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The First Derivative of the Transthoracic Electrical Impedance as an Index of Changes in Myocardial Contractility in the Intact Anaesthetised Dog

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Abstract. The suitability of the peak value of the first derivative of the thoracic electrical impedance dZ/dt_{max} has been investigated in dogs as a non-invasive index of changes in myocardial contracility by comparing if with the peak left ventricular dP/dt and the peak values of aortic blood velocity and acceleration. An increase in the inspired halothane concentration was used to produce changes in contracility. In 5 dogs the combined values for the correlation coefficient between dZ/dt_{max} peak velocity and acceleration were respectively 0.937, 0.954 and 0.950. In 14 out 15 comparisons $p < 0.001$ and in one, $p < 0.01$. On these grounds, dZ/dt_{max} is proposed as a variable worthy of consideration in patient monitoring.

Key words: Myocardiac contractility changes, Thoracic electrical impedance.

Introduction

It has been stated that the peak first derivative of the thoracic electrical impedance waveform may be used as an estimate of myocardial contractility [10, 23]. To date there is no accepted method for the non-invasive monitoring of changes in cardiac contracility and hence we planned to study the peak derivative of the thoracic electrical impedance in this respect.

Indices of contractility represent either the muscle function or the pump function of the heart. Since it had been found earlier [7] that dZ/dt was an index of heart pump function, it was compared with the maximum acceleration of blood in the aorta [18] and also with the peak aortic blood flow [10]. From the waveforms of dZ/dt, acceleration, ascending aortic flow and aortic pressure, other possible indices of contractility were calculated, namely the left ventricular ejection time, the pre-ejection period and the Heather Index [6]. Peak dZ/dt was also compared with the peak dP/dt [19] for the left ventricular pressure.

Methods

Five unselected mongrel dogs of either sex and weighing 21.3. - 28.7 kg were premedicated with Acepromazine (0.2 - 0.4 mg per kg) intramuscularly. They were anaesthetised 30 to 45 minutes later with thiopentone sodium (25 mg per ml, 0,5 ml per kg) intravenously. The trachea was intubated with a 12 mm cuffed endotracheal tube and anaesthesia maintained for control purposes with 0.5 per cent halothane v/v in nitrous oxide/oxygen (60/40). Intermittent positive pressure ventilation was provided by a Manley ventilator (Hutchinson Blease Ltd) with a non-rebreathing system at an inflation pressure of 20 - 30 cm of water. The minute volume of ventilation was adjusted in each case to maintain the arterial carbon dioxide tension within physiological limits. The body temperature was maintained at 37° C $\pm 1^{\circ}$ C.

Following the induction of anaesthesia, a fluid-filled nylon cannula (5F, Portex Ltd) was passed retrogradely via the right femoral artery into the ascending aorta and connected to an external pressure transducer (Bell and Howell Ltd, Type 4-327-L221). A similar cannula was inserted into the inferior vena cava via the right femoral vein for drug injection, together with a thermistor temperature probe (Light Laboratories Ltd, 5 F) for monitoring body temperature. The left ventricular pressure was measured using a catheter-tip micro-pressure transducer (Millar In-

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struments Inc., Model PC-350) inserted retrogradely into the left ventricle via the left carotid artery. The sternum was split from the neck to approximately the ninth intercostal space and the root of the ascending aorta dissected free. A pre-calibrated cuffed flow sensor (Carolina Medical Electronics Inc) was placed around the aortic root and connected to a Carolina square wave electromagnetic flowmeter (Model 501). Outputs from the flowmeter provided pulsatile and mean aortic flow signals. Following placement of the flow sensor, the chest was closed and the overlying tissue sewn together with surgical sutures. Immediately before the final closure, the lungs were manually inflated to exclude as much air as possible from the pleural cavity.

Four disposable Mylar tape electrodes (3M Co, Type 6001) each having an aluminium strip along its mid-line were placed around the dog in order to measure the transthoracic impedance, the skin underlying the electrodes having first been closely shaved and smeared with Cambridge electrode jelly. Two electrodes were placed around the neck and two around the thorax, the innermost of the latter pair being situated at the level of the xiphisternal joint. The outer pair of electrodes was fed with a constant sinusoidal current of approximately 4ma.rms at 100 kHz from an impedance cardiograph (Instrumentation for Medicine Inc, Model 304A). The inner pair of electrodes was connected to the input of the impedance cardiograph. A 1 ml sample of blood was taken from each dog and the haematocrit measured using a Hawksley micro-centrifuge. The haematocrit was used to set the appropriate probe factor for the electromagnetic blood flow meter and also to obtain the specific resistance of the blood from a previously prepared graph relating dog blood haematocrit and specific resistance [3].

