CONTINUOUS ALVEOLAR CARBON DIOXIDE ANALYSIS AS A MONITOR OF PULMONARY BLOOD FLOW

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Several reports have appeared indicating the value of alveolar carbon dioxide analysis during anesthesia as a physiological monitor of pulmonary ventilation (1–6). However, alveolar carbon dioxide tension has not been correlated with pulmonary blood flow during anesthesia.

This report indicates that with uniform, adequate ventilation and diffusion, fluctuations in alveolar carbon dioxide tension may indicate critical changes in pulmonary capillary blood flow. Furthermore, certain acute fluctuations in pulmonary blood flow may be rapidly and sensitively reflected in alveolar carbon dioxide tension as measured by the Liston-Becker rapid infrared absorption analyzer.

Methods and Results

Alveolar carbon dioxide was sampled from the endotracheal tube by standard methods (1, 7, 8), through microcatheter polyethylene tubing of calibrated bore inner diameter 0.034 inches, outer diameter of 0.050 inches. Carbon dioxide tension was measured in a Liston-Becker Model 16 continuous rapid infrared absorption analyzer, the results recorded by an Esterline-Angus recording milliammeter. The procedure described by Collier (7) for calibration of the instrument was followed.

Anesthetized patients were studied during cardiac and great vessel surgery. The selected cases presented in figures 1 to 9 illustrate the

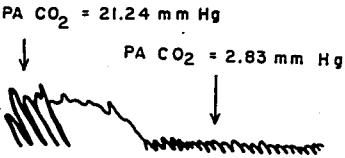

Fig. 1. Alveolar carbon dioxide tension (P_ACO_2) decrease from 21.24 mm. of mercury to 2.83 mm. of mercury following systemic hypotension (blood pressure fall from 110/70 to 0) secondary to severe, abrupt hemorrhage in a six-year-old patient with tetralogy of Fallot, left superior vena cava and left azygous vein. Hypothermia (32.2 C) and cyclopropane anesthesia. Potts anastomosis.

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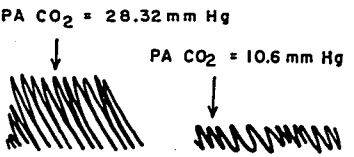

FIG. 2. P_ACO_2 decline from 28.32 mm. of mercury to 10.6 mm. of mercury following fall in cardiac output secondary to poor myocardial tone and weak cardiac contraction in a fifteen-year-old patient with atrial septal defect and anomalous pulmonary venous drainage to right atrium. Hypothermia (30.0 C) and cyclopropane anesthesia. Closure of atrial septal defect. Following closure the patient developed ventricular fibrillation. Circulation occluded five minutes. (See also figure 10.)

features relating alveolar carbon dioxide tension to critical change in pulmonary blood flow. The rapidity with which critical change in dangerously low pulmonary blood flow is reflected in tracings of carbon dioxide tension is remarkable. For example, in figure 1 there is a decline in alveolar carbon dioxide tension (P_ACO_2) from 21.24 mm. of mercury to 2.83 mm. of mercury subsequent to severe, abrupt systemic

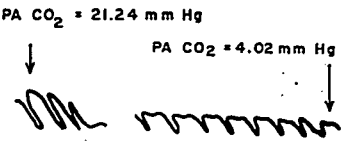

FIG. 3. P_ACO_2 decline from 21.24 mm. of mercury to 4.02 mm. of mercury during occlusion of pulmonary artery in a seven-year-old patient with congenital pulmonary stenosis and accessory superior vena cava. Hypothermia (28.5 C) and cyclopropane anesthesia. Supravalvular pulmonary valvulotomy and ligation of accessory superior vena cava.

hypotension. Also, figure 2 illustrates an abrupt decline in P_ACO_2, from 28.32 mm. of mercury to 10.6 mm. of mercury, in this instance, following a fall in cardiac output secondary to poor myocardial tone and weak cardiac contraction. Figures 3 and 4 indicate more directly decline and increase respectively, in P_ACO_2 during occluded pulmonary circulation and following restoration of pulmonary blood flow. The carbon dioxide tracing in figure 5 (lowered alveolar plateau curves)

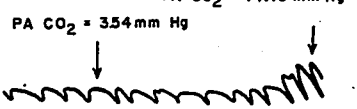

FIG. 4. P_ACO_2 increase from 3.54 mm. of mercury to 14.16 mm. of mercury on reopening clamped pulmonary artery, in a three-month-old patient with tetralogy of Fallot. Hypothermia (30.8 C) and cyclopropane anesthesia. Blalock procedure.

differs from that in figure 6 (absent alveolar plateau curves) during the complete occlusion of inflow and outflow vascular tracts because ventilation was continued in the former and not in the latter. Both figures demonstrate decline in P_ACO_2 during occlusion of systemic circulation. On several occasions concern for circulatory adequacy because of failure of alveolar carbon dioxide tracings to revert to former

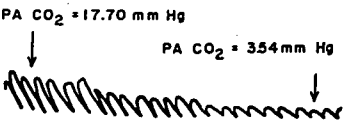

FIG. 5. P_ACO_2 decrease from 17.70 mm. of mercury to 3.54 mm. of mercury following occlusion of systemic circulation with ventilation continued in a nine-year-old patient with atrial septal defect. Hypothermia (29.0 C) and cyclopropane anesthesia. Closure of atrial septal defect. Digitalized prior to surgery. Circulation occluded 6 minutes, 45 seconds. (See also figure 8.)

