fall in Qs/Qt from 9.8 to 6.4%. With induced pulmonary oedema, a fall in cardiac output from 211 ± 0.24 l/min to 137 ± 0.19 l/min was accompanied by a fall in Qs/Qt from 29% to 21.6%. In no instance was there a significant change in arterial oxygen tension despite a fall in mixed venous saturation from 75 ± 4.5% to 59 ± 3.6% in dogs with normal lungs and from 62 ± 3.4% to 49 ± 3.0% in dogs with pulmonary oedema.

In another series of experiments on 11 pentobarbitone-anaesthetized dogs, mixed venous oxygenation was varied independent of cardiac output by means of veno-venous shunting of blood through a disc oxygenator. Changing the composition of the gas flow through the oxygenator was without effect on pulmonary artery pressure, pulmonary artery wedge pressure, cardiac output or pulmonary vascular resistance. However, increasing SvO₂ from 50 to 70% in normal lungs caused a significant increase in Qs/Qt from 7.9 to 10.1%. In 7 dogs with pulmonary oedema, a change in SvO₂ from 32.2 to 59.3% was without effect on pulmonary pressure or flow but caused a significant increase in Qs/Qt from 25.7 to 28.8%.

We conclude that change in SvO₂ contributes a small but significant amount to the direct relationship between changes in cardiac output and Qs/Qt.

REFERENCES

EFFECT OF FREQUENCY OF AUTOMATIC VENTILATION ON CARDIORESPIRATORY FUNCTION IN THE DOG
J. N. Lunn, W. W. Mapleson and R. T. Cheilolot
Department of Anaesthetics, Welsh National School of Medicine, Cardiff

Seven alaskan dogs were anaesthetized with thiopentone followed by halothane in air and oxygen; after paralysis with pancuronium they were ventilated with a modified Barnet Mark III ventilator at the following frequencies in sequence 25, 50, 25, 6 and 25/min. The I/E ratio was kept at 1/2; expiration was always passive to atmosphere. At each frequency, the tidal volume and the inspired oxygen concentration were adjusted to maintain PaO₂ and PaCO₂ constant to within ±1 and ±10 mm Hg respectively, at levels of about 40 and 140 mm Hg respectively; the inspiratory resistance was adjusted so that flow had just fallen nearly to zero by the end of the inspiratory phase.

After preparations lasting 3–5 hours, each frequency was maintained until conditions had been stable for 10 min—about half an hour at each frequency.

Blood pressure, carinal pressure, oesophageal pressure, expiratory fourth heart sound rate, arterial CO₂ and O₂ tensions (by flow-through electrodes), mixed-expired CO₂ concentration, and inspired-to-expired O₂ concentration difference, were continuously monitored; in addition, when conditions were stable at each frequency, measurements were made of total ventilation, inspired and mixed-expired halothane concentration, inspired O₂ concentration, cardiac output, pulse rate, volume of blood out of the circulation, nasal temperature, various apparatus temperatures necessary for correction purposes and the pH, Hb and Hct of arterial blood. From these measurements and the equation for the human oxy-haemoglobin dissociation curve the following were calculated: physiological deadspace, venous admixture and mean carinal pressure.

For each parameter the value at a frequency of 50 or 6/ min was expressed as a difference from the mean of the values at the bracketing frequencies of 25/min.

<table>
<thead>
<tr>
<th>Parameter</th>
<th>Mean control</th>
<th>Difference from control</th>
</tr>
</thead>
<tbody>
<tr>
<td>Vt (ml/kg)</td>
<td>10.9</td>
<td>-2.1*** + 14.6***</td>
</tr>
<tr>
<td>FiO₂ (%)</td>
<td>35.6</td>
<td>-0.3 + 0.4*</td>
</tr>
<tr>
<td>Vd (ml/kg)</td>
<td>6.7</td>
<td>+0.03 + 1.6*</td>
</tr>
<tr>
<td>Q (ml/min/kg)</td>
<td>148</td>
<td>-6.7 - 1.3</td>
</tr>
<tr>
<td>Qva/Q</td>
<td>5.9</td>
<td>-0.09 + 0.15</td>
</tr>
</tbody>
</table>

* = P<0.05; *** = P<0.001.

Analysis of the results (table I) showed that physiological deadspace increased with decreasing frequency and hence with increasing tidal volume—in a manner broadly similar to that found by Cooper (1967) in man—and that changes of venous admixture and cardiac output with frequency were small and not significant.

Calculation of 95% confidence limits showed that, under the conditions of the experiments, any real change of cardiac output with frequency was probably less than ±12% of the control value, and any change of venous admixture was probably less than ±1.2% of cardiac output.

REFERENCE

THE HAEMODYNAMIC EFFECTS OF INTRAVENOUS SODIUM BICARBONATE
J. H. Chamberlain, R. G. F. L. Seed and A. M. Barrett
Department of Clinical Physiology and Anaesthetics, Guy’s Hospital, London

During correction of metabolic acidosis in anaesthetized dogs it was noted that a rapid intravenous injection of NaHCO₃ aq. (8.4%) caused a triphasic haemodynamic change. An initial Starling effect was seen, evidenced by an increase in left ventricular end diastolic pressure (LVEDP) and stroke volume (SV) (phase I). This was transient and was followed by a more prolonged depression of myocardial function (approximately 1 min duration) (phase II). The LVEDP rose at the same time as maximum left ventricular dp/dt (max.dp/dt), SV, peak flow (PF) and maximum acceleration (MA) decreased. The third phase, which lasted for much longer, consisted of a general improvement in function. LVEDP gradually returned to control levels, whilst max.dp/dt, PF, MA, SV and cardiac output (CO) rose above control levels. The end-tidal CO₂ showed a striking increase during the depressed phase and blood-gas analysis confirmed a substantial increase in PaCO₂. Standard bicarbonate and pH were above normal at this time.

In an attempt to analyse these changes, the effects of intravenous saline, NH₄Cl, tris buffer and hypertonic agents were examined, the same volume being injected at the same rate. CO₂ by inhalation was also studied. Intravenous saline produced the transient Starling effect of the first phase and no long-term effects. As 8.4% NaHCO₃ aq. is hypertonic, an equivalent solution of dextrose was injected in order to simulate the osmotic effects. After the phase I effect this produced a fall in peripheral resistance (PR) and LVEDP, with increases