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TITLE: Low O_2 Cost if Preload Increase Triples Cardiac Work

AUTHORS: Michael Nugent, M.D., Joseph T. Walls, M.D., John H. Tinker, M.D., Carlos E. Harrison, M.D.

AFFILIATION: Departments of Anesthesiology, Surgery, and Cardiology, Mayo Clinic, Rochester, Minnesota 55901
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There is controversy over whether severely depressed arterial pressure during anesthesia should be returned toward normal by increasing inotropic state and/or heart rate with a catecholamine or by increasing preload with fluid infusion. Except in special circumstances, few would utilize afterload increases alone. We investigated the increases in myocardial oxygen consumption ($M\dot{V}O_2$) "costs" of incremental increases in left ventricular output and stroke work index, at constant heart rates and mean arterial pressures, using a method which solely increased preload. Using four different anesthetic techniques, these studies were done before and after one hour of global myocardial ischemia (aortic crossclamp). Our purposes were: 1) to quantitate the increase in $M\dot{V}O_2$ associated with tripling external cardiac work by preload increases alone, 2) to examine the possibility that anesthetics might differentially affect this relationship, 3) to study the same relationships after global ischemia (simulating the clinical situation).

Methods

We used a right heart bypass preparation in dogs. Vena caval flow was routed via heat exchanger and bubble oxygenator to the left atrium. Coronary blood flow (CBF) was measured by timed collection from right atrium and ventricle. $M\dot{V}O_2$ was determined as the product of CBF and arterial-coronary venous oxygen content difference. Four anesthetics were studied, halothane 1 MAC (0.86 ± .01%, n=7), halothane ½ MAC (0.42 ± .01%, n=6), morphine 3 mg/kg (n=5) and pentobarbital 40 mg/kg (n=6). Standard ventricular function curves were produced by recording left ventricular end-diastolic pressures (LVEDP) at cardiac indices (pump flows) of 0.8, 1.2, 1.6, 2.0, and 2.4 l/min/m^2. At each flow increment, after allowing at least 2 minutes, $M\dot{V}O_2$ was determined. Heart rates were paced to 156 ± 2 beats per minute after crushing the SA nodal tissue. Afterload was held constant by utilizing either an aortic snare, or an artery-to-venous reservoir shunt. Mean arterial pressure was controlled at 64 ± 1 mmHg and left ventricular systolic pressure at 109 ± 2 mmHg at each incremental increase in cardiac index. The animal was cooled to 28°C, the aorta crossclamped, aortic root perfused with 200 ml normal saline at 28°C, and additional 50 ml increments were given q 15 minutes. The clamp was removed after 1 hour, the animal rewarmed, and the above ventricular function curves repeated with $M\dot{V}O_2$ determinations.

Results

During a tripling of cardiac index, and therefore a tripling of LV stroke work index because rate and afterload were held constant, myocardial oxygen consumption increased only 1.15 ± 0.40 ml O_2/100 gm/min (23%) ($p < .001$), taking all four anesthetics together (n=19) (Figure 1). Individually, $M\dot{V}O_2$ increased 0.60 ± 0.62 ml/O_2/100 gm/m with ½ MAC halothane, 1.56 ± 0.21 ml/100 gr/min ($p=.001$) with 1 MAC halothane, 1.13 ± 1.32 ml/O_2/100 gm/min with morphine, and 1.62 ± .85 ml/100 gm/min ($p=.05$) with pentobarbital. The differences between anesthetic groups are not signficant at any increment of cardiac index. A left ventricular end diastolic pressure increase from 5 ± 1 mmHg to 8 ± 1 mmHg was associated with the tripling of cardiac index. After 1 hour aortic crossclamp, $M\dot{V}O_2$'s were not lower at each cardiac index despite decreased ventricular function. No major differences between anesthetics regarding oxygen cost to increase CI by increasing preload were seen either pre- or post-ischemia.

Discussion

Most clinical anesthesiologists would probably prefer to treat hypotension with additional preload than with catecholamines. This study verifies and quantifies that clinical impression. To illustrate the low O_2 cost of increasing CI by increasing preload, an increase in heart rate of only 14 beats/min coupled with an increase in aortic systolic pressure of 14 mmHg in this preparation resulted in the same increase in $M\dot{V}O_2$ (23%) as was required to triple the cardiac index by increasing preload (unpublished data). Theye reported in 1967 that halothane-induced decreases in $M\dot{V}O_2$ were well correlated to decreases in external cardiac work. If changes in external cardiac work are the result of changes in inotropic state, afterload, or heart rate, $M\dot{V}O_2$ is markedly affected. If changes in cardiac work are achieved via alterations in preload, as in the present study, changes in $M\dot{V}O_2$ are small and there is little correlation between $M\dot{V}O_2$ and external cardiac work. This study also shows that the oxygen cost of preload increases remains small with various anesthetic techniques and even after aortic crossclamping.

The clinical implications are two-fold: 1) alterations in cardiac index are more economically achieved, in terms of oxygen cost, by altering preload, rather than heart rate or inotropic state; 2) following global ischemic injury, e.g. following cardiopulmonary bypass with aortic crossclamping, the same holds true, implying that optimal preload should be achieved before resorting to catecholamines.

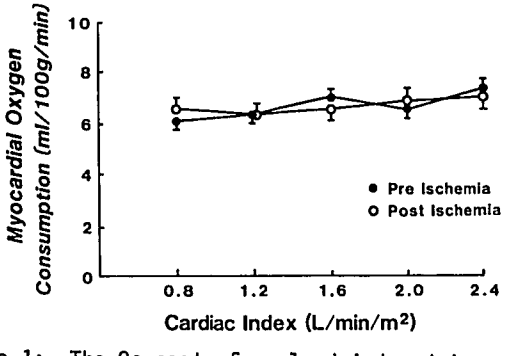

Figure 1: The O_2 cost of preload induced increases in cardiac index.