DYSMATURITY

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(RECEIVED FOR PUBLICATION AUGUST 23, 1957)

History

The Scottish obstetrician Ballantyne (1902) seems to have been the first to call attention to dysmaturity. He described the dry, parched skin, the long nails, the paucity of the amniotic fluid, the presence of meconium in amniotic fluid, and the advanced ossification in the skull. He found the weight and length of these children to be nearly always above average.

Bossi (1907) gave a description of dysmature infants which in the main agrees with Ballantyne's, but he maintained that they have a reduced amount of subcutaneous fat and that therefore their weight is lower than might be expected.

Bäcker (1915) described dysmaturity and emphasized that dysmature infants are subnormal as far as fat and weight are concerned.

Then, for some time, no attention seems to have been paid to dysmaturity, until Runge (1939, 1942, 1948) gave a detailed description of the dysmature child. He also indicated a method of recognizing the condition in some cases even before delivery. He called attention to the fact that in some women who eventually are delivered of dysmature children the amount of amniotic fluid and concomitantly the circumference of the abdomen decreases successively before delivery. Runge considers dysmaturity to be a sign of placental insufficiency as regards the transport of oxygen as well as other substances. He made no chemical analyses and his conclusion was probably based on the presence of meconium in the amniotic fluid and the loss of weight of the child.

Although no further major studies have been published, it is obvious that German obstetricians have realized the difference between prolonged gestation and dysmaturity, as is evident from Martius' textbook on obstetrics published in 1943.

In other countries, dysmaturity does not seem to have been recognized until Clifford, in 1945, reported 46 cases; he has more recently (1951, 1953, 1954,

1957) described the syndrome and its clinical importance in detail. Like Runge, Clifford considers dysmaturity to be caused by a placental insufficiency and introduces the term 'placental dysfunction syndrome'. However, he has no further proof that a placental insufficiency is the cause of dysmaturity, but seems to base his conclusions on the same clinical grounds as Runge and finds support in Walker's work (1954) on prolonged gestation. Clifford's papers are of considerable importance because he classified dysmaturity in different stages and because he has made the dysmaturity syndrome known to pediatricians. Consequently, the condition has since been the subject of reports from several countries (Taylor *et al.*, 1952; Selander, 1954; Ravera and Bottinelli, 1955; Kunstadter and Schnitz, 1956; and others).

Present Investigation

Definitions. In this investigation, the duration of pregnancy is given in weeks calculated from the first day of the last menstrual period. Thus, for example, if a child is born in the 40th week, this implies that the pregnancy has lasted between 274 and 280 days. If the gestation time exceeded 294 days, we have called this prolonged gestation or prolonged pregnancy.

If the child showed the clinical syndrome which Clifford (1954) designates as the 'placental dysfunction' syndrome, known in German as 'überreif', we use the expression 'dysmaturity' without regard to the duration of pregnancy.

The term 'postmaturity', which is often seen in the literature, is avoided because some authors use it to denote prolonged pregnancy, others to denote dysmaturity, others again to denote both prolonged pregnancy and dysmaturity.

Material. An investigation has been made of 1,171 newborn infants, all delivered at the Depart-

ment of Obstetrics, University Hospital, Lund, between January 1, 1956, and March 31, 1957.

Only during the later part of the investigation were all children born classified according to dysmaturity. The material was selected, in so far as mostly children with prolonged gestation were included. The selection in these cases was always made before the children were born.

The infants were classified, largely according to Clifford's classification, the day after delivery. This was done by only one of us, who at that time had no information concerning the mother, the duration of pregnancy, the delivery, the amniotic fluid or the placenta. Thus the classification was made only on the basis of inspection and observation of the naked infant.

Normal and premature infants without signs of dysmaturity have been grouped in Stage 0.

Stage 1 corresponds to Stage 1 of Clifford. The skin is cracked, parchment-like and peeling and the arms and legs are thin. The infants are more awake and alert than usual but they may have respiratory distress and a tendency to vomit. The bones of the skull are harder than is ordinarily found.

