

## ICTERUS NEONATORUM

### III. THE OXYGEN CAPACITY AND SATURATION OF THE MOTHER AND FOETUS<sup>1</sup>

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In our first communication on this subject (1) we concluded that "Icterus neonatorum is a physiologic condition, which is the result of a post-natal readjustment from an environment requiring the presence of polycythaemia, for the maintenance of oxygenation, to one in which no such extraordinary measures are necessary. Icterus is present in all infants, visibility being only a matter of degree."

In our second study (2) we succeeded in producing polycythaemia, and later icterus, in guinea pigs, after keeping them in a chamber under reduced atmospheric pressure. Very soon after the animals were removed from the low pressure chamber they all showed positive indirect Van den Bergh reactions, and increased icteric indices. These findings, just as in new born infants, coincided with the reduction of the polycythaemia. We had thus succeeded in producing experimentally, in animals, an icterus in which the antecedent causes were analogous to the conditions under which, in our opinion, icterus neonatorum appeared.

We had some evidence to warrant the assumption that the foetus lived in a condition of relative oxygen unsaturation. The polycythaemia itself, the large number of immature red cells, and the anatomy of the foetal circulation in which the bulk of the circulating blood is mixed, venous and arterial, all pointed towards this conclusion. We felt, therefore, that in order to complete our chain of evidence, it was essential to study the oxygen capacity and saturation in the mother and the foetus.

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Studies of this nature have already been made in goats by Huggett (3). He has shown that the oxygen saturation of the arterial blood of the goat foetus is much lower than that of the mother. No human studies have ever been made.

We first studied twenty cases in which the labour was normal, or required only slight obstetrical aid, such as low forceps. In none of the cases studied did the child require any special methods for the initiation of pulmonary respiration. In addition, studies were made in five cases of cesarean section. The oxygen capacity and saturation were estimated by the Van Slyke method (4). The foetal blood was

TABLE 1  
*Patients delivered with chloroform anaesthesia*

Case number	Foetus		Mother		Anaesthetic	Mode of delivery
	O <sub>2</sub> capacity	O <sub>2</sub> saturation	O <sub>2</sub> capacity	O <sub>2</sub> saturation		
	<i>volumes per cent</i>	<i>per cent</i>	<i>volumes per cent</i>	<i>per cent</i>		
2	22.82	78	19.56	91	Chloroform	Normal
5	21.34	76	18.92	93	Chloroform	Normal
7	22.23	70	18.56	89	Chloroform	Normal
9	21.92	79	18.35	93	Chloroform	Normal
11	22.35	79	18.79	93	Chloroform	Normal
12	21.82	70	19.24	89	Chloroform	Low forceps
13	22.34	71	17.53	88	Chloroform	Low forceps
14	20.87	82	18.03	93	Chloroform	Normal
15	21.56	79	18.21	92	Chloroform	Normal
18	20.83	70	18.76	89	Chloroform	Low forceps
19	21.73	71	19.92	89	Chloroform	Low forceps

taken from the umbilical vein, under oil, at the moment of clamping the umbilical cord. A tightly fitting needle on an all glass syringe was introduced into the umbilical vein. The barrel of the syringe contained a few potassium oxalate crystals, and a small quantity of oil. Estimations were made immediately afterwards. Maternal blood was taken from the radial artery, also under oil.

The twenty-five cases were divided into three groups. Group I, (table 1) comprised eleven cases in which chloroform anaesthesia was used. Of these eleven cases, seven were normal deliveries, and in four, low forceps were used. Group 2 (table 2) consists of five ces-

arean sections, in which nitrous oxide and oxygen was the anaesthetic used, and group 3 (table 3) represents nine cases in which neither anaesthetic nor other aids were required.

In all twenty-five cases, the oxygen saturation of the blood was distinctly lower in the foetus. The maternal figures were practically

TABLE 2  
*Patients delivered by Cesarean section with nitrous oxide—oxygen anesthesia*

Case number	Foetus		Mother		Anaesthetic	Mode of delivery
	O <sub>2</sub> capacity	O <sub>2</sub> saturation	O <sub>2</sub> capacity	O <sub>2</sub> saturation		
	volumes per cent	per cent	volumes per cent	per cent		
21	22.86	81	18.53	93	Gas and oxygen	Cesarean section
22	21.57	78	18.21	94	Gas and oxygen	Cesarean section
23	21.98	80	19.04	94	Gas and oxygen	Cesarean section
24	22.56	80	18.05	95	Gas and oxygen	Cesarean section
25	21.83	78	17.93	94	Gas and oxygen	Cesarean section

TABLE 3  
*Patients with unaided delivery, no anaesthetic*

Case number	Foetus		Mother		Anaesthetic	Mode of delivery
	O <sub>2</sub> capacity	O <sub>2</sub> saturation	O <sub>2</sub> capacity	O <sub>2</sub> saturation		
	volumes per cent	per cent	volumes per cent	per cent		
1	21.72	81.1	19.34	94	None	Normal
3	20.89	79	18.1	94	None	Normal
4	22.12	80	17.95	95	None	Normal
6	22.32	76	18.12	94	None	Normal
8	20.82	78	18.92	95	None	Normal
10	22.85	82	17.89	94	None	Normal
16	20.98	80	19.21	95	None	Normal
17	21.34	79	19.34	94	None	Normal
20	20.93	81	18.96	94	None	Normal

normal, though lowest in the four cases requiring low forceps. They were slightly lower in the whole group receiving chloroform than in the other two groups. The oxygen capacity was greater in foetal than in maternal blood, in all twenty-five cases, obviously because of the polycythaemia.

## COMMENT

In a previous communication we have drawn attention to the admixture of blood from the umbilical vein with the general venous circulation. This admixture takes place in the liver and heart. (Alpha factor of Lundsgaard and Van Slyke (5).

In the light of the experiments presented here, it now becomes apparent that the oxygen exchange in the placenta itself is defective. The placenta is a relatively poor respiratory organ as compared to the post-natal lung. (This is analogous to the "L" factor of Lundsgaard and Van Slyke.) How much of this deficiency of oxygenation of the umbilical vein blood can be accounted for by placental metabolism has not yet been determined.

## CONCLUSIONS

1. Foetal arterial blood (umbilical vein) possesses an increased oxygen capacity and a diminished oxygen saturation.
2. This diminution in oxygen saturation of the blood in the umbilical vein is to be accounted for by the deficient respiratory function of the placenta.
3. Maternal blood from the radial artery shows values for capacity and saturation which are within normal limits.
4. Further evidence is thus adduced in support of our theory that the polycythaemia in the newborn infant is the result of oxygen unsaturation of the foetus in utero.

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