CORRESPONDENCE

Volume Monitor Malfunction Caused by an Overhead Operating Room Light

To the Editor:—Respiratory volume monitors are standard equipment on most anesthesia machines in use today. Although monitor malfunctions have been reported in the past, a new malfunction occurred during anesthesia for an anterior cruciate ligament repair. General anesthesia was induced and the trachea intubated in an uneventful fashion. Since this procedure requires the use of arthroscopy, the room lights were turned off and one of the overhead operating room lights was used to light the anesthesiologist’s area. The Ohmeda 5420 Volume Monitor began to alarm and indicated tidal volumes of 200 ml to 6 l without a change in ventilator settings. The lungs were manually ventilated and breath sounds, pulse oximetry, and end-tidal CO2 monitors verified adequate ventilation. The existing monitor and transducer were replaced with an identical system; however, the new monitor continued to display widely varying readings. It was noted that the overhead OR light was focused on the turbine vane transducer in the expiratory limb of the anesthesia circle. When the light was directed away from the transducer, accurate tidal volumes were displayed.

The Ohmeda 5420 Volume Monitor functions by using a turbine vane transducer affixed to the expiratory limb of the anesthesia machine to convert gas flows into electrical pulses for a microprocessor. This conversion is accomplished through the use of optical sensing circuits located within the transducer. Each sensing circuit has an infrared light emitting diode and a photosensitive detector that detects interruptions caused by gas flow through the rotating vane. These interruptions are converted by the microprocessor to tidal volume or minute ventilation. The high intensity OR light interfered with the photosensitive detector, causing the monitor to display inaccurate tidal volumes. Redirecting this intense light away from the photosensitive detector enabled it to function accurately.

TRACY P. COTTER, M.D.
Resident Physician

GEORGE L. BUSH, M.D.
Associate Professor

Department of Anesthesiology, B6 / 387
Clinical Science Center
600 Highland Avenue
Madison, Wisconsin 53792

REFERENCE


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Pain Control during Extracorporeal Shock Wave Lithotripsy of Gallstones by Titrated Alfentanil Infusion

To the Editor:—Shock wave lithotripsy of gallbladder stones has been used in more than 600 humans at our institution since 1985. Despite major technical improvements, gallstone lithotripsy employing Dornier’s new MPL 9000 non-immersion lithotripter still causes considerable discomfort. Analgesia with intravenous pentazocine or meperidine may be inadequate. On the other hand, due to the noninvasive character of biliary ESWL, general or epidural anesthesia may be excessive in terms of pain control for the average patient.

We therefore studied an alternative anesthetic approach using an alfentanil infusion titrated to the analgesic needs of the conscious and spontaneously breathing patient. In order to analyze degree and distribution of pain during gallstone lithotripsy and to evaluate pain control by titrated alfentanil infusion, we studied 44 consecutively treated patients with no previous ESWL therapy.

Blood pressure, heart rate, cutaneous $\rho_{\text{O}_2}/\rho_{\text{CO}_2}$, and oxygen saturation using a pulse oximeter were continuously recorded. Pain intensity during shock wave application was evaluated according to a five-point verbal rating scale which ranged from 0 = no pain to 4 = unbearable pain.

All patients were treated in the prone position. After stone localization and imaging, oxygen (6 l/min) was given by face mask and an alfentanil infusion at an initial rate of 2.5 $\mu$g · kg$^{-1}$ · min$^{-1}$ was started. Single shock waves were begun as test shocks after approximately 2 min. If well tolerated by the patient, stone fragmentation was begun. If not, more alfentanil was allowed to accumulate until continuous treatment was tolerated. Further increases or decreases of the infusion rate were titrated according to patient response. We recorded median pain scores and corresponding alfentanil dose-rates over the time course of treatment. $P_0$ indicates baseline values before shock wave release with the patient in prone position. At point $P_1$, tolerance of test shocks terminates the initial loading dose of alfentanil. Points $T_1$ to $T_6$ are at 5, 10, 20, 30, 40, and 50 min, respectively, after beginning of continuous shock wave application. Median and maximum values of verbal pain scores are shown: 4 indicates unbearable pain, 3 severe pain, 2 moderate pain, 1 mild pain, and 0 no pain. The minimum values of pain were 0 at all points (not shown); * indicates $P < 0.05$ compared to $P_0$ and $T_1$.