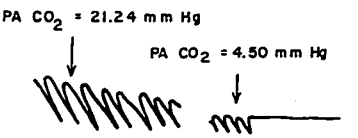

FIG. 6. P_ACO_2 decline from 21.24 mm. of mercury to 4.50 mm. of mercury following occlusion of systemic circulation without ventilation in a fourteen-year-old patient with atrial septal defect. Hypothermia (30.5 C) and cyclopropane anesthesia. Closure of atrial septal defect. Circulation occluded 4 minutes, 32 seconds.

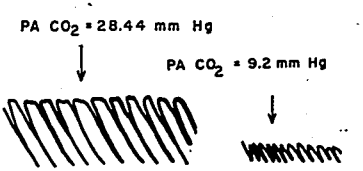

FIG. 7. P_ACO_2 decline from 28.44 mm. of mercury before occlusion to 9.2 mm. of mercury following release of occluded circulation prior to and during ventricular fibrillation in a ten-year-old patient with congenital aorticopulmonary fenestration and extensive pericardial adhesions, secondary to previous exploratory thoracotomy. Hypothermia (30.0 C) and cyclopropane anesthesia. Attempted closure of aorticopulmonary fenestration. Death from massive hemorrhage from tear in pulmonary artery. (See also Figure 9.)

levels following release of occluded circulation preceded similar conclusions from clinical evaluation by several minutes. For example, in figure 7, the P_ACO_2 was 28.44 mm. of mercury before occlusion of systemic circulation. During occlusion the P_ACO_2 was negligible. Following restoration of circulation the P_ACO_2 was only 9.2 mm. of mercury. Ventricular fibrillation soon followed. In contrast, figure 8 il-

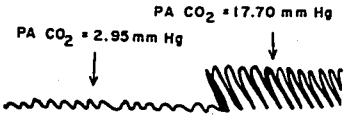

FIG. 8. P_ACO_2 increase from 2.95 mm. of mercury to 17.70 mm. of mercury in a patient when occluded systemic circulation was restored. (See also figure 5.)

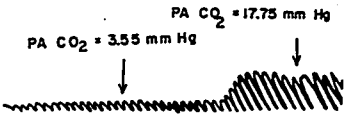

FIG. 9. Alveolar carbon dioxide tension (P_ACO_2) increase from 3.55 mm. of mercury to 17.75 mm. of mercury in a patient during effective cardiac massage. (See also figure 7.)

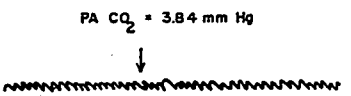

FIG. 10. P_ACO_2 decline from 28.32 mm. of mercury to 3.84 mm. of mercury during ineffective cardiac massage. (See also figure 2.)

lustrates an abrupt return of P_ACO_2 of 2.95 mm. of mercury during occlusion to preocclusive tensions of 17.70 mm. of mercury subsequent to restoration of systemic circulation. Effective cardiac massage was reflected in carbon dioxide tension approximating normal (fig. 9) and the values are much lower when cardiac massage is ineffective (fig. 10).

These observations coincide with physiologic relationships between ventilation and pulmonary blood flow (9). They are primarily in the category of clinical conditions demonstrating the uniform ventilation-nonuniform blood flow variety of ventilation-blood flow ratio. The anesthesiologist who interprets only the uniformity of ventilation from P_ACO_2 values will fail to recognize cases in this category.

We have demonstrated a sensitive, practical, inexpensive method for evaluating circulatory integrity in certain critical situations.

SUMMARY

By means of continuous alveolar carbon dioxide analysis in anesthetized patients during heart and great vessel surgery, certain critical relationships between pulmonary blood flow and alveolar carbon dioxide tensions were noted. With adequate uniform ventilation and diffusion, alveolar carbon dioxide declines when pulmonary blood flow falls below a critical point. This may occur when systemic hypotension occurs from severe, abrupt pulmonary artery hemorrhage, when

cardiac output declines secondary to poor myocardial tone and weak cardiac contraction, during occlusion of systemic circulation, during ventricular fibrillation, with ineffective cardiac massage, and with occlusion of pulmonary circulation. Adequate alveolar carbon dioxide tension is restored when pulmonary blood flow rises above the critical level as the occluded systemic circulation is reopened, during effective cardiac massage, and when a clamp on a pulmonary artery is released.

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