

tories of CO_2 retention had significant attenuation of the ventilatory responses to CO_2 compared with controls. Pulmonary function data from Pietak's patients¹ with COPD indicate that neither hypoxemia nor hypercapnia was present. Thus, one would be reasonably accurate in ascribing the basic alterations in their ventilatory responses to CO_2 , awake or anesthetized, to abnormal respiratory mechanics. The role of a greatly altered central nervous system response to CO_2 in these patients seems unlikely.

Many of the adverse effects of anesthesia on respiration result from the pattern of ventilation common with most inhalational anesthetics, namely, reduced tidal volume and increased respiratory rate. Although the magnitude of these changes is roughly similar in COPD patients,¹ the physiologic impact is greater. The diminished tidal volumes render ventilation less efficient in both groups of patients, but the increased respiratory rate helps to compensate somewhat in normals. However, in patients with airway obstruction it exacts a serious price in the form of reduced dynamic compliance, non-uniform gas distribution, and increased resistance to breathing. Thus, the wasted ventilation as well as the work of breathing increases significantly.

The authors¹ state that controlled or assisted ventilation is mandatory in patients with COPD. One might further recommend the pattern of ventilation. Slow deep breathing

is effective in improving gas exchange in COPD patients, compared with their usual rapid, shallow ambient breathing patterns.⁵ A similar approach to controlled ventilation, with paralysis if necessary, seems appropriate in patients with significant airflow obstruction. This, of course, does not preclude close monitoring of Pa_{CO_2} , as would be appropriate with any ventilatory mode in these patients.

THOMAS J. GAL, LCDR MC USNR
Department of Anesthesia
Naval Regional Medical Center
Portsmouth, Va. 23708

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Gas Embolism during Pneumoencephalography

To the Editor:—We read with interest the case report "Air embolism through a ventriculoatrial shunt during pneumoencephalography" by Youngberg, Kaplan and Miller (*ANESTHESIOLOGY* 42:487, 1975), as we recently encountered a similar case.

A 16-year-old boy, who four years previously had had a pinealoma removed, developed headache and anorexia. Increase in cerebrospinal fluid (CSF) pressure was suspected, even though a ventriculo-peritoneal shunt

had been placed at the time of operation. In preparation for ventriculography the patient was sedated with Innovar and a ventriculostomy was performed using local anesthesia. The ventricular catheter was attached to a pressure transducer and CSF pressure was continuously recorded. Oxygen was used as the contrast medium. Following an exchange of 50 ml CSF with oxygen, nitrous oxide inhalation was administered in 50 per cent concentration as part of an experimental study

to determine the effect of the anesthetic on CSF pressure. While our previous experience had shown CSF pressure to increase, in this instance no change in CSF pressure was found. A radiograph of the thoraco-abdominal area revealed gas within the ventriculo-peritoneal catheter.

The events of this case indicate that evaluation of ventriculo-peritoneal catheter patency could be made with this procedure. They also emphasize that using nitrous oxide in patients with ventriculoatrial shunts may be hazardous. Had Youngberg and his colleagues administered nitrous oxide to their patient following air encephalography, diffusion of nitrous oxide

into this air space would have resulted in a significant increase in intracranial gas volume. This undoubtedly would have increased the severity of the venous gas embolism described, since greater transfer of gas to the right heart through the ventriculoatrial catheter probably would have occurred.

WILLIAM L. PAUL, M.D.
EDWIN S. MUNSON, M.D.
*Department of Anesthesiology
University of Florida College of Medicine
Gainesville, Florida 32610*

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Pediatric Anesthesiology Consultant Program

To the Editor:—The Section on Anesthesiology of the American Academy of Pediatrics is co-sponsoring a program of visits to community hospitals by pediatric anesthesiologists acting as consultants from nearby children's hospitals. Each visit will be for two days, with the morning of day 1 and late afternoon of day 2 for travel by the consultant. Each consultant will give two talks, in addition to participating in informal discussions. The community hospital will also be asked to arrange for the morning of the second day a conference at which topics such as ventilatory care for neonates, advances in pediatric anesthesia, respiratory treatment of pediatric patients, temperature maintenance and transport of sick infants, and evaluation of new equipment will be discussed.

Either one or two pediatric consultants will go to each community hospital. The Section on Anesthesiology will pay the honorarium for the consultant, but the hospital, the medical society, or the local community will be asked to pay the consultant's expenses.

The program will initially involve consultants in five areas: 1) Massachusetts General Hospital, Boston, John Ryan, M.D.; 2) Hospital for Sick Children, Toronto, I. A. Sloan, M.D.; 3) Philadelphia Children's Hospital, Philadelphia, John Downes, M.D.; 4) Akron Children's Hospital, Akron, Donald Nelson, M.D.; University Hospitals of Cleveland, Cleveland, Robert Crumrine, M.D.; 5) Children's Orthopedic Hospital, Seattle, Eric Furman, M.D.

Later, additional institutions will also participate. Those interested in availing themselves of this consultant program should contact the individuals listed above if they wish them to visit a community hospital in the area.

LIDA S. DAHM, M.D.
*Vice Chairman and Treasurer
American Academy of Pediatrics
Section on Anesthesiology
(1403 Skirvanek Court,
College Station, Texas 77840)*

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