

The closing behaviour of the natural aortic valve

Citation for published version (APA):

Steenhoven, van, A. A., Schaar, van der, P. J., Veenstra, P. C., & Reneman, R. S. (1978). The closing behaviour of the natural aortic valve. In J. Baan, & A. Noordergraaf (Eds.), *Cardiovascular system dynamics [international conference on cardiovascular system dynamics, Valley Forge, Pennsylvania, 6-10 April 1975]* MIT Press.

Document status and date:

Published: 01/01/1978

Document Version:

Publisher's PDF, also known as Version of Record (includes final page, issue and volume numbers)

Please check the document version of this publication:

- A submitted manuscript is the version of the article upon submission and before peer-review. There can be important differences between the submitted version and the official published version of record. People interested in the research are advised to contact the author for the final version of the publication, or visit the DOI to the publisher's website.
- The final author version and the galley proof are versions of the publication after peer review.
- The final published version features the final layout of the paper including the volume, issue and page numbers.

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QUANTITATING THE CONTRACTILE STATE OF THE LEFT VENTRICLE IN INTACT MAN BY APPLICATION OF A NEW MATHEMATICAL MODEL. George T. Daughters and Neil B. Ingels, Jr., Palo Alto Medical Research Foundation, Palo Alto, CA. 94301.

Attempts to measure changes in the contractile state of the left ventricle in intact man often meet with the difficulty that indices of contractility such as velocity of fiber shortening (Vcf), rate of left ventricular (LV) pressure rise (dP/dt) etc. are strongly affected by preload and afterload as well as the contractile state. For example, in recent studies of the effect of heart rate on contractility in 7 patients after coronary artery surgery we measured LV volumes, Vcf (using computer assisted analysis of motion of surgically inserted radiopaque markers) and aortic pressure. Heart rate was controlled by atrial pacing at rates of 76 and 135 beats per minute. A 23% decrease in LV end diastolic volume (EDV) accompanied increasing heart rate and compensated for the inotropic effect of the rate increase, with the result that measured Vcf was unchanged. Using a recently developed mathematical model of LV dynamics of the form

$$Vcf = (aEDV+b) P + Vmax,$$

we were able to demonstrate a 16% increase in Vmax. This model allows quantitation of the effects of preload and afterload on the dynamics of the intact human heart and can be used in studies of the effects of pharmacological and physiological interventions on the contractile state of the heart. (This work was supported by NIH Grant HL17993.)

THE CLOSING BEHAVIOUR OF THE NATURAL AORTIC VALVE. A.A. van Steenhoven*, P.J. van der Schaar*, P.C. Veenstra*, R.S. Reneman. Eindhoven University of Technology and University of Limburg, Maastricht, The Netherlands.

For an effective design of artificial leaflet-valves, a proper understanding of the closing behaviour of the aortic valve is required. In open-chest dogs direct cinematographic high-speed recordings of the aortic valve movement were made, using a thin (4 mm) flexible fiberoptic. Simultaneously ECG, ascending aortic flow (electromagnetically) and the pressures in the aorta (A), left ventricle and left atrium (LA) were recorded. Replacement of blood by a transparent liquid (Tyrode solution), was done with two roller-pumps, the one connected to the LA and the other to the femoral artery. The latter is necessary for maintaining peripheral arterial blood pressure at physiological levels. Free outflow occurred through a canula in the pulmonary artery. Comparison of the film frames with the aortic flow signals reveals that:

- i aortic valve closure starts during the deceleration phase of systolic aortic flow and at least 60-80% of the closure is completed before aortic flow becomes zero;
- ii the experimental results are in agreement with the results of a theoretical model of closure, based on the presence of a wake behind the moving cusps as observed in model-studies.

CENTRAL PULMONARY ARTERIAL HEMODYNAMICS IN PATIENTS WITH PULMONARY VALVAR STENOSIS: ROLE OF BLOOD VELOCITY AND ANATOMY. A. van Grondelle*, A.J. Muster* and M.H. Paul* (SPON: H.K. Chang). Child. Mem. Hosp., Chicago, IL. 60614.

Markedly different pressures in the right (RPA) and left (LPA) pulmonary artery were observed at catheterization in patients with pulmonary valvar stenosis in the absence of any branch stenosis. A large part of the right ventricular pressure is converted into kinetic energy resulting in a 'valley' in the systolic part of the main pulmonary artery (MPA) pressure. The jet in the MPA bypasses the RPA, whose orifice is closer to the pulmonary valve than that of the LPA, and breaks up at the origin of the LPA. The RPA pressure is consequently similar to the MPA pressure and shows the same 'valley' during systole. The peak and mean LPA pressure, however, are higher than those in the MPA, since part of the kinetic energy is reconverted to pressure, and the pressure pulse contour becomes normal. Similar pressure differences in contralateral branches have been observed in patients with transposition of the great arteries; in this case, however, the pressure in the RPA increases due to the different anatomy. Our findings indicate the importance of the blood velocity and central pulmonary arterial anatomy in the genesis of the difference in RPA and LPA pressure and in the reported unequal vascularity of and blood flow distribution between the lungs in patients with these lesions.