If the clinical picture just described was less pronounced and there was doubt whether to classify the infants as normal or dysmature, they were placed in Stage 0-1.

Infants who exhibited the signs attributed to Stage 1 in a very marked degree, have been grouped in Stage 2. In addition, the trunk was thin in these cases. In Stage 2, Clifford includes meconium staining of the amniotic fluid, but as the examining doctor did not have any knowledge of the amniotic fluid, this sign was not taken into account in grouping the infants. However, they all had greenish skin, umbilicus or nails. The amount of meconium needed in the amniotic fluid to stain the infants is very small, as shown by Desmond *et al.* (1956). This can also be deduced from the fact that in only half of our Stage 2 cases was meconium staining of the amniotic fluid noted at the time of delivery. Some of the infants also produced meconium stained vomit.

Stage 3 corresponds precisely to that of Clifford. In these infants the trunk and extremities are strikingly thin, moreover they have a rather pronounced dystrophic appearance. The skin peels off in large flakes. The nails and the skin are yellowish in colour.

Great care was taken in order to obtain as reliable information as possible about the time of gestation. Most of the mothers were under observation by the staff during pregnancy and, after labour, their menstrual history was examined in detail by us. Only cases with a regular menstrual cycle have been

included and any case in which the duration of pregnancy was at all doubtful has been rejected in the calculation of the gestation time.

Blood samples from the newborn infants were taken from the umbilical cord, which was clamped in two places immediately after delivery. Blood was drawn from either the vein or the arteries and mixed with heparin or sodium oxalate. If not indicated otherwise in the tables, the blood was always drawn from the vein.

The investigation was in two parts, the first covering the period from January 1 to October 31, 1956, and the second from November, 1956, to March 31, 1957. During the first part, 581 infants were investigated in the manner outlined above. However, the series was considered too small and therefore another 590 infants were studied. As both parts of the investigation have given the same results, no detailed statistical analysis of the two series is necessary.

Frequency. Only a few reports on the frequency of dysmaturity are available. Selander (1954) gives a figure of 12.0% from a study of 1,330 infants. If the infants were born more than four days after the calculated date, 20.3% displayed the syndrome. Taylor *et al.* (1952) stated that the incidence of yellow vernix not associated with haemolytic disease—that is, dysmaturity Stage 3, was 1.2%. Ravera and Bottinelli (1955) report that 2.25% of their infants were born more than 14 days after term and often had symptoms of dysmaturity (in 38 out of 42 cases). Kunstadter and Schnitz (1956) investigated 247 infants with a gestational age of 294 days or more and among these found 11 infants showing clinical symptoms consistent with dysmaturity; this gives a figure of 0.4% for infants delivered liveborn. Hinselmann (1957) in 5,258 deliveries had 600 infants with a gestation time of more than 294 days and among these were 42 infants with all or nearly all the signs of dysmaturity—that is, 7.0% of those with prolonged gestation and 0.8% of all the deliveries.

The frequency of dysmaturity in our material is shown in Table 1. The calculation of the frequency has been made only for the period when all of the infants delivered were classified.

TABLE 1
STAGE AND PERCENTAGE DISTRIBUTION OF
DYSMATURITY IN 444 NEWBORN INFANTS

Stage of Dysmaturity					
0	0-1	1	2	3	1-3
58.6%	24.8%	14.9%	1.6%	0.2%	16.7%

Thus, the frequency of dysmaturity in our material is 16.7%. The advanced Stages 2 and 3 were only seen in 1.8% of the newborn infants.

Gestation Time. Runge, and later Clifford, pointed out that dysmaturity is usually associated with prolonged pregnancy, but that it may be present even in infants born before term. Selander (1954) gives a dysmaturity frequency of 2.4% for infants born two to three weeks before term and a frequency of 1.1% for infants born more than three weeks before term.

Our material consists of 968 cases (Table 2).