The impedance cardiograph provided (via its own low-noise differentiator) an output signal proportional to dZ/dt where Z is the thoracic electrical impedance in ohms.

Two other differentiators incorporating buffer amplifiers were used to derive the left ventricular dP/dt and the acceleration (dV/dt, where V is the aortic blood velocity) from the left ventricular pressure signal and the pulsatile aortic flow signal respectively. Both the peak dP/dt and the peak dV/dt were taken as indices of myocardial contractility against which to compare values of peak dZ/dt. Additionally, the left ventricular pressure signal was stored on a magnetic tape recorder (Racal Ltd) and later replayed into a PDP12 computer (Digital Electronics Corporation) via an analogue-to-digital converter, for analysis. The pressure was also differentiated during playback and the resulting dP/dt signal fed into a second input channel of the computer. The computer was used to plot curves of left ventricular pressure at 2 ms intervals against $(dP/dt)/(32P)$ where P is the instantaneous isovolumic ventricular pressure and 32 is the series elastic constant for cardiac muscle [24]. The resultant pressure-velocity curve was extrapolated to zero pressure to give V_{max} the maximal no-load velocity of the contractile element of cardiac muscle [13]. Additionally, the peak value of the curve or V_{pm} [15] was noted.

The following signals were displayed on an 8-channel heated stylus recorder (Devices Ltd); Lead II ECG; aortic pressure; left ventricular pressure; left ventricular dP/dt; phasic aortic flow; mean aortic flow; aortic acceleration and dZ/dt. The left ventricular pressure was also displayed on an ultra-violet recorder (S.E. Laboratories Ltd, Model 3006/Dl) in order to measure accurately the left ventricular end-diastolic pressure.

From these eight recorded variables, the following time intervals were calculated: by measuring from the preceding R-wave of the ECG; the time to the peak dP/dt; the time to the peak acceleration; the time to the peak flow; the time to the peak dZ/dt; and the pre-ejection period. Additionally, the left ventricular ejection time and the Heather Index were calculated, where the Heather Index [6] is defined as the ratio of $(dZ/dt)_{max}$/the time to the $(dZ/dt)_{max}$.

During the initial 20 minute control period, each animal received halothane at an inspired concentration of 0.5 per cent by volume. Control readings were taken at the end of this period. The inspired halothane concentration was then increased in 0.5 per cent steps at 20 minute intervals up to a maximum af 2.5 per cent. Measurements were made at 10 minute intervals. At the end of the 20 minute administration of 2.5 per cent halothane, the inspired concentration was reduced to 0.5 per cent and maintained at that level for a further 20 minutes when a set of 'recovery' readings was taken. Immediately following each set of readings, a 1 ml blood sample was withdrawn for the estimation of blood halothane levels by the gas chromatographic technique [1,11].

Results

Pre-load, After-load and Heart Rate

Figure 1 shows the mean values found throughout the experiments expressed as percentages of the control values for the left ventricular end-diastolic pressure (pre-load), the aortic diastolic pressure (after-load) and the heart rate.

Consequent upon the administration of substantial amounts of halothane, there were slight, variable, decreases in left ventricular end-diastolic pressure. From a mean control value of 8.1 mm Hg, the maximum decreases occurred after 10 minutes administration of 1 per cent halothane (6.5 mm Hg) and 20 minutes of 2 per cent halothane (6.4 mm Hg). Neither of these values was significantly different from the control value ($p > 0.05$).

