Automatic End-Tidal Gas Sampling System for Non-Invasive rCBF Measurements

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SUMMARY Non-invasive measurements of regional cerebral blood flow ($^{133}$Xe inhalation or IV injection) require knowledge of the arterial $^{133}$Xe concentration, which is usually estimated from the end-tidal expiration. An automated sampling system is described for measurement of end-tidal $^{133}$Xe concentration that can be used in both spontaneously breathing and mechanically ventilated patients. A comparison is made between the automatic sampling system and manual measurements performed on a continuous gas sample.

MEASUREMENT OF regional cerebral blood flow (rCBF) by $^{133}$xenon inhalation or IV injection requires that changes in the arterial concentration of the tracer be known in order to correct for recirculation. In practice, it is assumed that $^{133}$Xe in the end-tidal expiration represents alveolar, and therefore, arterial concentration. The end-tidal $^{133}$Xe concentration is usually determined by manual or computer analysis of continuous gas samples. Recently, an automated system for discontinuous sampling of the end-tidal gas has been developed for use in spontaneously breathing patients. We have designed and used an automatic end-tidal sampling system which can be employed in both spontaneously breathing patients and in critically ill individuals requiring mechanical ventilation.

Technique

The system employs a modified anesthesia valve through which airway pressures are measured and the end-tidal sample taken. Sampling is intermittently accomplished by a specially designed timing circuit connected via a solenoid valve to a vacuum pump. An in-line scintillation counter is employed to measure the activity of the $^{133}$Xe contained in a small lead-shielded glass helix.

Figure 1 shows the modifications performed on an Ohio Swivel “Y” one-way valve to permit triggering of the solenoid valve by either negative changes in airway pressure initiated by spontaneous breathing, or by the positive inspiratory pressure of a mechanical ventilator. Continuous registration of expired CO$_2$ is made possible through a second tube inserted between the rubber diaphragms of the one-way valve and extending down to face level within the anesthesia mask or inserted into the endotracheal tube. The $^{133}$Xe sample is taken by means of a third port located immediately distal to the downstream diaphragm of the valve.

Samples taken from this location represent end-tidal or alveolar gas insofar as it is “trapped” at the end of expiration. It is assumed that a reasonable degree of stratification of gas remains within the expiratory limb of the breathing circuit.

A schematic diagram of the automated end-tidal sampling network is presented in figure 2. The system operates in two distinct modes relative to spontaneous or controlled ventilation. During spontaneous ventilation the patient breathes into a snugly fitting anesthesia mask equipped with head straps. The negative pressure generated by the initiation of inspiration is transmitted to the negative side of a pressure-sensitive switch, which passes a signal to the sample timing device (see fig. 5 for schematic of the timer). This event indicates the presence of a “trapped” end-tidal gas sample in the expiratory limb of the breathing circuit, and initiates the withdrawal of gas into the radioactivity analysis cell. The volume of gas sampled is determined by the length of time that the 3-way solenoid valve remains open to the patient, and by the flow rate (1000 ml/min) provided by the vacuum pump. In our system, sampling continues for 0.3 seconds (adjustable up to 2 sec), which results in a flow sufficient to flush the dead space of the tubing in the scintillation counting circuit 3 to 4 times. This washout provides better than 90% resolution of the expired air curve. Sampling for more than 1.3 seconds leads to dilution of the end-tidal gas.

The same signal which actuates gas withdrawal in the spontaneously breathing patient also activates an “inhibit” circuit within the same timer. This “inhibit” prevents additional triggering of the circuit during a normal sampling cycle. It is required for rejection of false triggers of the system by the patient coughing, etc. The “inhibit” period is under variable control, and is usually slightly less than the patient’s normal respiratory cycle time. When not sampling gas from the patient, the vacuum pump remains open to room air. This is necessary to avoid irregular flow patterns due to pump start-up inertia and/or variable loading of the pump.

Only a few simple changes in the automated gas-sampling system are needed to make rCBF measurements in mechanically ventilated patients. The ventilator applies a positive pressure to the inspiratory limb of the sampling valve, and this causes both rubber diaphragms in the one-way valve to open. This converts the one-way valve system to a simple “T” connector, and the valve system intrinsic to the mechanical respirator takes over control of ventilation (see fig. 2). Therefore, the uni-directional valve arrangement with its sampling and pressure-sensing ports is in the system during both spontaneous or controlled ventilation. Timing of the ventilatory cycle is controlled by the respiratory settings (in our case, a Bennett MA-1), which is regular if adequate muscle relaxation and/or sedation are employed.

Because the one-way valve is open during positive insufflation of the patient, a valid end-tidal sample cannot be obtained during inspiration. To perform automatic end-tidal sampling in such circumstances, the positive pressure during...
Inspiration and expiration are continuous processes that can be monitored using various apparatuses. The diagram illustrates a non-rebreathing valve modified to permit continuous measurement of end-tidal CO₂ (F₄₇CO₂) and airway pressure between the two diaphragm valves of the assembly. The port for withdrawal of the end-tidal ⁱ³³Xe sample (F₄₇¹³³Xe) is located on the expiratory side, just beyond the rubber valve. Arrows indicate the direction of gas flow in the breathing circuit.