TABLE 2
DISTRIBUTION ACCORDING TO GESTATION TIME
(968 CASES)

Stage of Dysmaturity	Duration of Pregnancy in Weeks									
	36	37	38	39	40	41	42	43	≥44	
0	3	9	21	103	141	149	42	14	11	
0-1		1	5	25	57	77	41	9	8	
1	1		2	13	47	61	50	26	12	
2				1	2	8	13	6	10	
Total	4	10	28	142	247	295	146	55	41	

Two hundred and three cases have been excluded because, as already mentioned, the gestation time was uncertain. It should be noted that the frequency displayed in Table 2 does not give a true picture of the dysmaturity distribution as sometimes only infants of mothers with a gestation time of 41 weeks or more were investigated. Thus, the frequency distribution has been somewhat shifted towards the right—that is, towards prolonged gestation. However, the table clearly shows the increased frequency of dysmaturity with increasing gestation time, but it also shows that most of the infants with Stage 2 dysmaturity are born within 294 days.

Parity of Mother and Sex of Infant. Several investigators have shown (Rütte, 1949; Hosemann, 1948; Clifford, 1954; Lindell, 1956; Strand, 1956) that the increase in infant mortality observed in prolonged pregnancy only refers to primiparae. It is, therefore, of interest to study whether dysmature infants are mostly born to primiparae or not. This information is presented in Table 3 together with the sex distribution of the infants.

If Stage 0-1 is included in the dysmaturity group, this syndrome is more frequent among primiparae than among multiparae. A statistical analysis shows that this difference is significant ($0.01 > p > 0.001$). With advancing stage of dysmaturity the frequency of boys dominates over that of girls. If again Stage 0-1 is included, this difference is statistically almost significant ($0.02 > p > 0.01$).

TABLE 3
SEX DISTRIBUTION AND DYSMATURITY AMONG
PRIMIPARAE AND MULTIPARAE (1,171 CASES)

Stage of Dysmaturity	Primiparae		Multiparae	
	Boys	Girls	Boys	Girls
0	100	107	201	196
0-1	61	41	86	80
1	69	55	75	55
2	12	6	15	8
3			4	
Total	242	209	381	339

Age of Mother. Clifford stresses that dysmature infants born of elderly primigravidae must be watched with great care, since experience has shown that foetal morbidity and mortality are high in these cases. It is therefore of interest to study whether the frequency of dysmaturity also increases with the age of the mother. As seen from Table 4 in our material there is no increasing frequency of dysmaturity with advancing age of the mother.

TABLE 4
PERCENTAGE DISTRIBUTION OF MATERNAL AGE

Maternal Age	Stage of Dysmaturity			
	0	0-1	1	2+3
<20	% 7.0	% 6.2	% 7.5	% 8.9
20-24	22.8	22.4	27.5	17.8
25-29	32.9	35.0	31.0	29.0
30-34	23.5	22.0	19.3	22.2
35-39	10.8	12.3	13.0	15.6
≥40	2.8	1.9	1.6	6.7

Weight of Infant. The growth of the foetus has been the subject of much speculation, particularly in the case of prolonged gestation. Solth (1947), Calkins (1948), McKiddie (1949), Hosemann (1952) and Lindell (1956) state that growth slows down towards the end of pregnancy and then become almost stationary or at least increases very little as gestation proceeds. Some authors (Rathburn, 1943; Freudenberg, 1950) even state that in prolonged gestation the foetus will lose weight. Table 5 gives the weight of the infants for the 921 cases in which the gestation time was between 39 and 44 weeks.

