Correspondence

Dear Editor:

I would like to congratulate the authors of "Cardiopulmonary Bypass and the Pregnant Patient: A Review" (J Extra-Corpor Technol. 1993; 25(2): 61-66) on an excellent article. I would, however, like to comment on the use of epinepherine as a "vasopressor of choice"during cardiopulmonary bypass.

Although frequently used in the clinical setting as a vasopressor, epinepherine relies on its strong beta and alpha agonist properties to create a pressor response. On bypass, however, the therapeutic benefits of beta receptor stimulation are lost and epinepherine may actually produce a paradoxical hypotension.

Epinepherine uses three primary mechanisms for blood pressure elevation. These are: 1) increasing the force of ventricular contraction, 2) increasing heart rate, and 3) vasoconstriction of precapillary sphincters. The first two effects of inotropy and chronotropy are done through the beta-1 stimulation in the heart. The third effect, constriction of the precapillary sphincters, is accomplished through alpha adrenergic stimulation. Since the heart contributes little to the cardiac output on bypass, any vasopressor response to epinepherine is probably due to alpha receptor stimulation.

Interestingly, should no alpha stimulation occur, a pure beta-2 response during total cardiopulmonary bypass will likely produce a drop in blood pressure rather than the expected pressor response. This may be explained by the decrease in peripheral vascular resistance caused by beta-2 dilation of the skeletal muscle vascular beds.

As we are aware from the review, alpha adrenergic stimulation in the pregnant patient poses significant risk to the fetus. Unfortunately, any pressor response to an exogenous catecholamine administration on cardiopulmonary bypass is probably due to alpha adrenergic stimulation.

Recognizing that there are no safe vasopressors, alternative means to control blood pressure such as maintaining a normal potassium, a normal to high normal calcium, and higher than normal flow rates should be tried before alpha agonists, regardless of their form, are employed.

Bibliography

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IN RESPONSE:

Thank you for the opportunity to address Mr. Morin's concerns regarding the use of epinepherine in the pregnant patient during cardiopulmonary bypass. Epinepherine has been recommended because of its rapid onset, brief duration, and few unwanted side effects. While a separate text could be written on the use of any pharmacological agent and its extrapolated positive and/or negative potential in this clinical situation, we should like to elaborate on some key points of consideration regarding the use of epinepherine that were not addressed in our article for the sake of conciseness. Mr Morin's observation is correct in that the maternal heart isolated during the aortic cross-clamping period will lose the beta-1 inotropic and chronotropic effects provided by epinepherine. However, we would like to emphasize that operating on a pregnant patient is a rare clinical phenomenon in which two simultaneous patients, both mother and fetus, may be compromised. Therefore, the following points must be made:

1. The inotropic and chronotropic beta-1 effects on the fetus are not lost and may prove quite beneficial.
2. We would like to reiterate that adjustment to blood flow is the primary response to counteract hypotension. This concurs with our article in which we recommended that the already augmented CI of 2.6 - 3.0 L/m^2/min be used in response to fetal heart rate.
3. We agree that vasopressors be avoided if at all possible and that any pure alpha adrenergic agents that would strongly decrease utero-placental blood flow also be avoided.
4. In normothermic conditions and under routine circumstances, vasopressors will be needed. Our experience and that of other authors regarding the gravid patient is that vasopressors are usually required.
5. Our identification of epinepherine as the vasopressor of choice falls far short of a recommendation and as we state in the article, any pharmacological agent should be used "only when the benefits outweigh the risk to the fetus."

In addition to the aforementioned preservation of the favorable fetal beta-1 inotropic and chronotropic responses to epinepherine, which freely traverses the placenta, we would like to elaborate further regarding the preferential selection of epinepherine:

1. Epinepherine inhibits spontaneous uterine contractions initiated by maternal influences or induced by oxytocin release from the stressed fetal hypothalamus.

2. Epinepherine increases maternal glycogenolysis and restores fetal glucagon stores, lessening the potential for critical glucose depletion to the fetus and avoiding the inherent deleterious effects of anaerobic metabolism during transient hypoxemia.
3. Epinepherine is not a pure beta adrenergic agent but elicits a combined beta and alpha effect with alpha receptors of the skin and subcutaneous blood vessels extremely sensitive to circulating epinepherine. Stimulation of these venous plexi forces substantial quantities of blood (estimated to be as much as 10% of the blood volume) into the core vasculature. The benefit of a volume gain of 10% and an increase in blood flow is apparent.
4. Should clinicians be concerned about a paradoxical hypotension, a nonspecific beta adrenergic blocking agent could be utilized whereby the beneficial alpha cutaneous effect to epinepherine could be gained without stimulation of the beta-2 receptors. Propananol administered generally at .05 mg/kg offers non-specific beta blocking in addition to an antiarrhythmic effect.

We would like to elaborate with apprehension regarding any discussion or intended administration of potassium to adjust maternal potassium levels. Specifically, with Mr. Morin's discussion maintaining potassium as an "alternative means to control blood pressure." Any potassium infusion as an acute response to hypotension in an effort to restore normal potassium values cannot be realistically considered in that rapid influx threatens to evoke fetal asystole before the maternal systemic levels would be affected. Potassium therapy must be undertaken with extreme caution and deficits restored over a prolonged period of time.

We would like to thank Mr. Morin for his interest in our article and for the opportunity to respond to this letter.

References

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