ASPHYXIA IN THE PRIMATE FOETUS

By

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My comments will be confined to describing the pattern of foetal cardiovascular responses to asphyxia in the *Macaca mulatta* monkey. The control values presented were similar to those obtained in the pigtail and stump tail macaques. We have also studied a series of baboon pregnancies with similar methods. In general, the results are comparable, although the umbilical blood flow of the baboon is less but placental diffusion of antipyrine is higher as is the foetal PO$_2$. The oxygen uptakes of the foetuses are similar.

We studied two groups of seven animals (*M. mulatta*) each during acute experiments under halothine anaesthesia. In one group the near term pregnant animals were made hypoxaemic by gradually decreasing the concentration of maternal inspired oxygen. In the other group, adequate maternal oxygenation was maintained.

The foetuses of each group were studied after inserting catheters in the foetus' inferior and superior vena cava, umbilical artery and vein, and systemic artery and vein at maternal laparotomy. Umbilical blood flow was measured by an antipyrine tracer method and the cardiac output and blood flows to all organs were measured using three differently labelled radioactive microspheres, 50 microns in diameter, which were injected simultaneously into the umbilical vein and inferior and superior vena cava. Each foetus was replaced in the uterus and the uterus closed and replaced in the abdomen prior to obtaining any measurements.

The oxygen in the maternal arterial blood of the hypoxaemic group was significantly decreased in oxygen saturation and oxygen tension to average levels of 72% and 72 mmHg, respectively. There were no significant differences in pH, PCO$_2$, heart rate, or maternal systemic artery blood pressure.

The foetuses of the hypoxaemic mothers became asphyxiated as defined in
terms of a decrease in umbilical artery saturation (control 55% versus 19% in foetal distress groups), oxygen tension (control 23 mmHg versus 14 mmHg in foetal distress groups), and oxygen uptake (control 7 ml/min/kg versus 3 ml/min/kg in foetal distress groups).

The asphyxiated foetuses also developed a mixed acidosis with an average pH of 7.18, PCO₂ of 53 mmHg and lactic acid of 5.2 mEq./liter.

In the asphyxiated distress group the umbilical blood flow decreased from average control levels of 90 ml/min to 40 ml/min and the average cardiac output decreased from 194 ml/min to 132 ml/min. The flow of oxygenated, umbilical blood through the ductus venosus increased from 53% to 90% of the umbilical blood flow.

The return of the inferior vena cava and the superior vena cava venous blood to the heart was thus suddenly reduced.

The percentage of the cardiac output going to the lungs decreased by two-thirds (from 10.7% to 3.2%).

However, the percentage of the cardiac output going to the adrenals, brain and heart increased, maintaining the blood flow to these organs per gram of tissue. Further, the composition of the blood going to these vital organs indicated that there was a marked increase of blood returning from the superior vena cava which was shunted through the foramen ovale. There was a 26 fold increase in the percentage of brain blood flow derived from the superior vena cava and an over 4 fold increase in the percentage of coronary blood derived from the superior vena cava.

A similar series of events occurred when term pregnant mothers were subjected to hypotension as occurred when they were subjected to hypoxaemia.

In all of these experiments the foetuses were born alive. However, the asphyxiated foetuses required resuscitation and were never able to breath spontaneously. All of the foetuses of the control mothers breathed spontaneously and survived in good health until they were electively sacrificed.

In conclusion, the cardiovascular responses of the foetus to asphyxia are characterized by a decrease in umbilical blood flow and cardiac output with an increase shunting of a greater percentage of the reduced umbilical blood flow through the ductus venosus into the inferior vena cava to return to the heart. The superior vena cava and inferior vena cava returns to the heart are decreased. However, perfusion of the heart and brain is maintained as a greater percentage of the reduced cardiac output goes to these organs; this blood is composed of a greater proportion of unsaturated venous blood returning from the superior vena cava and shunted through the foramen ovale. Perfusion of the lungs is greatly reduced but blood flow to the adrenals is maintained within the normal range.

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