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Delayed Meconium Passage and Jaundice in Newborn Infants

By

J. Rosta, Zita Makói and Adrienne Kertész

First Department of Paediatrics, Semmelweis University Medical School, Budapest

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The time of the first meconium passage was observed in 1000 healthy full-term newborns. Fifty per cent of the babies produced their first faeces before the 9th hour, 90% before 17 hours 45 minutes, and 97% before the 22nd hour of life. Caesarean section resulted in a significant delay of the first passage of meconium ($P < 0.1$), a phenomenon to be explained with the effect of anaesthetics given to the mother and influencing the nervous system of the foetus. The babies of toxaemic mothers also displayed a retarded meconium passage.

In 742 newborns with a negative obstetric history and no immune-haemolysis, the influence of meconium passage on the course of neonatal jaundice has been investigated. In this group of babies delayed passage of meconium was significantly ($P < 0.01$) correlated with the frequency of pathologic jaundice (maximum serumbilirubin level above 15 mg/100 ml). In two groups of newborns, composed of 43 and 62 cases, respectively, suffering from pathologic jaundice, the first passage of faeces occurred 3 hours later than in a control group of 699 babies.

The importance of enterohepatic shunting of indirectly reacting bilirubin in the aetiology of neonatal jaundice is discussed.

A substantial part of the indirectly reacting free bilirubin produced in the foetal organism passes through the placenta into the maternal bloodstream, then is glucuronized in the mother's liver and excreted with her faeces.

Further quantities of bile pigment accumulate in the foetal gut. As the glucuronization capacity of the foetal liver is minimal, bilirubin reaches the gastrointestinal tract through an alternative pathway, *i.e.* the intestinal mucosa, which is permeable to free bilirubin in both directions [7, 14, 24].

A second source of bilirubin in the gut is the swallowed amniotic fluid which obtains the bile pigment from the foetal lung, the placentar vessels and through Wharton's jelly. This happens even under normal conditions, but in cases where the normal route of excretion of the foetal pigment through the placenta is disturbed, the secondary pathways of excretion are gaining in importance [5, 13, 16].

As the intestinal mucosa is permeable to bile pigments in both directions, some of the pigment may be reabsorbed postnatally into the circulation of the newborn infant. This so-called entero-hepatic circulation or shunting of the indirectly reacting free bilirubin was first observed in Gunn rats and later in

babies suffering from Crigler—Najjar's disease [12, 15].

During the first hours of life there are circumstances such as the low level of serum bilirubin, which are in favour of entero-hepatic shunting. Though several authors have examined the peculiarities of entero-hepatic bile circulation, no data have been found concerning the correlation between the time of meconium passage and the severity of neonatal jaundice.

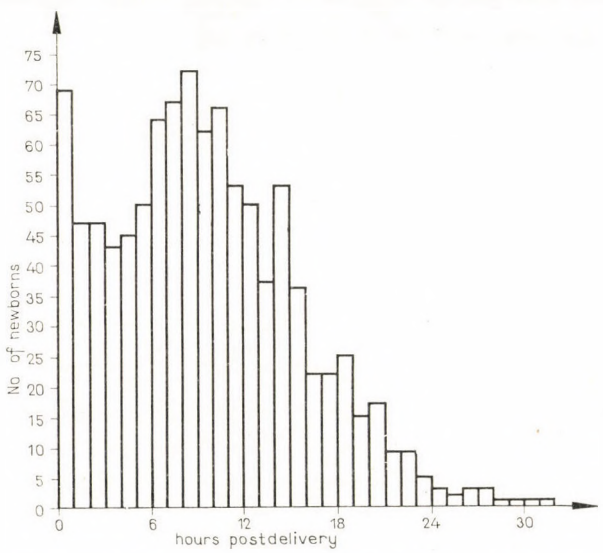

FIG. 1. Time of first passage of meconium in 1000 healthy full-term newborns with a birth weight above 2500 g

MATERIAL AND METHODS

The time of the first passage of meconium was recorded at the Neonatal Unit of our Department and of the Second Department of Obstetrics in 1000 consecutive singletons delivered during the first half of 1966. Babies weighing less than 2500 g were excluded from the study. In cases where amniotic fluid was stained with meconium, the first passage was considered for the first hour of life. If meconium was found in the napkin on arrival to the ward from the delivery room, the first or second hour of life was recorded according to the time of transfer. Further observations were done at regular three hour intervals when the napkins were changed. Data collected from babies kept naked would have been more accurate; lacking the necessary number of incubators we were, however, reluctant to expose newborns to an eventual cold injury.

Meconium found at the time of changing the napkins could have been passed at any time during the previous three hour period. As in correlating the time of appearance of the first faeces with the severity of neonatal jaundice we have considered three hour periods, the method of collecting data did not invalidate the results, which are tabulated in Fig. 1.

Results

The distribution of data except those for the first hour of life, follows the Gauss formula. If the meconium-stained amniotic fluids could have been subdivided according to the precise time of passing faeces *in utero*, the missing left part of the function might be evident.

As our patient material contained no case of gastrointestinal malformation, we regarded our data as featuring normal circumstances in meconium passage and have computed the following percentiles

3rd percentile	34 min
10th percentile	1 hour 20 min
25th percentile	5 hours
50th percentile	9 hours
75th percentile	13 hours 25 min
90th percentile	17 hours 45 min
97th percentile	22 hours

Our observations have supported the earlier ones stressing the influence of toxaemia and operative delivery on the first meconium passage and on bowel movements in general [6, 20, 27, 30]. In a group of babies born by caesarean section, the first meconium was voided significantly later ($P < 0.1$). This postponement might have been caused by drugs administered to the mother and which could influence the central and vegetative nervous systems of the foetus.

