secretors and lowest in O and B secretors, the opposite of the gradient found. Another possible explanation of the serum enzyme variation is that blood group and secretor status affect the rate of removal of intestinal alkaline phosphatase from the circulation. If this were the case, however, then mucosal enzyme concentrations would be unlikely to show any gradient according to the genetic constitution of the individuals.

The precise metabolic role of intestinal alkaline phosphatase is not understood. The rise in the serum concentration of the enzyme after a fatty meal suggests that it may play a part in fat absorption or transport, which therefore might be affected by the ABO blood group and secretor status of the individuals. It has been claimed from studies of blood lipid concentrations in twins that day-to-day variation in free serum cholesterol levels is affected by ABO blood group[20]. On the other hand, the increase of serum turbidity after a fatty meal has not been found to differ with the ABO blood group or secretor status of the individuals. These observations could be reconciled if genetic characteristics affected transport of some specific class of lipid.

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## Storage of Carbon Dioxide in Muscle

WHEN ventilation is suddenly arrested, the carbon dioxide produced by the body is stored within the body tissues as bicarbonate and dissolved carbon dioxide. It has been suggested that this storage of carbon dioxide does not proceed uniformly in all tissues. Rather it has been proposed that the body can be conceived as being composed of multiple compartments which store carbon dioxide at different rates.

In the multicompartment model of carbon dioxide stores proposed by Fahri and Rahn, during apnoea, muscle—because it has a low ratio of perfusion per unit volume—acts as a repository for carbon dioxide produced by other organs[1]. When there is no ventilation, therefore, the carbon dioxide content of the venous blood should be less than the carbon dioxide content of the arterial blood—the reverse of the usual situation.

To test this prediction, we measured the carbon dioxide and oxygen tension, the hydrogen ion concentration and the haematocrit of blood from the femoral artery and vein, and calculated carbon dioxide contents in nine anaesthetized and paralysed dogs during apnoea.

After the paralysed dogs had been ventilated with 100 per cent oxygen for 30 min, arterial and venous blood was obtained from one hind limb, and carbon dioxide production was measured. Apnoea was produced by stopping artificial ventilation. During the apnoeic period, blood samples were obtained at intervals of 1 or 2 min while the trachea of the dog was connected to a spirometer which contained oxygen so that full arterial saturation could be maintained. In three dogs, blood flow in the opposite limb was measured with a rotameter.

Table 1. FEMORAL ARTERIAL AND VENOUS CARBON DIOXIDE TENSIONS AND CONCENTRATIONS DURING APNOEA IN NINE DOGS (MEAN ± STANDARD ERROR)

| Time (min) | $CO_2$ tension (mm Hg) | | $CO_2$ concentration difference (vol. per cent) |
|---|---|---|---|
| | Artery | Vein | Vein–artery |
| 0 | 35 ± 4 | 40 ± 5 | +2·1 ± 0·7 |
| 1 | 47 ± 4 | 44 ± 4 | −1·4 ± 0·3 |
| 2 | 52 ± 5 | 47 ± 4 | −2·5 ± 0·6 |
| 4 | 63 ± 6 | 53 ± 5 | −3·3 ± 0·9 |
| 6 | 73 ± 6 | 60 ± 5 | −3·3 ± 0·6 |

The carbon dioxide production of the dogs during the control period of artificial ventilation averaged 74 ml./min (standard error, ± 4). The mean blood flow in the femoral artery was 135 ml./min in the control period but decreased to an average of 92 ml./min during apnoea. Table 1 shows the carbon dioxide tensions and the venous minus the arterial carbon dioxide concentration differences observed during apnoea. During artificial ventilation the venous carbon dioxide tension exceeded the arterial by 5 mm of mercury while the carbon dioxide content of the vein was 2·1 vol. per cent greater than the artery. After 1 min of apnoea, these gradients were reversed and the venous carbon dioxide tension and concentration were less than those of the artery. At 6 min of apnoea, the average venous carbon dioxide tension was 13 mm of mercury less than that measured in the artery while the carbon dioxide concentration was 3·3 vol. per cent less.

The result of multiplying the average blood flow of the femoral artery by the mean venous–arterial carbon dioxide concentration differences shows that, during each minute of apnoea, muscle stores about 3 ml. of carbon dioxide produced elsewhere. This is about 4 per cent of the total production of carbon dioxide by the body each minute.

According to the multicompartment theory, carbon dioxide is stored in different tissue compartments, each with its own level of perfusion, metabolic rate and dissociation curve for carbon dioxide[2]. Ventilation changes produce rapid alterations in carbon dioxide content in richly perfused tissue such as the brain, but only slow changes in tissues with a large volume but a relatively poor perfusion such as muscle. The results of this study agree with the idea that muscle carbon dioxide reaches equilibrium sluggishly after a change in arterial carbon dioxide tension, thus producing the reversal of the usual venous–arterial concentration differences.

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