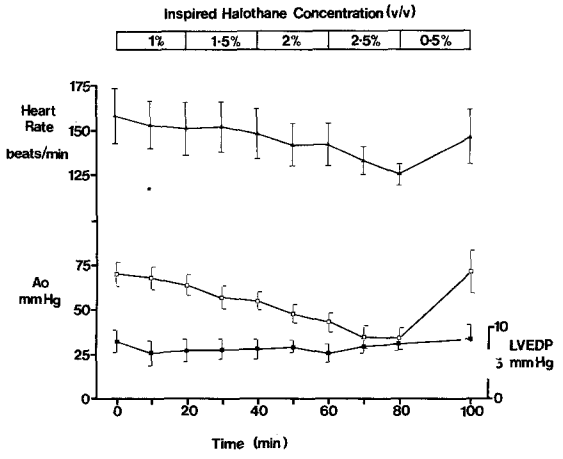

Fig. 1. Values for the left ventricular enddiastolic pressure (LVEDP), aortic diastolic pressure (Ao) and heart rate during ventilation with increasing inspired concentrations of halothane. After 80 minutes, the 20 minute recovery period was begun on 0.5 % halothane. Each value is the mean of five dogs plus or minus the Standard Error of the Mean

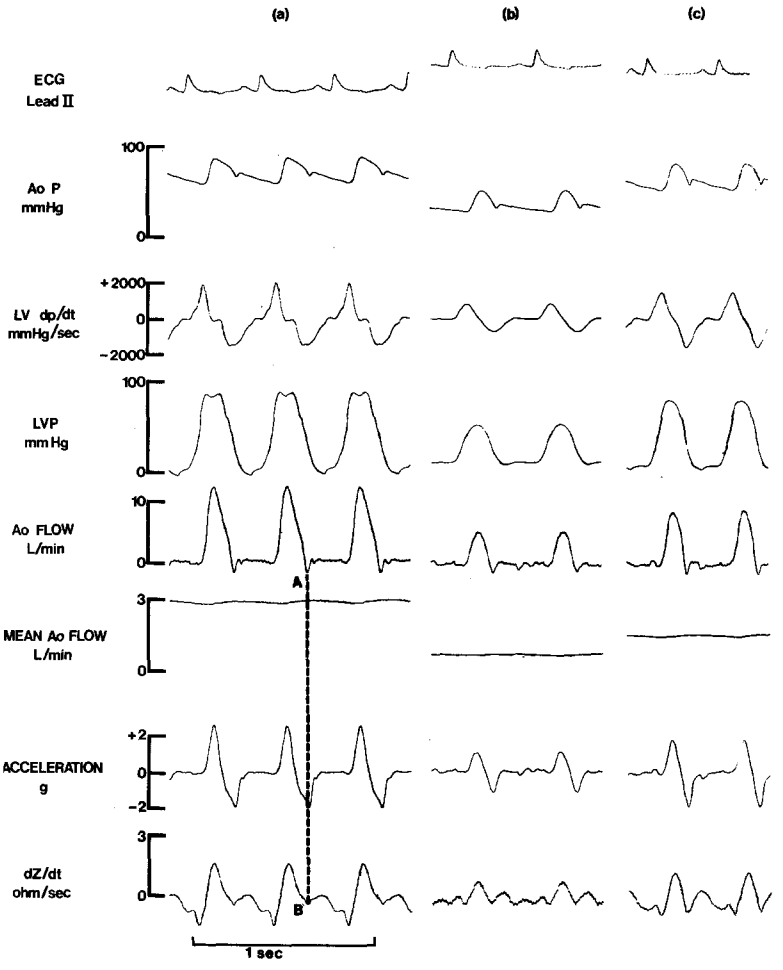

Fig. 2. A typical recording taken from dog No. 4 at a fast paper speed (100 mm/s) during the initial control period on 0.5 % halothane (Panel A), after 20 minutes on 2.5 % halothane (Panel B) and after a 20 minute recovery period on 0.5 % halothane (Panel C). The line AB shows the phasic relationships, of the pulsatile aortic blood flow with the peak dZ/dt waveform. Point A represents the short negative aortic flow peak and Point B represents the x-notch of the dZ/dt waveform

The aortic diastolic pressure (after load) steadily decreased from a mean control value of 70 mm Hg to a minimum value of 34 mm Hg after 80 minutes. From 50 to 80 minutes the values were statistically significantly different from the control (at pressures of 48, 43, 35 and 34 mm Hg the respective p-values were 0.05, 0.02, 0.01 and 0.01).

None of the values for the mean heart rate were significantly different from the initial control value, although the trend was for the heart rate to decrease slightly as the inspired halothane concentration was increased.

The recorded waveforms from a typical experiment (Dog No. 4) are shown in Figure 2. The line AB has been drawn to show the phasic relationship between the pulsatile aortic flow and the dZ/dt waveform. The negative peak flow, which occurs immediately after the closure of the aortic valve occurs at the same time as the x-notch in the dZ/dt waveform.

