Title : ACTIVE STIFFNESS OF LEFT VENTRICLE DURING ANESTHESIA

Authors : S. Shimosato, M.D. and O. Kemmotsu, M.D.

Affiliation: Department of Anesthesia
Iowa University Hospitals and Clinics, Iowa City, IA. 52242
Department of Anesthesiology, Kitazato University, School of Medicine, Kanagawa, Japan

Introduction: In the intact heart, the internal shortening of the contractile element may be influenced by passive factors other than the series elasticity of cardiac muscle. The overall stiffness of the contracting ventricle (active stiffness) may not be the same as the series elasticity of isolated heart muscle.[1] Hamilton, et al, reported that cyclopropane anesthesia causes increases in ventricular endiastolic pressure with relatively unchanged endiastolic volume in dogs.[2] These investigators advocated that cylcopropane causes a decrease in ventricular distensibility. In contrast they found that halothane anesthesia produced no significant changes in left ventricular endiastolic pressure with a slight increase in endiastolic volume. However, the effects of these anesthetic agents on the active stiffness of the contracting left ventricle has not been studied. The present study was undertaken to evaluate influences of cyclopropane and halothane on the overall active stiffness of the intact left ventricle

Methods. Fourteen mongrel dogs were anesthetized with i.v., a chloralose-urethane solution. The trachea of each dog was intubated with a cuffed endotracheal tube following i.v. gallamine (40 mg). Controlled respiration was maintained throughout the experiment. A sternum-splitting thoracotomy was performed and the root of the aorta exposed for placement of a blood flow meter probe. Volume decrement (dV) for each 5-msec interval of systole was derived by integration of the flow rate curve. The following calculations were also made at 5-msec intervals: Volume (V) = endiastolic volume - dV, in ml; radius (r) = $\sqrt[3]{.239V}$, in cm; circumferential fiber length (c) = $2\pi r$, in cm, fiber shortening rate (FSR) = flow rate/$2r^2$, in cm/sec; force (F) = $\pi r^2 P$, in dynes; dF/dt = πr^2(dp/dt) - rP (FSR), in dynes/sec. The active stiffness was calculated using the method previously described.[1] Two adjacent cardiac systoles were examined; one an auxotonic control beat and the other an isovolumic contraction of the left ventricle at the same endiastolic volume. The force-time curves of the two beats were plotted and superimposed. At the point of peak force of the auxotonic systole (F) the instantaneous fiber shortening rate is measured. As the series elastic velocity is zero at this moment, FSR is equal to contractile element velocity (Vce). The rate of force development (dF/dt) of the isolumic beat is then measured at the same level of wall force. The dF/dl is derived as follows: dF/dl = (dF/dt)/FRS. The modulus of active stiffness is calculated by dividing dF/dl by F and is then normalized for unit fiber length by multiplying by endiastolic fiber length. The normalized modulus of functional active stiffness (Sn) is expressed as cm^{-1} per cm of muscle length. After control measurements of Sn, cyclopropane was administered in five dogs and halothane in nine dogs. Measurements were repeated at the steady level of anesthesia.

Results. The results are summarized in table 1. The normalized modulus of active stiffness (Sn) averaged 32.7 ± 18.5 (±S.D.) cm^{-1} per cm of muscle length during control state in 5 dogs. During cyclopropane anesthesia (average blood concentration: 20.0 ± 7.7 mg%) Sn decreased on average of 11.9 ± 34.9 (± S.D.) % (P>0.5). Halothane anesthesia (average blood concentration: 18.3 ± 3.4 mg%) reduced the functional active stiffness of left ventrical significantly (P<0.01). Average change in Sn during halothane anesthesia in nine dogs was -23.0 ± 3.0 (±S.D.)per cent.

Table I, Functional Active Stiffness

	No. of Determinations	Sn (cm^{-1}/cm)	
Control	11	32.7	11.8*
Cyclopropane	18	28.3	15.2
Control	18	33.0	14.3
Halothane	54	25.4	9.8

*Standard deviation

Discussion. Findings of the present study indicate that the functional active stiffness of the left ventricle was not significantly altered by cyclopropane anesthesia. During halothane anesthesia, on the other hand, the functional active stiffness of the entire left ventricle decreased significantly. Decreased force development and velocity of shortening occur during halothane anesthesia may be partly related to the reduced active stiffness of left ventricle.

References.

1. Forwand S F, McIntyre K M, Lipana J G et al: Active stiffness of the intact canine left ventricle: with observations on the effect of acute and chronic myocardial infarction, Circ Res 19: 970, 1966.
2. Hamilton W K, Larson C P, Bristow J D et al: Effect of cyclopropane and halothane on ventricular mechanics; A change in ventricular diastolic pressure-volume relationships, J Pharm & Expt. Therap. 154: 566, 1966.