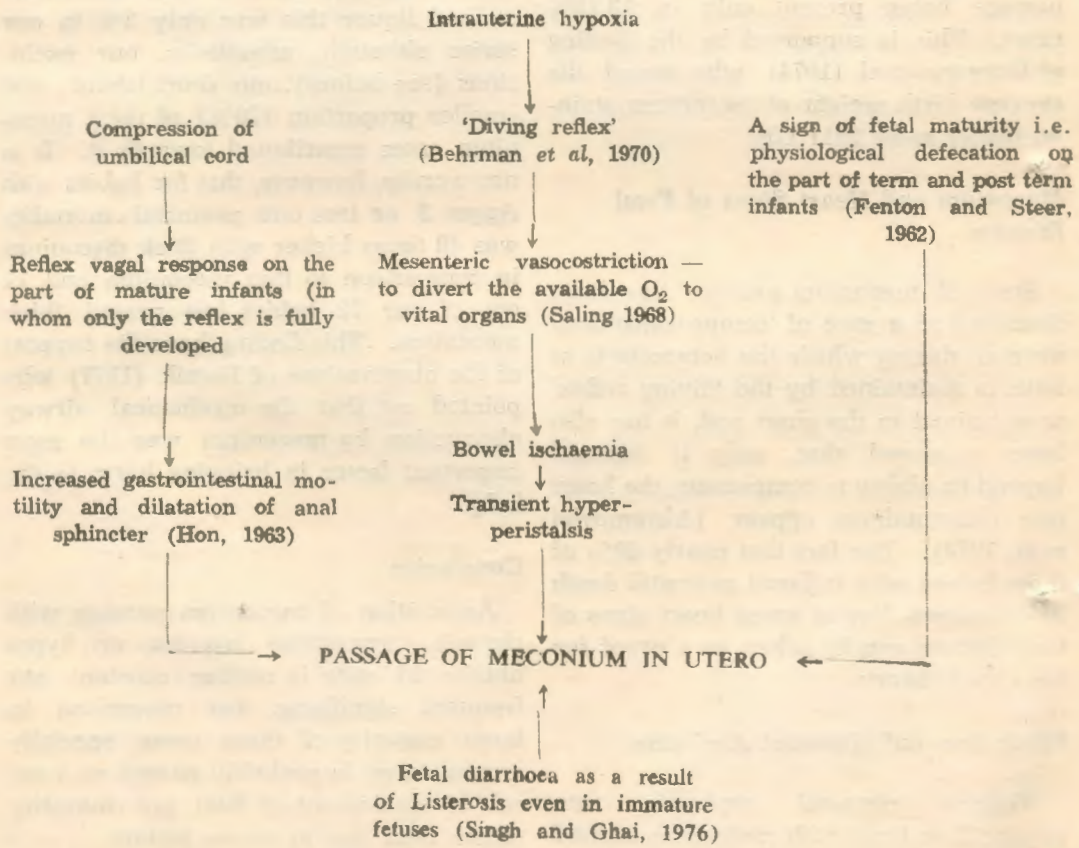
However, as shown by the present study, meconium is not really dangerous unless it is thick and until it is inhaled. Hypoxia plays its most important role by promoting the latter which generally happens in labour (due to further hypoxia) and hence lies here the prevention spot.

Mutually re-enforcing triple role of hypoxia cannot be over-emphasised.

The Chart: Causes of passage of meconium in liquor

It is apparent from the chart that meconium may be passed by the fetus due to four basic causes of which only one is hypoxia and the rest are non-hypoxic. However, as it is the hypoxic factor that has always remained the main source of worry, various workers from time to time have sought clinical correlation to resolve the doubts regarding its real role.

Walker, J. (1959) found rising incidence of meconium staining, with increasing maternal and gestational age, specially past term. He also found high incidence of the same with antepartum haemorrhage and pre-eclampsia and thought that in all these conditions oxygen deprivation due to deterioration of transplacental oxygen transfer is the cause of meconium passage. Desmond *et al* (1974) found no ever, Abramovici *et al* (1974) found no



(1957) also had the similar experience and drew similar conclusion but added anaemia and chronic pulmonary disease in the list. However, the findings of Wood and Pinkerton (1961) was to the contrary on this matter for cases of pre-eclamptic toxaemia, hypertension and antepartum haemorrhage. Besides, the fact that only 1 in 4 cases of anoxic still-birth show meconium in liquor (Wood and Pinkerton, 1961) and that even acute hypoxic intra-uterine death sometimes occurs without passage of any meconium (Macafee and Bancroft-Livingston, 1958; Morris and Beard, 1966) also would stand against the cause and effect relationship suggested by Walker, J. 1959).

Now a closer look at the effect of post-maturity on meconium passage. Although review of literature (Walker, J., 1959, Bacsik, 1977 and many other workers) establishes it well that there is a close relation between the two, the reason for this is still not quite clear. Hence, an Obstetrician is left to speculate as to whether it is the hypoxia, as a consequence of dwindling placental function near, at or beyond term (Walker, J., 1959) or whether it is just the attainment of maturity on the part of the fetus (Fenton and Steer, 1962) that is responsible for the occurrence of this age related defecation. The findings of the present series that 90% of babies of our 400 patients with meconium stained liquor were born in excellent condition and that they suffered a perinatal mortality of only 3%, although as high as 57% of them were carrying post dated pregnancy and even the rest—the pregnancy of minimum duration of 258 days, would all go more in favour of non-hypoxic rather than hypoxic theory of meconium passage.