Pressure Contractility Indices

Figure 3 includes the mean values for the peak dP/dt expressed as a percentage of the control value found during

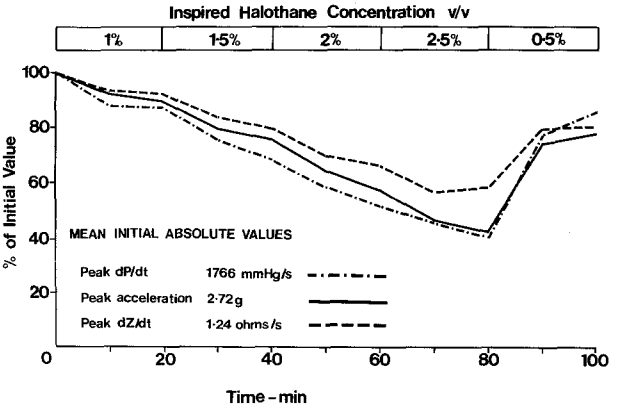

Fig. 3. This graph shows the trends of the peak values of dP/dt (–.–.–), acceleration (——) and dZ/dt (– – – –). Each value is the mean of 5 readings and is expressed as a percentage of the initial control value (100 %). After 80 minutes, recovery was commenced by switching the vaporiser to 0.5 % halothane. Mean initial absolute values are also indicated. g = gravitational constant

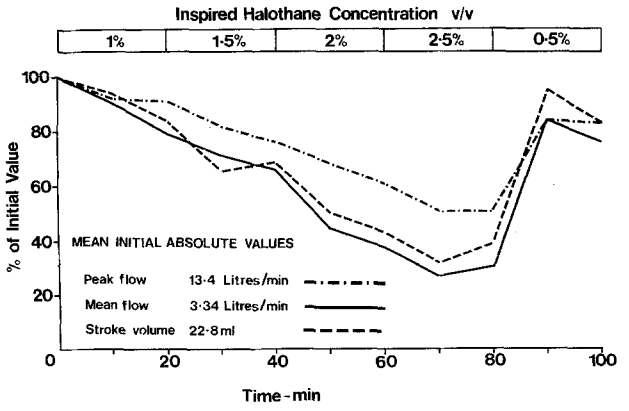

Fig. 4. This graph shows the trends of the peak pulsatile aortic blood flow (–.–.–), the mean aortic flow (———) and the stroke volume (– – – –). Each value is the mean of 5 readings and is expressed as a percentage of the initial control value (100 %). After 80 minutes, recovery was commenced by switching the vaporiser to 0.5 % halothane. Mean initial absolute values are also indicated.

the increasing concentrations of the inspired halothane. The initial absolute value was 1766 mm Hg/sec. There was a gradual diminution to a minimum of 41 per cent of the initial value after 20 minutes on 2.5 per cent inspired halothane, i.e. 80 minutes from the start of the experiment. Following a recovery period of 20 minutes on 0.5 per cent inspired halothane, the mean value of peak dP/dt had attained 86 per cent of its initial value.

Values for V_{max} and V_{pm} have not been included in the results since we found, that many of the pressure-velocity curves tended to be asymptotic to the Y-axis [25]. This often made extrapolation impossible. For similar reasons values for peak (dP/dt)/(kP) could not be estimated with any confidence.

Ejection Contractility Indices

The mean initial absolute values for the peak acceleration and peak dZ/dt were 2.72 g (where g = gravity constant) and 1.24 ohms/sec respectively. The mean values for these two variables expressed as a percentage of the initial value are also shown in Figure 3. The acceleration followed a similar trend to peak dP/dt, falling to a minimum value of 43 per cent of the initial value at 80 minutes. After changing to 0.5 per cent inspired halothane for 20 minutes the final recovery value had attained 78 per cent of the initial.

Although there was a decrease in peak dZ/dt with increasing concentrations of inspired halothane, the fall was not as marked as for peak dP/dt and peak acceleration. The minimum value attained was 57 per cent of the initial value and the final recovery value was 81 per cent of the initial value.