Toxaemia and some of the anaesthetics are well known for their effect on neonatal jaundice. Jaundice is appearing sooner than normal and culminating at a lower serum bilirubin level in babies of toxaemic mothers. Premedicated newborns on the other hand are known to be inclined to develop a marked jaundice [21, 22, 28].

In view of these circumstances, babies of toxaemic mothers and those delivered with caesarean section were excluded from the study. Infants with immuno-haemolytic disease were also excluded, their jaundice having been so deeply connected with the haemolytic process itself that further factors such as a delayed passage of meconium could not be judged. After such selection, 742 babies remained from the original 1000 newborns.

Of these 742 babies, 699 did not develop pathologic jaundice; they were used as a control group. In 43 cases where an immune-haemolytic process could not be detected, the maximum serum bilirubin level exceeded 15 mg/100 ml during the first days of life (Group pathologic jaundice I).

In Fig. 2 the correlation between the time of the first meconium passage and the relative frequency of pathologic jaundice (maximum serum bilirubin level above 15 mg/100 ml) is shown. A significant rise in the frequency of pathologic jaundice with the delay in the first meconium passage was obvious.

To extend the studies we selected further 62 babies with pathologic jaundice where other circumstances were analogous to the pathologic jaundice I group. This latter group was called pathologic jaundice II and

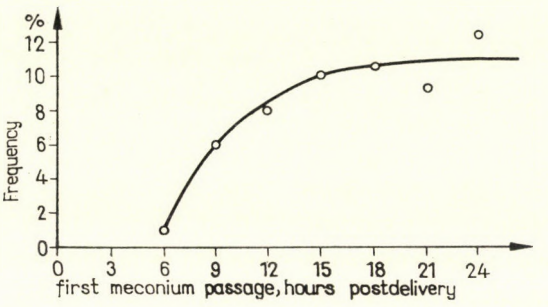

FIG. 2. Frequency of pathologic jaundice (maximum serum bilirubin level above 15 mg/100 ml) and time of passage of the first meconium

was evaluated separately in comparison with the above control group.

In Fig. 3 the percentual distribution of the time of first meconium passage is shown in the three different groups. The majority of cases passed the first meconium at 12 hours of life in the two icteric groups and at 9 hours in the control group.

In spite of the close correlation between the time of meconium passage and the pathologic jaundice, the peak serum bilirubin level was not directly correlated with the time of meconium passage.

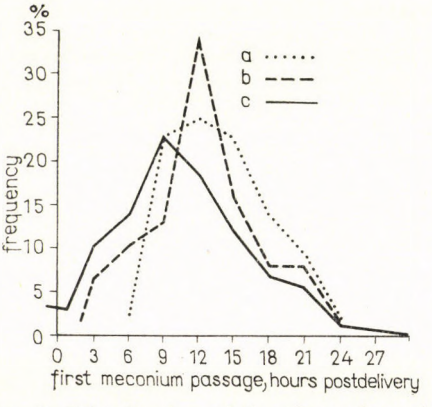

FIG. 3. Distribution of the time of passing the first meconium in three groups of newborn babies: *a)* and *b)*, 43 and 62 cases, respectively, with pathologic jaundice (maximum serum bilirubin level above 15 mg/100 ml) without immune-haemolysis; *c)* control group of 699 babies without pathologic jaundice. Obstetric history was uneventful in all three groups

DISCUSSION

Data regarding the regular passage of meconium are scarce. In SMITH's Physiology of the Newborn [26] the only work cited in this aspect was published in 1934; it deals with the bacteriology of the meconium of 35 newborns and mentions that 32 of them passed the first faeces before the 20th hour of life [8]. More recent textbooks rely on SHERRY and KRAMER's paper [25] including 500 healthy full-time babies. Sixty-nine per cent of them produced the first meconium before the 12th hour and 94% before the 24th hour of life. SCHAFFER's monograph too gives a 24 hour limit for passing the first meconium under normal circumstances [23]. Our own data showed identical results in that 97% of babies born with a birth weight above 2500 g voided their first meconium

before the 22nd hour of extrauterine life.

In cases of congenital atresia and strictures of the gut several observations pointed to the frequency of an increased and/or protracted jaundice. It seems reasonable to correlate at least the early phase of such jaundices with the disturbed meconium passage in the malformed intestines [1, 2, 3, 17, 18].

The beneficial effect of early feeding on the course of neonatal jaundice might also be explained with an enhanced meconium passage and diminished rate of entero-hepatic shunting. This phenomenon was repeatedly observed with early feeding of premature infants whose jaundice culminated at a low serum bilirubin level [9, 10, 31].

The experimental works on enterohepatic shunting and the above mentioned clinical experience have given rise to trials to diminish the reabsorption of bilirubin from the gut by the use of drugs. LESTER et al. fed to Gunn rats cholestyramine, a compound which, by binding bilirubin, could prevent its reabsorption from the gastrointestinal tract. The result was not only a 30—40% fall in the indirectly reacting bile pigment level, but subsequently the clearance of repeatedly injected bilirubin was significantly promoted [15]. It was on the same reasoning that charcoal administration was recommended. This measure, however, was effective only if carried out before the 4th hour of life; after the 12th hour it was unsuccessful [11, 29]. The failure may be explained by the early course of enterohepatic shunting during the first hours of life, when no equilibrium has yet been attained between the intravascular and intestinal pools of bilirubin. The high intestinal beta-glucuronidase level is also contributing to the shunting of pigment in the earliest period of extrauterine life [4].

Our observations are in support of an enhanced indirect bilirubin shunting in cases of delayed meconium passage. The bile pigment accumulated in the foetal gut is contributing in a significant degree to pathologic jaundice if the first passage of meconium is retarded.

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Dr. J. Rosta
Bókay J. u. 53.
Budapest VIII, Hungary