About the association between meconium and growth retardation syndrome Morris (1968) commented—"passage of meconium would not be expected in the small for dates syndrome in the absence of asphyxia". In support of this he reported only a 10% incidence of meconium staining amongst his series of 240 high risk cases. The present series which looks at the problem from the opposite angle also suggests that growth retardation is rather an uncommon accompaniment of intra-uterine meconium passage since as high as 86.25% of our babies with meconium stained liquor were not growth retarded (weight under 10th percentile). In fact, Gregory *et al* (1974) found the average birth weight of meconium stained babies to be 2911 Gm. which is quite remarkable. There were, however, in the present series, nearly double (13.75%) the proportion of growth retarded babies in the meconium group in comparison to contral group (8%).

So, the possible conclusion that the above review lends is this that although the meconium passage may be associated with chronic intrauterine hypoxic or hypo nutritional state, the association is, by no means constant or frequent raising the possibility of existence of some other cause of meconium passage in addition to hypoxia.

Now the effect of meconium passage on neonatal asphyxia and perinatal mortality rate. White (1955) noted that as high as 33% of all cases with meconium stained liquor and 42% of those who pass thick meconium require resuscitation. However, Abramovici *et al* (1974) found no correlation between the appearance of meconium with fetal acidosis and low Apgar score provided the fetus was delivered within reasonable time. The fact that nearly 90% of babies of present series were born with Apgar 7-10 would support the findings of Abramovici *et al*

(1974). But with passage of thick meconium however, also in our series, the proportion of babies born in moderately and severly depressed state increased many fold—the finding which is in agreement with that of White (1955).

Walker, J. (1959) and also Walker, N. (1959) both found perinatal mortality of 9% with meconium stained liquor. O'Driscoll *et al* (1977) commented that "meconium or no liquor marks the fetus who may suffer death or brain damage". However, observations of Macafee and Bancroft-Livingstone (1958), Fenton and Steer (1962) and Abramovici *et al* (1974) were by no means so gloomy. But the last group of workers have put forward the interesting suggestion that meconium passage represents a state of "compensated fetal distress". During this precarious state, according to them, the homeostasis of fetus is maintained by the so called "diving reflex" as explained earlier. These authors also go on to explain that—only if stressed beyond its ability to compensate the heart rate abnormalities and/or acidosis appear.

Our results though fall in line with those of second group of workers need some explanation as to why it is so superbly good. The contributory factors for this, in our opinion, were our exclusion of all the common conditions (as mentioned before) generally associated with high asphyxia and perinatal mortality even in absence of meconium passage, our relatively short labours (hence shorter exposure to the stress) and the great preponderance (80%) in our series of cases with thinly stained (non-particulate) liquor.

In this connection it is noteworthy that the neonatal asphyxia rate (Apgar 3 or less) was 24 times and the perinatal mortality rate was 40 times higher in our series with thick meconium in comparsion to those with thin meconium. In fact, 11 out of our 12 babies lost passed thick neconium. These findings have the support of the observations of Bacsic (1977) who pointed that the mechanical airway obstruction by meconium was the most important factor in bringing harm to the baby.

There is general agreement that appearance of heart signs of fetal distress in a case with meconium stained liquors is particularly dangerous (Fenton and Steer, 1962; Abramovici *et al* 1974). In line with it is our finding of persistent gross fetal heart abnormality even on ordinary auscultation in 7 out of 12 cases of perinatal death.

In the following section an attempt has been made to describe a course of events for meconium associated pregnancies and labours based on review of literature and the results of the present series.

At around term a small quantity of meconium may be passed in utero by a proportion of fetus just due to attainment of physiological maturity of its gut even in total absence of hypoxia and this group constitutes the large majority of cases of meconium stained liquor. Hence babies of this group are not generally depressed at birth and do not have any higher perinatal mortality rate in comparison to those with clear liquor. In these cases the liquor also is generally plenty and is only thinly stained without obvious meconium particle in it.

When, however, the liquor is heavily meconium laden, it is probably most likely that the causative stimulus for meconium passage in such a case had been intra-uterine hypoxia rather than simple gut maturity. This contention is based on the presumption that hypoxia, being a pathological stimulus, is a much stronger

excitor of bowel movement and hence would cause expulsion of a lot of meconium. The other contributory factor towards heavily meconium laden liquor is the low liquor volume which is so commonly associated with placental insufficiency and hence hypoxia. This fact further establishes the association between thick meconium and hypoxia and the consequent high neonatal morbidity and perinatal mortality rate.

Now the more important event meconium aspiration. Obviously, only those fetuses are likely to be adversely affected by the presence of meconium in liquor who, due to some untoward intra-uterine circumstances are compelled to take intra-uterine gasp and thereby inhale the soiled liquor; and, as is well known, intra-uterine hypoxia is the prime cause of this gasp. The conclusion that the above state of events lend is that intra-uterine hypoxia can not only cause meconium passage but, if severe and prolonged, also its inhalation. The other possible cause of intra-uterine gasp is traumatic delivery.

So, the preventive measures that would transpire from the above are—prevention of prolongation and/or further exaggeration of hypoxia, avoidence of traumatic delivery of any kind and prompt clearing of upper respiratory tract of neonate at birth before the onset of its first breath.

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