The trends for three other indices of ejection function, namely the peak pulsatile aortic flow, the mean aortic flow and the stroke volume are indicated in Figure 4. The curve for peak flow is similar to that for peak dZ/dt (Fig. 3). From a mean initial value of 13.4 litres per minute there was a steady decrease to reach a minimum at the end of 20 minutes on 2.5 per cent halothane of 51 per cent of the initial value. The final recovery value was 84 per cent of the initial value. In contrast, the mean aortic flow and the stroke volume fell by greater amounts from the initial values of 3.34 litres per minute and 22.8 ml respectively to their minimum values of 27 and 32 per cent respectively of the initial values. At the end of the recovery period the mean flow had attained 84 per cent and the stroke volume 95 per cent of their initial values.

Correlation Coefficients

Table 1 gives the correlation coefficients for the values of dZ/dt_{max}, maximum acceleration, dP/dt_{max} and the Heather Index obtained at each 10 minute period throughout the experiment. The number of stars indicates the significance level. Good correlations, all significant at the $p < 0.05$ level were found between dZ/dt_{max} and the maximum rate of change of left ventricular pressure, the peak aortic blood velocity and the peak aortic blood acceleration. The respective combined correlation coefficients were 0.937, 0.954 and 0.950. Good correlations all significant at the $p < 0.05$ level, were also found between dZ/dt_{max} and the systolic, diastolic and mean aortic blood pressures, the combined correlation coefficients being respectively 0.887, 0.887 and 0.892.

Good correlations existed between the Heather Index and the maximum rate of change of left ventricular pressure, the peak aortic blood velocity and acceleration, but the values were not quite so good as in the case of dZ/dt_{max} alone, the respective combined correlation coefficients being 0.841, 0.897 and 0.900. This trend is reflected in the combined correlation coefficients for the Heather Index and the systolic, diastolic and mean aortic pressures of 0.782, 0.757 and 0.776 respectively.

Aortic mean blood pressure and aortic diastolic blood pressure were each compared with left ventricular dP/dt_{max}, peak aortic blood velocity and peak aortic blood acceleration (Table 2). Very good correlations were found between these variables. In 28 out of 30 readings, $p < 0.001$. Also shown are the good correlations ($p < 0.001$) between left ventricular dP/dt_{max} and peak aortic flow and acceleration.

Taking the group of 5 dogs as a whole, the time interval (R- dZ/dt_{max}) was not found to correlate significantly with heart rate; pre-ejection period (PEP); $1/PEP^2$; PEP/LVET where LVET is the left ventricular ejection time; the systolic diastolic or mean aortic pressures; the maximum rate of change of left ventricular pressure; or the peak aortic velocity or acceleration.

Table 1. Correlation coefficients (r) for each dog from comparisons of contractilitiy indices and aortic blood pressure variables

Dog Number	ONE n = 13			TWO n = 9			THREE n = 11			FOUR n = 10			FIVE n = 11			All dogs r by z-transform
	r	m	c	r	m	c	r	m	c	r	m	c	r	m	c	
dZ/dt_{max}																
v Mean BP	0.804 xxx	113.4	- 34.54	0.994 xxx	190.17	- 56	0.804 xx	86.03	- 37.1	0.865 xx	46.29	4.03	0.819 xx	26.18	32.18	0.892
v Systolic BP	0.802 xxx	125.4	- 31.5	0.994 xxx	192.29	- 44.88	0.825 xx	91.85	- 24.04	0.806 xx	51.57	16.84	0.845 xx	34.35	39.12	0.887
v Diastolic BP	0.797 xxx	108.9	- 37.5	0.995 xxx	187.38	- 60.56	0.783 xx	82.45	- 42.9	0.885 xxx	44.10	- 2.53	0.753 xx	21.64	29.61	0.887
v LV dP/dt_{max}	0.957 xxx	255.75	- 109.02	0.983 xxx	3313.58	- 786.9	0.815 xx	1736.4	- 773.15	0.934 xxx	1702.3	- 483	0.918 xxx	925.15	- 31.87	0.937
v Peak velocity	0.939 xxx	11.49	- 3.34	0.989 xxx	1080.0	- 1.29	0.907 xxx	9.44	- 0.357	0.955 xxx	10.65	- 2.65	0.951 xxx	16.72	0.245	0.954
v Peak acc-eleration	0.955 xxx	2.96	- 1.16	0.958 xxx	2.72	- 0.244	0.904 xxx	2.164	- 0.038	0.954 xxx	2.51	- 0.91	0.963 xxx	3.37	- 1.1	0.950
v Heartrate	0.531 n/s	47.08	77.35	0.528 n/s	84.6	112.1	0.715 x	59.52	107.1	0.699 x	48.5	91.5	0.696 x	21.38	97.16	0.640
Heather Index																
v Mean BP				0.643 n/s	20.31	- 39.3	0.786 xx	6.5	- 15.46	0.852 xx	4.5	13.3	0.767 xx	2.91	38.7	0.776
v Systolic BP				0.644 n/s	20.58	- 28.14	0.821 xx	7.07	- 2.49	0.802 xx	5.13	26.77	0.804 xx	3.89	47.23	0.782
v Diastolic BP				0.642 n/s	19.98	- 43.9	0.760 xx	6.19	- 21.62	0.866 xx	4.32	6.53	0.701 x	2.4	35.09	0.757
v LV dP/dtmax				0.608 n/s	338.34	- 419.5	0.860 xxx	141.9	- 472.7	0.919 xxx	167.47	- 139.9	0.884 xxx	106	177.1	0.841
v Peak				0.676 x	1.21	- 0.651	0.934 xxx	0.751	1.53	0.899 xxx	1.002	- 0.127	0.934 xxx	1.95	3.74	0.897
v Peak acc-eleration				0.473 n/s	0.222	0.33	0.954 xxx	0.176	0.342	0.903 xxx	0.238	- 0.328	0.944 xxx	0.393	- 0.391	0.900

