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.

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(accepted for publication February 15, 1989)

Anesthesiology
70:1023–1024, 1989

Altered Response to Vasopressors before and during Cardiopulmonary Bypass

To the Editor—Massagee et al., as well as Schwinn et al., 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.

In contrast to phenylephrine, we have previously shown that ephedrine produces less vasopressor response during CPB + AXC compared with that achieved off-bypass. The injection of ephedrine 30 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 Unpublished data.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. Circulating catecholamines are known to increase during CPB. Also, hypothermia may increase blood viscosity and induce vasocostriction. Thus, both hypothermia and increased endogenous catecholamine release may explain the augmented vasopressor response to phenylephrine during hypothermic CPB. 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 ephedrine 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

* Baraka A: Response to vasopressors before and during cardiopulmonary bypass. Unpublished data.
different mechanisms by which these drugs increase the blood pressure. PH is a selective alpha₂-adrenergic agonist that increases the blood pressure by increasing the systemic vascular resistance (SVR). Prior to CPB, the vasopressor effect of phenylephrine is usually accompanied by an increased afterload and by reflex bradycardia, which can decrease the cardiac output (CO). Since the arterial blood pressure is the product of CO x SVR, the decreased CO can partially counteract the effect of phenylephrine on the SVR. On the other hand, during CPB + AXC, the heart is excluded from the circulation and the pump flow can be maintained at a constant level, and hence the action of phenylephrine on SVR is not counteracted. In contrast to phenylephrine, which is a selective alpha₁-adrenergic agonist, a vasopressor acting as mixed beta- and alpha-adrenergic agonist, such as epinephrine or ephedrine, can increase both the CO and SVR. The resultingpressor response is due to vasocostruction but mainly to cardiac stimulation, and hence a greater response may result during the bypass period than during bypass.

It may be concluded that vaspressors which increase the blood pressure by a predominant increase of SVR will be more effective during CPB, while those acting by a predominant increase of CO will be more effective during the bypass period.

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Anesthesiology
70:1024–1025, 1989

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(Accepted for publication February 21, 1989.)

Pulse Oximetry is Accurate in Patients with Dysrhythmias and a Pulse Deficit

To the Editor—Some authors have recommended* or used † the agreement of the pulse oximeter pulse rate with the EKG heart rate as a criterion for arterial oxygen saturation (SpO₂) reliability. Since the pulse rate of patients with arrhythmias may not match their heart rate, we wondered if pulse deficits truly predict SpO₂ reliability.

Following approval from the institutional review board, we studied 163 consecutive surgical Intensive Care Unit patients with pre-existing arterial catheters. We measured SpO₂ with an Ohmeda 3700, software version M (Ohmeda, Boulder, CO), and followed the manufacturer’s recommendations for ensuring a reliable SpO₂ (a stable SpO₂, a signal strength greater than 5 pixels, and at least three consecutive pulsatile waveforms). During SpO₂ and oximeter pulse rate recording, an arterial blood sample was withdrawn and immediately analyzed for %HbO₂ and carboxyhemoglobin (COHb) with an IL 282 Co-Oximeter (Instrumentation Labs, Lexington, MA). EKG heart rate and rhythm were obtained from a simultaneous 1-min EKG rhythm strip recording.

We defined normal sinus rhythm (NSR) as an EKG rhythm with regular p-waves, QRS complexes, and <5 ectopic beats per minute. We defined non-NSR as an irregularly irregular rhythm or >10 ectopic beats per minute. We defined the pulse deficit (PD) as the EKG heart rate less the oximeter pulse rate. The mean (SpO₂-%HbO₂) difference (bias) was calculated for the NSR, non-NSR, PD, and no PD groups. The different groups were compared by unpaired t test.

Of 163 patients, 24 had PD > 3 (nine were NSR and 15 were non-NSR) and 139 did not. In ten patients, the EKG rhythm changed during their ICU stay and they entered both NSR and non-NSR groups. Paired t test of these patients showed no difference between the two groups (P > 0.45; power > 0.30). Eight patients had 5–10 ectopic beats per minute and were excluded from EKG rhythm comparison, leaving 154 NSR patients and 31 non-NSR patients. Other statistics are summarized in table 1. Our bias results for all groups are similar to those previously reported with the Ohmeda 3700 in normal volunteers* and in hospital patients.† There was no relationship between the pulse deficit and (SpO₂-%HbO₂).

A PD > 3 does not necessarily mean the pulse oximeter is unreliable. True pulse deficits may exist because irregular or premature myocardial electrical depolarizations do not always produce a peripheral pulse. Since the PD is the EKG heart rate less the oximeter pulse rate, different oximeter pulse rate and EKG heart rate algorithm machines may cause artificial pulse deficits. SpO₂ is accurate in non-NSR patients and patients with pulse deficits as in NSR patients when SpO₂ is stable, the signal strength is greater than 5 pixels, and at least three consecutive pulsatile plethysmograms are noted. When evaluating SpO₂ reliability, a noise-free pulsatile plethysmogram is more important than the absence of a PD.