What can SIMV and PSV provide?

While both methods provide assistance to the spontaneously breathing patient, they differ slightly. SIMV provides for a minimum, assured respiratory rate and minute volume. PSV depends on the patient's own respiratory rate as a trigger and by providing a ventilatory assist through pressurizing the airway, relieves some of the work of breathing.

Both SIMV and PSV provide added volume to the patient's minute ventilation and, though they differ in whether they assure rate or pressure, the end result is that part of the work of breathing is assumed by the ventilator.

With the help of the ventilator:

1. Minute volume can be maintained or enhanced
2. Normocapnea can be maintained
3. Newer airway devices can be employed and still provide ventilatory support.

Additional Reading

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Clinical Focus

by Datex-Ohmeda

Assisted Ventilation

It All Adds Up!

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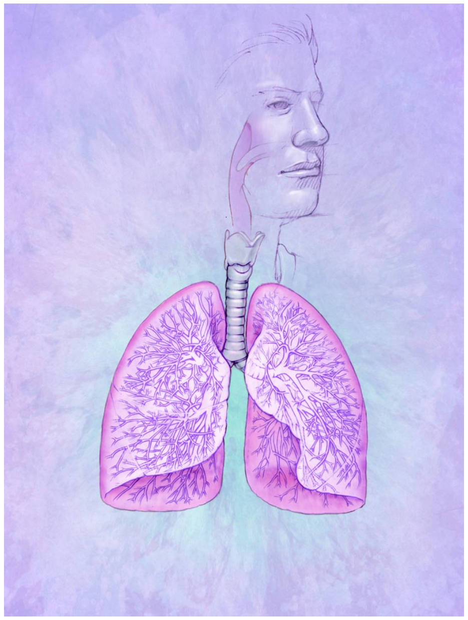

From the Ventilation Series

Assisted Ventilation: It All Adds Up!

Over the past two decades the use of Laryngeal Mask Airways (LMA) and Cuffed Oral-pharyngeal Airways (COPA) have changed the way mechanical ventilation and spontaneous breathing are viewed; patients are allowed to breathe spontaneously during some or all of a general anesthetic. However, assuring adequate ventilation, while allowing a patient to breathe spontaneously, is more complicated than it may initially appear. This *Clinical Focus*, produced by the Department of Clinical Affairs, is designed to review some of the considerations that general anesthesia and spontaneous breathing place on each other and suggest some solutions to the problems presented.

What effect does general anesthesia have on breathing?

When inhalation anesthesia was new, allowing patients to breathe was not only common, it was essentially the only way anesthesia was delivered. Ether was thought to be auto-regulating. As the level of anesthesia deepened, breathing became shallower. As a patient got "lighter" the breathing would become deeper. This regulation tended to produce a relatively stable depth of inhalation anesthesia.

Modern inhalation agents are quite different. This is true not only of their composition, but also with respect to their effects on breathing. Halothane, for instance, actually increases the rate and essentially compensates for the decreased tidal volume that accompanies its administration. Normocapneic patients under halothane anesthesia can easily maintain adequate minute ventilation. However, the addition of intravenous narcotics, sedative or muscle relaxants to inhalation anesthesia may suppress breathing and cause hypoventilation.

Sevoflurane, the modern replacement for halothane, also produces a decrease in tidal volume but without the compensatory increase in respiratory rate. Sevoflurane may produce hypercarbia resulting from the combined effect this agent has on the brainstem, spinal cord, and peripheral chemoreflexes. The addition of pre-operative sedation, narcotics, or relaxants, increase the need to assist ventilation to counter CO_2 retention, prevent apnea, and maintain adequate levels of inhalation anesthesia.

Beyond the effects these agents have on the chemoreflexes and pneumotaxic centers, general anesthesia also reduces functional residual capacity (FRC). When FRC is reduced, oxygenation is reduced, atelectasis encouraged, and inhalation agent distribution and diffusion is decreased.

Differences between inhalation anesthesia and balanced anesthesia.

While both techniques can produce a quiescent patient, the effect that each has on the respiratory centers is quite different. Narcotics, as either the sole analgesic portion of an anesthetic or as an element of balanced anesthesia, produce a dose-related decrease of the centrally controlled respiratory rate. Inhalation agents, on the other hand, produce effects on the central respiratory center as well as effects on the spinal, carotid, and peripheral chemoreflexes. These effects exhibit patient-to-patient variability reflected in the absence of a direct relationship between depth of anesthesia and decreased breathing, as reflected in the rise in end-tidal carbon-dioxide ($P_{et}CO_2$) levels. A rising $P_{et}CO_2$ relates to the degree of ventilatory depression from all sources as well as the patient-specific responses to these.

Studies have suggested that patients may respond more appropriately to changes in CO_2 levels only during periods of relative normocapnea. At high $P_{et}CO_2$ levels the respiratory drive is severely blunted, CO_2 itself producing a narcotic-like effect. Additional studies have demonstrated that the local effect of inhalation agents on the muscles of breathing differs from the central effect. The end result is that the effect on muscle tone and contractility, translated into V_t, is more profound than the effect on the central CO_2 response alone.

Key questions remain

When considering the choice of spontaneous breathing, key questions arise. How can adequate breathing volumes be assured? How can adequate gas exchange be assured? Will assisting a patient prolong the patient's recovery from anesthesia phase?

Studies have shown that maintaining a patient's normal $PaCO_2$ levels, as reflected in $P_{et}CO_2$ values, provides the greatest level of anesthesia control and the best emergence results. The relative depressant effect of inhalation anesthesia on the medullary centers ends with the elimination of the inhalation anesthetic.

In addition to the clinical questions, the technical question is "what kind of ventilation mode can assist patient breathing without being competitive?" Synchronous Intermittent Mandatory Ventilation (SIMV) and Pressure Support Ventilation (PSV) are two possible answers to this technical question.