Key

m = slope
c = intercept
r = mean correlation coefficient
xxx, xx, x represent probability levels of $p < 0.001$, $p < 0.01$, $p < 0.05$ respectively
n/s = not significant

Table 2. Correlations (r) between blood pressure variables and standard contractility indices

Dog Number	ONE n = 13			TWO n = 9			THREE n = 11			FOUR n = 10			FIVE n = 11			All dogs r by z-trans-form
	r	m	c	r	m	c	r	m	c	r	m	c	r	m	c	
Mean BP																
v LV dP/dt_{max}	0.931 xxx	24.96	-488.1	0.992 xxx	17.47	186.8	0.937 xxx	18.65	80.25	0.965 xxx	32.86	-430.9	0.928 xxx	29.32	-805.4	0.956
v Peak velocity	0.926 xxx	0.111	- 0.44	0.993 xxx	0.057	1.9	0.893 xxx	0.087	5.27	0.881 xxx	0.184	- 1.19	0.906 xxx	0.499	- 11.9	0.936
v Peak acc-eleration	0.907 xxx	0.028	- 0.411	0.972 xxx	0.014	0.55	0.858 xxx	0.019	1.30	0.888 xxx	0.044	- 0.586	0.913 xxx	0.1	- 3.52	0.914
Diastolic BP																
v LV dP/dt_{max}	0.926 xxx	26.07	-381	0.993 xxx	17.77	279.44	0.922 xxx	18.64	270.5	0.973 xxx	35.58	-257.2	0.854 xxx	39.9	-623.7	0.950
v Peak velocity	0.925 xxx	0.115	- 0.007	0.994 xxx	0.058	2.20	0.873 xxx	0.086	6.187	0.900 xxx	0.201	- 0.332	0.856 xxx	0.524	- 9.511	0.931
v Peak acc-eleration	0.904 xxx	0.029	- 0.296	0.973 xxx	0.015	0.628	0.839 xx	0.019	1.50	0.905 xxx	0.048	- 0.378	0.839 xx	0.102	- 2.89	0.902
LV dP/dt_{max}																
v Peak velocity	0.981 xxx	0.004	1.78	0.991 xxx	0.003	1.33	0.909 xxx	0.004	5.19	0.962 xxx	0.006	0.834	0.983 xxx	0.017	1.71	0.974
v Peak acc-eleration	0.989 xxx	0.001	0.115	0.982 xxx	0.008	0.394	0.897 xxx	0.001	1.25	0.967 xxx	0.001	0.099	0.976 xxx	0.003	- 7.44	0.973

Key: – as for Table 1

Discussion

The maximum rate of change of left ventricular pressure has been used in this study as a standard contractility index representing cardiac muscle function against which to compare dZ/dt_{max}. Similarly, we have chosen the maximum acceleration of blood from the left ventricle [18] as a contractility index which reflects the pump function of the heart and compared it with dZ/dt_{max}.

