effect on the metabolic regulation of breathing. In the seven studies quoted above, however, it was taken to be, in effect, the sole index of overall ventilatory control. If the CO₂ response is to be used and interpreted in this way, it may be important to consider uncertainties about the test and about the relationship of the response to fundamental variables of ventilatory control—resting ventilation and P_{CO₂}.

The CO₂ stimulus of conventional CO₂ response tests is not relevant to most physiologic circumstances, and the response may not be the same as the response to more physiologically pertinent types of CO₂ stimuli. The magnitude of response varies considerably among and within individuals, and this variation does not correlate with resting minute ventilation or end-tidal P_{CO₂}. Effects of drugs on the sensitivity of response appear not to parallel effects on resting breathing and P_{CO₂} in a consistent way—differing quantitatively (e.g., thiopental, morphine, and individual halogenated hydrocarbons) and sometimes even qualitatively (e.g., ether, nitrous oxide and ketamine). For these and other reasons, effects on the CO₂ response may not be sufficient in themselves for conclusions about how drugs affect relevant aspects of ventilatory control.

In pharmacologic studies of breathing, data on the response to added CO₂ should be supplementary to values of resting ventilation and end-tidal and/or arterial P_{CO₂}. Careful measurements of resting variables always should be reported, no matter how crude or insensitive they might seem. This is crucial and relevant information we need to know.

Let's wrest, not rest, resting ventilation!

R. L. KNILL, M.D., F.R.C.P.(C)
Associate Professor
Department of Anesthesia
University of Western Ontario
London, Ontario, Canada, N6A 5A5

REFERENCES


(Accepted for publication May 25, 1983.)

Further Suggestions on Epidural Spread in Pregnancy

To the Editor:—In their report, "Spread of Epidural Analgesia in Early Pregnancy," Fagraeus et al., state that the compensated alkaloisis secondary to hyperventilation during pregnancy favors an increase in the ionized form of local anesthetic agents as compared with the nonionized form (i.e., free base). This argument is used to explain the increased rostral spread of a given dose of local anesthetic agent used for epidural anesthesia in the pregnant state.

Bonica has pointed out that pregnancy results in an incompletely compensated respiratory alkalosis. This has been defined more specifically by Sjostedt as a rise in pH of 0.02 by the tenth week of pregnancy. For a given dose of local anesthetic, the ratio of ionized to un-ionized form is determined uniquely by the PKₐ and pH through the Henderson-Hasselbach equation. For lidocaine a change in pH from 7.40 to 7.42 will increase the un-ionized form from 26.19 to 27.09% a
rise of 3.4%. The ionized form therefore will be decreased by the same amount.

The increased free base form of the local anesthetic agent could traverse the dura and lead to a higher level of epidural blockade in the pregnant patient. It might also explain the initial faster spread of the block reported.

MITCHEL SOSIS, M.D., PH.D.
ARNOLD BONNER, M.D.
Department of Anesthesiology
St. Barnabas Medical Center
Livingston, New Jersey

REFERENCES


(Accepted for publication May 28, 1983)

Corrections Concerning Altered Disconnect Alarm Failure

To the Editor:—The recently published letter by Reynolds1 regarding an alleged failure of a Drager DPM® requires some correction and clarification. The DPM® (Drager Pressure Monitor, not disconnect pressure monitor as incorrectly stated) is a patient system pressure monitor that produces an audible and visual warning when the peak pressure in the system falls to exceed the dialed-in monitoring pressure within a period of nominal 15 s.

Due to the fact that the pressure in the system during the reported incident obviously exceeded the dialed-in monitoring pressure (9 cmH2O) within the preset time period, the alarm function of the monitor was not actuated. The conclusion by the author that the monitor failed is, therefore, incorrect.

It can be assumed, however, that the incident was caused by a failure of the operator to follow the instructions of the Instruction Manual when dialing in the alarm level. The instruction manual clearly states (and explains with various examples) that a setting of 5 cmH2O monitoring pressure should be used only if the peak pressure in the system is below 15 cmH2O. While the information concerning the pressure, flow, resistance, and compliance conditions in the report concerning the incident itself and the following tests are incomplete, the findings lead to the conclusion that the DPM® was used incorrectly.

The conclusion of the author that North American Drager changed the lowest setting from 5 cmH2O to 7.5 cmH2O on subsequent models of the DPM® (DPM2 and DPM-S) in recognition of a shortcoming is incorrect. The increase of pressure for the lowest setting was necessary to establish overlapping of pressure sensors in the unit to utilize a self-diagnostic circuit that reveals malfunctioning of the circuitry.

While separation of the 15-mm connector from the Y-piece is the most common cause for circuit disconnects, the separation of the 15-mm connector and tube is rather uncommon. In our opinion, it would have been worthwhile to investigate why this uncommon disconnection took place and if, possibly, an undersized connector was used in the circuit.

The reported incident clearly reveals the necessity to follow manufacturer’s instructions when operating life-supporting equipment. Reference is made to North American Drager’s information in the January issue of ANESTHESIOLOGY entitled “Overcoming the Disconnect Hazard,” which contains a clear warning concerning the habit of leaving pressure monitors at the lowest pressure setting.

PETER J. SCHREIBER
President
North American Drager
148B Quarry Road
Telford, Pennsylvania 18969

REFERENCE


(Accepted for publication May 23, 1983)