(Supported in part by Research Fellowship F75-4 from the Chicago Heart Association.)

SIMULATION OF RIGHT VENTRICULAR FILLING PROCESS BY RIGHT ATRIAL AND VENTRICULAR MODELS AS TIME-VARYING ELASTANCE. Veng-Kin Lau* and Kiichi Sagawa. The Johns Hopkins Univ., Baltimore, Md. 21205

To gain a quantitative insight into the contribution of right atrial contraction to right ventricular filling, we simulated blood flow through the systemic vein - right heart - pulmonary artery section. The major assumption is that the concept of a time-varying elastance, reduced from experimental data on isovolumic atrial contraction, also represents ejecting contraction of the atrium. With a set of system parameter values appropriately chosen for the control condition, pressure and flow waveforms in various portions of the model agreed with the experimental curves. The effects of changing the time interval between atrial and ventricular systole (A-V interval), atrial contractility, atrial compliance, heart rate, and blood inertance were then studied. There was an optimal A-V interval of about 0.1 sec for stroke volume. The effect of altering atrial compliance on stroke volume was heart rate dependent. Increase in atrial contractility, decrease in heart rate, and increase in blood inertance also increased stroke volume. Contribution of atrial contraction to ventricular filling was more significant during exercise than at rest. These parameter sensitivity tests indicated good agreement with the known experimental results. [Supported in part by PHS NHLBI Grant 14529 and AHA-Maryland Fellowship].

MYOCARDIAL ENERGETICS: AN ANALYSIS USING IRREVERSIBLE THERMODYNAMICS. Chandler A. Phillips, Edward S. Grood*, and William J. Scott*. Wright State Univ., Dayton, OH 45431

A model has been developed which relates the power generated by the chemical reactions of the ventricular myocardium to the mechanical work performed. In order to accomplish this, the cross-bridges of heart muscle are modeled as linear energy converters, and analyzed using irreversible thermodynamics. The final equation relates the chemical power (CP) consumed to the muscle force (P) and shortening velocity (V) of the muscle:

$$CP = \frac{k(P \cdot V) [V_R/V + K]}{K [1 - V/V_R]}$$

The mechanical power (MP) is equal to P·V and hence this equation relates CP to MP through the efficiency of the conversion process (η). Three model constants are required and the values are estimated from available experimental data: $k = 1.00$, $K = 15.00$, and $V_R =$ maximum (unloaded) shortening velocity. Previous investigators have considered myocardial energetics only during the isovolumic phase of systole. The analysis presented in this study is general in that it relates chemical power consumed to mechanical performance throughout the entire cardiac cycle. (Supported in part by grant from the AHA, Miami Valley Heart Chapter).

THE INFLUENCE OF TRANSMURAL PRESSURE ON MECHANICAL PARAMETERS OF THE TWO-COMPARTMENT MODEL OF SYSTEMIC CIRCULATION. Alan Jackman and Jerry F. Green. University of California, Davis California 95616

A right-heart bypass preparation in 7 dogs separated the venous return into splanchnic (Q_s) and non-splanchnic (termed peripheral, Q_p) flows. Flows from the two channels drained by gravity into an external reservoir. Reservoir blood was returned by a pump to the pulmonary artery. Venous resistances (R_v and R_{vp}), arterial resistances (R_a and R_{ap}), compliances (C_v and C_{vp}) and mean systemic pressures (P_{ms} and P_{msp}) for the splanchnic and peripheral channels were calculated from the steady-state and transient volume shifts which occurred following rapid drops in venous pressure. Parameters were determined at cardiac outputs of 48, 64, 80, 96 and 112 l/min. Venous resistances and compliances were regressed against mean systemic pressure for the associated channel; arterial resistances against aortic pressure. Over the range of mean systemic channel pressures and aortic pressures produced by the cardiac output variations, R_{vp} , C_v , C_{vp} , and P_{ms} showed no statistically significant dependence on intravascular pressures. However, R_a was found to have significant dependence on P_a , the resistance increasing with mean systemic pressure. While not statistically significant, C_a , R_{ap} , and R_{asp} all display a consistent tendency to decrease with increasing intravascular pressure. (Supported by USPHS NIH Grant HL 20371)