TABLE 5
MEAN WEIGHT IN G. AT TERM AND IN PROLONGED
PREGNANCY (921 CASES)

Duration of Pregnancy in Weeks	Stage of Dysmaturity			
	0	0-1	1	2+3
	g.	g.	g.	g.
39	3,320	3,425	3,118	2,600
40	3,440	3,363	3,332	3,140
41	3,675	3,597	3,423	3,230
42	3,708	3,677	3,649	3,233
43+44	3,819	3,761	3,561	3,603

Two curves are given in Fig. 1, one corresponding to Stages 0 and 0-1 considered together, the other to Stages 1, 2 and 3, also considered together. Fig. 1 shows that the mean weight of the normal infants increased continuously from the 39th to the 43rd and 44th week and that this increase was of the same magnitude each week. The mean weight of the dysmature infants was at 39 weeks already some 200 g. less than the mean weight of the normal infants but showed a similar weekly increase from the 39th to the 44th week.

FIG. 1.—Weight curves of normal and dysmature infants.
— Normal infants, Stages 0 and 0-1
- - - - - Dysmature infants, Stages 1, 2 and 3

From a consideration of the gestation times in Table 2 and the weight curves in Fig. 1 two important conclusions can be drawn:

1. Although the frequency of dysmaturity is increased in prolonged gestation, it is not caused by prolonged gestation since evidence of dysmaturity is already manifest before term.
2. Although both normal and dysmature infants increase in weight with increasing gestation time, the difference in the weight curves can explain the apparent decrease in weight in prolonged pregnancy observed by other authors. Because of a steadily increasing proportion of dysmature infants in prolonged pregnancy, the percentage of the normal infants with high weight is gradually reduced,

whereas the percentage of the dysmature infants with small weight gradually increases. This causes a flattening of a growth curve which includes all the infants. With great foresight this explanation was hypothetically proposed by Clifford (1957).

The reduced weight which the dysmature infants show as compared with the normal ones indicates insufficient nutrition during a relatively extended period of time, as would be expected in the case of placental insufficiency.

Length of Infant. Most of the above-mentioned authors who have studied the weight of the newborn infant have also studied the length and found that it increases up to and including the 43rd week.

This is confirmed in Table 6 which shows that, according to our data, the infant's length increases irrespectively of dysmaturity until and including the 43rd week. Thus, dysmature infants are of normal length but reduced weight. However, it should be remembered that it is difficult to measure the length of the newborn infant with a satisfactory exactness. The reduction in length which is suggested in the 44th week may not be real because the material is small and only includes 40 infants, and furthermore, in spite of our precautions, the true gestation time may, in some cases, be only 40 weeks instead of 44.

TABLE 6
INFLUENCE OF GESTATION TIME AND DYSMATURITY ON MEAN LENGTH OF INFANT

Gestation Time in Weeks	Stage of Dysmaturity			
	0	0-1	1	2+3
	cm.	cm.	cm.	cm.
37	46.6	47.5	—	—
38	48.8	47.9	50.5	—
39	49.5	49.5	49.0	48.0
40	49.7	49.6	49.6	50.0
41	50.8	50.5	50.3	50.1
42	50.6	50.9	51.1	49.7
43	51.7	51.4	51.0	52.0
≥44	50.2	50.6	51.0	51.1

Oxygen Saturation in Cord Blood. The oxygen saturation in the cord blood during prolonged gestation has been the subject of much discussion. Walker and Turnbull (1953) claimed that the oxygen saturation in the cord blood decreases with advancing gestation time in prolonged pregnancy. However, the number of infants studied was small. Minkowski *et al.* (1953) support Walker's thesis although they find exceptions. MacKay (1957) in a recent study, like Walker's, also from Scotland, of 240 infants, reaches the same conclusion as Walker and Turnbull (1953). But MacKinney *et al.* (1955) studying cord blood oxygen saturation in 594 infants did not find significant differences between mature

infants and infants with prolonged gestation. Rooth and Sjöstedt (1957b), investigating 363 infants, found no changes in the oxygen saturation from the 39th week to the 43rd week.

As far as we know, the oxygen saturation in cord blood has not been studied before in relation to dysmaturity, although Runge and most subsequent authors on dysmaturity have considered dysmature infants to be hypoxic.