The control panel of the sample timing apparatus is shown in figure 3. The delay or inhibit times are adjustable following initiation of a signal from the diaphragm switch. The delay-inhibit mode of operation for either controlled or spontaneous respiration is selected via a switch. The volume of gas drawn into the ¹³³Xe analysis cell is determined by adjusting the sample timer control.

Inspiration is transmitted to the positive sensing side of the pressure switch and thence to the sample timer. Receipt of the inspiratory signal initiates a delay timer which is under variable control. The delay period is set so that the end-tidal sample is taken immediately prior to initiation of the next controlled breath, when expiration is essentially complete. Sample duration is timed as described for spontaneous respiration.

The control panel of the sample timer is shown in figure 3. Both the "inhibit" and "delay" controls use the same timing circuit and the mode of operation is chosen with a switch. Two light-emitting diodes are incorporated into the control panel to indicate when sampling or inhibit-delay periods occur. A variable tone indicator is also included to provide the same information.

A comparison of ¹³³Xe concentration curves simultaneously determined by manual analysis of continuous gas samples and by the automatic sampling system in both spontaneously (A) and mechanically (B) ventilated patients is shown in figure 4. The curves are normalized to 100% peak count rate. Continuous sampling was performed through the port usually employed for end-tidal CO₂ determination.
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Figure 2. Schematic diagram of the automatic end-tidal 133Xe sampling system. The circuit for continuous end-tidal CO₂ sampling is not shown. The negative and positive pressure detecting sides of the diaphragm switch are respectively indicated by N or P. See text for further details.

Results
Thus far, we have performed 124 rCBF measurements in 35 spontaneously breathing patients. Twenty-six additional rCBF studies have been carried out in 12 mechanically ventilated patients. Since its inception, the sampling system has had no failures. Figure 4 compares end-tidal 133Xe curves obtained by manual analysis of continuous gas samples with those simultaneously obtained by automatic sampling. The data were obtained from both spontaneously breathing and mechanically ventilated patients. There is excellent agreement between the contours of the normalized curves obtained by the two sampling methods.

Figure 4. Comparison of end-tidal 133Xe curves obtained by manual (A) and automatic (B) sampling. The data were obtained from both spontaneously breathing and mechanically ventilated patients. There is excellent agreement between the contours of the normalized curves obtained by the two sampling methods.

Figure 5. Electronic circuit diagram for the sample timer. A full list of the components of the automated system, with their commercial sources, is available from the authors upon request.
Cardiac Arrhythmias in Experimental Subarachnoid Hemorrhage

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SUMMARY Experimental subarachnoid hemorrhage (SAH) in dogs was produced by introducing blood into the subarachnoid space through a catheter connected to an artery of the animal. The intact animals and those with preserved vagi and heart sympathetic innervation, developed arrhythmias with short latencies which correlated with the sudden increase in the intracranial pressure. The animals with sections of both vagi and heart sympathetic innervation, but with an intact spinal cord, developed arrhythmias that were delayed and did not correlate with the changes in intracranial pressure. These arrhythmias were preceded by changes in the QT interval, T wave and ST segment. It was concluded that the arrhythmias could be produced either by direct autonomic discharges to the heart or by increased circulating and tissue catecholamines. The clinical implications of these findings are discussed.

IN 1930, BEATTIE et al. published a series of remarkable observations showing that extrasystolic arrhythmia, induced by chloroform anesthesia in the cat, could be prevented either by the combination of sectioning sympathetic nerves to the heart and removal of the adrenal glands or by destruction of the hypothalamus. This important work was largely ignored and it is only now that its relevance can be fully appreciated.1 That cardiac arrhythmias could occur as a consequence of acute cerebral lesions, was suspected in 1936 when Bramwell published a paper with the question: “Can head injury cause auricular fibrillation?” Although later reports mentioned the presence of cardiac arrhythmias in subarachnoid hemorrhage2-4 the fact that these could be life threatening was not emphasized until 1973 when ParizeF described two patients with ventricular tachycardia. He made the point that these patients might have died had they not been correctly diagnosed and treated in a coronary care unit when the arrhythmia occurred. A recent report has emphasized the severity of subarachnoid hemorrhage (SAH) with associated cardiac dysrhythmia and the high incidence of sudden unexpected death in primary subarachnoid hemorrhage presumably due to arrhythmia.5 In 1947, Byer et al.6 demonstrated that abnormal ECGs characterized by large upright T waves and prolonged QT intervals, could be seen in patients suffering from acute intracranial diseases, particularly subarachnoid hemorrhage secondary to ruptured aneurysms. Since that time, several authors have confirmed and extended their observations.7-14 Since the original description by Beattie et al., a number of experimental observations have confirmed the fact that sympathetic and perhaps vagal discharges were responsible for the disorders of the cardiac rhythm. The arrhythmias have been produced by electrical stimulation to different parts of the brain, including hypothalamus, mesencephalon, the orbital surfaces of the frontal lobes, insula and other areas.10-19 Arrhythmias can also be induced by stretching vessels of the circle of Willis20 or by producing sudden increase in the intracranial pressure with subdural balloons.6 It has been shown that acute hypoxia during mesencephalic stimulation precipitates these changes.18 It has been shown that patients and animals dying after intracranial hemorrhage had subendocardial lesions presumably secondary to excessive sympathetic stimulation.21-25 Offerhaus and Van Gool showed convincingly...
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