The influence of preload on these contractility indices was considered to be minimal since the mean values for the left ventricular end-diastolic pressure were never significantly different from the initial control value. This confirms the findings of [26]. Indeed these authors reported that halothane increased the ventricular end-diastolic volume. Our changes in preload were both small and variable.

The effect of afterload on contractility indices has been shown by other investigators to be less consistent. Wallace et al. [27] demonstrated that when the left ventricular end-diastolic pressure was held constant, the maximum rate of change of left ventricular pressure was increased by raising the afterload. However, Furnival et al. [27] and Veragut and Krayenbuhl [26] showed that dP/dt_{max} was only slightly affected by changes in the aortic diastolic pressure and Mason [12] and Mason et al. [13] found no correlation between the afterload and dP/dt_{max} in the left ventricle. In the present study, the significant decrease in the aortic end-diastolic pressure during the period of 2 and 2.5 % inspired halothane concentrations may have contributed to the decreases observed in the peak values of dP/dt, aortic acceleration and dZ/dt_{max}, but no quantitative assessment was possible of the effect in the intact animal of afterload on these three variables.

Changes in the heart rate have also been shown to cause proportional changes in peak dP/dt [12, 27] but Mitchell et al. [16] and Furnival et al. [2] have reported only slight increases in peak dP/dt when the heart rate was increased. Although the heart rates steadily decreased in the present investigation from a mean value of 158 beats per minute to 125 beats per minute, none of the rates were significantly different at the $p = 0.05$ level from the control value, and the effect of heart rate on the contractility indices was therefore considered to be minimal.

The effect of halothane on the myocardial contractility has been reported by others. Using maximum aortic acceleration as an index, reductions in contractility were reported by [4, 8, 20]. In these investigations the inspired halothane concentrations ranged from 1.5 to 4 % v/v. Gersh et al. [4] attempted to correct for pre-load and after-load by also using the peak dP/dt divided by the instantaneous left ventricular pressure [26], although in their study the left ventricular end-diastolic pressure was not significantly altered. Gil-Rodriguez et al. [5] showed that inspired halothane concentrations of 2 and 2.5 % v/v in dogs caused reductions in cardiac contractility as measured by left ventricular time tension index [22], and left ventricular peak dP/dt divided by the instantaneous left ventricular pressure.

In our study, the depression of myocardial contractility by halothane was reflected in decreases of both the standard indices of contractility, namely dP/dt_{max} and aortic acceleration. Concomitant with these findings was a decrease in dZ/dt_{max} as the inspired halothane concentration was raised, although the percentage decrease was not so great. Nevertheless, a high degree of correlation was found between these variables. In 14 out of 15 comparisons it was highly significant ($p < 0.001$) thus indicating that halothane had affected each of these variables in a similar manner. In an earlier study on the effects of a progressive decrease in the circulating blood volume of dogs [7] we showed that there was a good correlation (0.917, $p < 0.001$) between dZ/dt_{max} and the left ventricular stroke work, indicating with the present study that an increase in myocardial contractility under the conditions of the experiments results in a larger ejection fraction. Hill et al. [7] also found a good correlation (0.949, $p < 0.001$) between dZ/dt_{max} and the dye dilution stroke volume.

It is interesting to note that our standard contractility indices representing muscle function (peak dP/dt) and pump function (peak aortic acceleration) were decreased by similar amounts during each experiment, and it cannot be stated from this investigation which represents the better contractility index. Neither can it be stated what proportion of the fall in the value for each index is due to changes in the pre-load, after-load and heart rate. However, it seems that under the conditions of our study, dZ/dt_{max} may be taken as a non-invasive alternative index of changes in myocardial contractility. This has lent considerable support for the justification of using the thoracic impedance technique for monitoring under intensive care situations.

An approximate value for the stroke volume may be derived non-invasively by the electrical impedance technique of Kubicek et al. [9] and this can be used as a determinant of myocardial function in two ways [17]. Either stroke volume may be plotted against left ventricular end-diastolic presssure to yield a ventricular function curve [21] or it may be utilised in the calculation of the ejection fraction. Mitchell and Wildenthal recommend the first course so that inotropic interventions can be distinguished from those accompanying the Frank-Starling mechanism. However, this approach requires access to the left ventricular pressure and the angiographic determination of left ventricular volumes. Under intensive care conditions, the simple monitoring of dZ/dt_{max} holds promise of providing significant information.

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