The oxygen saturation, measured with a Brinkman haemoreflexor in the manner already described by Rooth and Sjöstedt (1955) has been measured in the umbilical vein in 210 infants and in the arteries in 165 infants in our series (Table 7).

TABLE 7
MEAN OXYGEN SATURATION IN PER CENT IN UMBILICAL CORD BLOOD

		Totals	Stage of Dysmaturity			
			0	0-1	1	2
Vein	Number	210	104	35	59	12
	HbO ₂		63.7%	58.6%	56.9%	51.1%
Artery	Number	165	79	29	47	10
	HbO ₂		38.7%	34.7%	34.6%	24.3%

The oxygen saturation decreases both in the umbilical vein and in the arteries with advancing stages of dysmaturity. Thus, according to our results, in prolonged pregnancy the cord blood oxygen saturation is decreased only if the infant also is dysmature. An infant with no signs of dysmaturity will retain its oxygenation in spite of a prolongation of pregnancy beyond term.

These observations could explain the differing results obtained by the various authors studying the oxygen saturation in the cord blood of infants in prolonged gestation. If the number of infants studied is small, as in Walker's report, the percentage of dysmature children may be much higher than if a sufficiently large number of cases had been studied. Furthermore, there may be a notable difference in the frequency of dysmaturity in different countries.

Haemoglobin in Cord Blood. Walker and Turnbull (1953) found that from the 40th to the 43rd week the amount of haemoglobin in cord blood increased considerably and interpret this as a compensation for the decreasing oxygen saturation. Marks *et al.* (1955) and Rooth and Sjöstedt (1957a) could not confirm these results, both groups finding no increase in the haemoglobin in cord blood during prolonged gestation. MacKay found a slight rise with increasing maturity but the results tended to agree with the results of Marks *et al.*

No studies are available regarding the influence of dysmaturity on the cord blood haemoglobin. We have measured this in 885 infants (Table 8). The technique has been described earlier by Rooth and Sjöstedt (1957a).

The haemoglobin values rise with advancing signs of dysmaturity and this rise is already evident in the intermediate group, Stage 0-1. This increase of haemoglobin in dysmature infants suggests that, *in utero*, they were hypoxic, but it may be only a sign of haemoconcentration.

TABLE 8
MEAN HAEMOGLOBIN IN G. PER 100 ML. UMBILICAL CORD BLOOD (885 CASES)

	Stage of Dysmaturity			
	0	0-1	1	2
Number	450	205	194	36
Haemoglobin	16.1	16.7	16.9	17.0

Plasma Pentoses. The concentration of pentoses in plasma has been studied by Green and his collaborators, among others. Green *et al.* (1949) noted a significant rise in the plasma pentose fractions in battle casualties and believed this to be the result of ischaemia in the injured tissues. In the pregnant woman an increase in the plasma pentose fractions has occasionally been noted. Green *et al.* (1951) consider this to be due to a relative uteroplacental ischaemia.

Using the method of Mejbbaum (1939) as modified by Green *et al.* (1951), we have measured the plasma pentoses in the cord blood of 198 infants (Table 9).

The plasma pentoses increase with advancing signs of dysmaturity. This probably indicates a

TABLE 9
MEAN PLASMA PENTOSSES IN MG. PER 100 ML. UMBILICAL CORD BLOOD (198 CASES)

	Stage of Dysmaturity			
	0	0-1	1	2
Number	92	35	58	13
Pentoses	4.6	5.0	5.6	6.5

progressive ischaemia and therefore supports our results on the analyses of the oxygen saturation and the haemoglobin in the cord blood.

Protein-bound Hexoses. For more than 50 years the protein-bound polysaccharides in serum have been studied mainly in connexion with diabetes mellitus. Abnormally high values have been found in a number of different pathological conditions. However, no final explanation for this abnormality has been found (Keiding, 1957). The

most commonly accepted explanation (Greenspan *et al.*, 1951) is that the protein-bound hexoses indicate tissue destruction. We have measured the protein-bound hexoses in the cord blood of 246 infants and our results are given in Table 10.