FIG. 1. Verbal pain scores and corresponding median alfentanil dose-rates over the time course of treatment. $P_0$ indicates baseline values before shock wave release with the patient in prone position. At point $P_1$, tolerance of test shocks terminates the initial loading dose of alfentanil. Points $T_1$ to $T_6$ are at 5, 10, 20, 30, 40, and 50 min, respectively, after beginning of continuous shock wave application. Median and maximum values of verbal pain scores are shown: 4 indicates unbearable pain, 3 severe pain, 2 moderate pain, 1 mild pain, and 0 no pain. The minimum values of pain were 0 at all points (not shown); * indicates $P < 0.05$ compared to $P_0$ and $T_1$. 1022
Pain during shock wave lithotripsy of gallstones is highly variable and appears to be related to the quantity of shock wave energy applied. Alfentanil, a rapidly distributed and eliminated synthetic opioid, is ideally administered to spontaneously breathing patients by titrated infusion rather than by intermittent bolus injections. It will not, however, replace general or epidural anesthesia in certain high-risk patients or in those suffering from mental or emotional disorders.

GUSTAV SCHELLING, M.D.
Resident in Anesthesia

WERNER WEBER, M.D.
Staff Anesthesiologist

MICHAEL SACKMANN, M.D.
Staff Internist

KLAUS PETER, M.D.
Professor and Chairman
Department of Anesthesiology
Klinikum Großhadern
Ludwig-Maximilians-University
Munich, FRG

REFERENCES


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To the Editor:—Massaguee et al.1 as well as Schwin et al.2 have shown that the pressor effect of phenylephrine is significantly greater during cardiopulmonary bypass and aortic cross-clamp (CPB + AXC), compared with that during the prebypass period. The increased potency of phenylephrine during CPB + AXC has been attributed to hypothermia, hemodilution, increased plasma catecholamines, exclusion of pulmonary circulation, and, possibly, altered PH pharmacokinetics.1,2

In contrast to phenylephrine PH, we have previously shown that ephedrine produces less vasopressor response during CPB + AXC compared with that achieved off-bypass.5 The injection of ephedrine 50 mg during CPB increased the MAP by about 30%, but decreased the reservoir volume in the oxygenator, suggesting that in humans ephedrine administration results in a moderate increase of systemic vascular resistance (SVR), associated with dilatation of the capacitance vessels. We also have shown that epinephrine 10–20 μg produces a greater vasopressor response when injected during the prebypass period than during CPB + AXC.*

* Baraka A: Response to vasopressors before and during cardiopulmonary bypass. Unpublished data.

The different response to vasopressors during CPB compared with that achieved without CPB may be attributed to the different events occurring during CPB, such as hemodilution, hypothermia, and increased endogenous catecholamines. Initiation of cardiopulmonary bypass results in an initial decrease of SVR due to hemodilution, to be followed by a gradual recovery and progressive increase of SVR that may be attributed to induction of hypothermia and increased catecholamine release.4 Circulating catecholamines are known to increase during CPB.5 Also, hypothermia may increase blood viscosity and induce vasoconstriction.6 Thus, both hypothermia and increased endogenous catecholamine release may explain the augmented vasopressor response to phenylephrine during hypothermic CPB.1,8 However, they fail to explain the decreased vasopressor response to ephedrine and epinephrine. Also, during CPB, the priming volume is added to the intravascular volume of the patient, and may lead to dilution of the administered medication. Although hemodilution may explain the decreased vasopressor response to epinephrine and epinephrine during CPB, it fails to explain the increased response to phenylephrine.

Alternatively, the increased vasopressor response to phenylephrine during CPB, compared with the decreased response to other vasopressors such as epinephrine and ephedrine, may be attributed to the