skin, in contrast to those with valvular heart disease, in whom flow increased only slightly. MAP's and HR's increased in both groups. Increased myocardial work and oxygen requirements following painful stimulation may be detrimental in a heart which cannot increase its oxygen delivery because of valvular or coronary-artery disease.

We conclude that morphine produced transient peripheral vasodilation and cardiovascular stimulation during its infusion. These changes were more prominent in patients with valvular heart disease, as was the degree of hypoventilation. Nitrous oxide added to morphine produced similar degrees of cardiovascular depression in the two groups. That observed changes were, in fact, the result of morphine and/or nitrous oxide was suggested by the return of most hemodynamic measurements to control levels with time or following substitution of nitrogen for nitrous oxide.

The authors acknowledge the cooperation of the following in performing this study: H. King, M.D., R. King, M.D., H. R. Shumacker, M.D., V. K. Stoelting, M.D., R. Kraus, P. Brumbaugh, S. Whitmore, L. McConnell, J. Craig, D. Dunn, J. Bailey, and J. Spreen.

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Obstetrics

PARACERVICAL BLOCK AND FETAL BRADYCARDIA Fetal bradycardia followed paracervical block anesthesia with mepivacaine (200 mg) in 24 of 92 patients who received 100 paracervical blocks. Gestational age, parity, fetal weight, and maternal complications did not influence the incidence of fetal bradycardia following paracervical block. There was a significant decrease in fetal *p*H and an increase in base deficit only when bradycardia lasted more than 10 minutes. Recovery from the transient metabolic acidosis was complete in all. Transient increases in uterine activity, as measured by the area under the uterine pressure curve, were found in the majority of patients with post-paracervical-block fetal bradycardia. Most patients who had no fetal bradycardia had decreases in uterine activity. (*Roger, K. F., and others: Fetal Cardiac Response to Paracervical Block Anesthesia, Am. J. Obstet. Gynecol. 113: 583–591, 1972.*)