The protein-bound hexoses increase with advancing signs of dysmaturity. Even if no definite explanation for this can be given, it points to an increasing pathological process in the infant or in the placenta.

TABLE 10
MEAN PROTEIN-BOUND HEXOSES IN MG. PER 100 ML.
UMBILICAL CORD BLOOD (246 CASES)

	Stage of Dysmaturity			
	0	0-1	1	2
Number	48	105	74	19
Hexoses	127.1	127.0	148.5	152.3

Bilirubin. Icterus neonatorum has been the subject of extensive investigations. Most of these studies have been devoted to the serum of the infant's blood after birth; however, others have been concerned with the cord blood as well. Davidson *et al.* (1941) report a mean value of 1.69 mg. per 100 ml. in 94 cases with a range of 0.42 to 3.9. As others before them they found that the higher the cord bilirubin the more intense became the icterus of the infant and the longer it lasted. Schick *et al.* (1942) compared the cord blood bilirubin in prematures, normal infants and overmatures (weight more than 3,800 g., length more than 52 cm.). With increasing maturity the bilirubin was reduced but the differences were so small that no conclusions could be drawn. We have analysed the bilirubin in the cord blood of 446 infants with Jendrassik's method (Table 11).

TABLE 11
MEAN BILIRUBIN IN MG. PER 100 ML. UMBILICAL CORD
BLOOD (446 CASES)

	0	0-1	1	2
	218	115	103	10
Number	1.52	1.58	1.86	2.82
Bilirubin				

Although the differences are not large, there is a gradual increase in the bilirubin values in the cord blood with advancing signs of dysmaturity.

Non-protein Nitrogen. Reports on the NPN in cord blood have been published by Hellmuth (1924), Naeslund (1931), Slemons (1919), Pommerenke (1936) and Lichtenstein (1931). Their values vary between 25 and 40 mg. per 100 ml. of cord blood, and in the umbilical vein the values are

slightly higher than in the arteries. Lichtenstein has correlated the NPN with the birth weight of the infant and shown that starting at a weight of 1,500 g., the NPN remains at the same level or 33 mg. per 100 ml. of cord blood.

McCance and Widdowson (1954) studied ten full term or post-mature infants all delivered by forceps. They analysed the urea and found a mean value of 39.9 mg. per 100 ml. of cord blood in six dysmature infants as compared with 30.9 in four normal infants. They took this as an indication that the urea concentration in cord blood is higher among dysmature infants and recommended further investigations.

We have analysed the cord blood of 804 infants for NPN (Table 12).

TABLE 12
MEAN NON-PROTEIN NITROGEN IN MG. PER 100 ML.
UMBILICAL CORD BLOOD (804 CASES)

	Stage of Dysmaturity				
	0	0-1	1	2	3
Number	398	209	170	23	4
Non-protein nitro- gen	28.8	29.4	29.7	34.7	37.0

The non-protein nitrogen in the cord blood also increases with increasing signs of dysmaturity. This is caused either by a placental insufficiency which prevents the foetus from excreting its NPN through the placenta or by an increase in tissue (notably placental tissue) destruction and subsequent increased production of NPN. Probably both of these mechanisms are of importance at the same time.

Albumin in Urine. Tests for proteinuria have been so consistently positive, especially during the second to fifth day after birth, that albumin in the urine is to be considered a more or less physiological constituent of the newborn infant's urine (Heller, 1913; Franz and Reuss, 1914; Ewald, 1916; Pfaundler, 1924). Ewald found that 75% of the urine samples taken from infants with birth weights below 3.5 kg. reacted positively to the albumin test; furthermore, 69% of the tests on larger infants were positive.

We have tested the urine for albumin using Heller's test on specimens from 577 newborn infants (Table 13). These specimens were taken as early as

TABLE 13
FREQUENCY IN PER CENT OF ALBUMIN IN URINE
(577 CASES)

	Stage of Dysmaturity			
	0	0-1	1	2+3
Number	114	208	217	38
Albumin	18%	29%	30%	63%

possible, always within the first three days of life. The urine of all infants with positive albumin tests showed a negative reaction within one week after birth.

The frequency of albumin in the urine of the newborn infant increases with the degree of dysmaturity, showing that these infants are also suffering from an impairment of the renal function.

Glycosuria. Hoeniger (1911) reports the frequent occurrence of reducing substances in infants after complicated deliveries. Lindig (1922) found this in only three cases out of 24 delivered by forceps.

We have, using the glucose oxidase reaction, tested the urine of 577 newborn infants shortly after birth (never later than the third day). (Table 14.)

TABLE 14
FREQUENCY IN PER CENT OF REDUCING SUBSTANCES
IN URINE (577 CASES)

	Stage of Dysmaturity			
	0	0-1	1	2+3
Number	114	208	217	38
Reducing substances	0.9%	1.9%	5.1%	10.5%

The frequency of glycosuria also increases with the degree of dysmaturity. In the normal infant glycosuria is rare, but is present in about 10% of the dysmature infants in Stages 2 and 3. Sometimes these infants may even show a clinical picture which resembles diabetes, a case report of which has been presented by Engleson and Zetterqvist (1957).

Discussion

The present study shows the importance of distinguishing between, on the one hand, the gestation time of the infant—that is, whether it is prolonged or not, and, on the other, the state of the infant—that is, whether the infant is normal or dysmature.

We find no untoward effects on the infants due to the prolongation of pregnancy *per se*, but we do find considerable changes in dysmature infants. Unfortunately, this important fact does not yet solve the clinical controversy as to whether labour should be induced or not in a woman who carries her child beyond term. The answer is, of course, that labour should be induced if the infant is dysmature, but this cannot as a rule be verified until after the child is born. If the Ballantyne-Runge sign is present—that is, if the circumference of the abdomen is rapidly reduced late in pregnancy, the child is likely to be dysmature. Foetal electrocardiogram studies

(Southern, 1957) also indicate a possible method of diagnosing dysmaturity before birth.

The clinical picture of the dysmature infant is distinct and can be recognized with a little training. It would be advantageous to register all the newborn infants according to dysmaturity. Thus, mortality and morbidity statistics on the neonatal period in different hospitals and countries would be more easily comparable.

Interpretation of Results

The reduced weight, the hypoxia, the increased haemoglobin, and possibly also the increased NPN, indicate a reduction in the diffusion capacity of the placenta. This reduction is probably caused by an ischaemia or destruction of placental tissue, as indicated by the increase in pentoses and protein-bound hexoses, and possibly also by the increased NPN. The increase in bilirubin may be a sign of the increased blood formation and a concomitant increased turnover of the haemoglobin pigments. It may also be due to hypoxic disturbances in the liver function, as the increased frequency of albumin in the urine might be a sign of hypoxic kidney function.

Summary

In order to prevent confusion between the terms 'prolonged pregnancy' and 'postmaturity' the term 'dysmaturity' is introduced to denote the placental insufficiency syndrome.

One thousand, one hundred and seventy-one newborn infants have been classified according to dysmaturity and studied from various aspects.

The frequency of dysmaturity in our series is 16.7% and increases with advancing gestation time; it is also higher for primiparae and male infants. Both normal and dysmature infants increase in weight during gestation, but from the 39th week onwards until delivery the dysmature infants have a mean birth weight 200 g. below that of normal infants. As dysmaturity becomes increasingly severe the oxygen saturation in the cord blood decreases, whereas there is an increase in the haemoglobin, in the plasma pentoses, in the protein-bound hexoses, in the bilirubin and in the non-protein nitrogen.

The more pronounced signs of dysmaturity are also allied to an increased frequency of albumin and glucose in the urine of the newborn infants.

This study was supported by grants from the Swedish Medical Research Council, Stiftelsen Synsvaga Barn and the Association for the Aid of Crippled Children.

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