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Heart and Vessels

Principle of a noninvasive method of measuring Max(dP/dt) of the left ventricle: Theory and experiments

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Summary. In early systole, before the effects of reflected waves from the periphery become significant, the following equation applies:

$$PA - PO = \rho cu \quad (1)$$

where PA and PO are the instantaneous and end-diastolic pressures in the ascending aorta, ρ the density of blood, c the velocity of the pulse wave in the aorta, and u the velocity of blood. Differentiation of Eq. (1) with respect to time t yields:

$$dPA/dt = \rho c(du/dt) \quad (2)$$

If there is no aortic stenosis, and if the pressure gradient due to the inertia of the blood during acceleration is neglected, the left ventricular pressure P is nearly equal to PA during the ejection period. Since both dP/dt and dPA/dt take their maximum values at times close to the time of aortic valve opening, the following equation applies:

$$\text{Max}(dP/dt) \fallingdotseq \text{Max}(dPA/dt) \quad (3)$$

where Max signifies the maximum value of a derivative. Equation (2) reduces to:

$$\text{Max}(dPA/dt) = \rho c\,\text{Max}(du/dt) \quad (4)$$

Substitution of Eq. (4) into Eq. (3) yields:

$$\text{Max}(dP/dt) \fallingdotseq \rho c\,\text{Max}(du/dt) \quad (5)$$

Experiments were performed on seven dogs. Max(dP/dt), Max(du/dt), and c were measured during volume loading, pressure loading and unloading, and before and after administration of positive and negative inotropic agents.

There was a good linear correlation ($Y = 1.01X - 2$, $r = 0.97$) between Max(dP/dt) and $\rho c\,\text{Max}(du/dt)$. Therefore, Eq. (5) is a universal equation which holds, irrespective of the dogs and interventions employed to change the hemodynamic state. The absolute value of Max(dP/dt) of the left ventricle can be obtained by measuring noninvasively the velocity of the pulse wave and the maximum acceleration of blood in the ascending aorta.

Key words: Max(dP/dt) – Velocity of pulse wave – Velocity of blood – Maximum acceleration of blood – Noninvasive measurement

Although slightly affected by alterations in preload, the maximum value of the derivative of left ventricular pressure with respect to time, Max(dP/dt), is a simple and convenient index of cardiac contractility for clinical use [1]. However, according to a conventional method of obtaining Max(dP/dt), it is necessary to measure the left ventricular pressure with a high-fidelity catheter-tip micromanometer during catheterization. As this cannot be performed in the same patient repeatedly, it has been practically impossible to carry out follow-up investigations of changes in Max(dP/dt) in the same patient over a long period.

There have been several studies in which correlation of the maximum acceleration of blood in the ascending aorta with left ventricular contractility was attempted [2–5]. This maximum acceleration can be measured noninvasively by the ultrasound Doppler method [5–8]. However, the maximum acceleration alone cannot be used as an index of cardiac contractility, since it is markedly influenced by alterations in preload, afterload, and heart rate [1].

We derived a simple equation relating Max(dP/dt) to the maximum acceleration of blood in the ascending aorta and the pulse wave velocity, which can also be measured noninvasively [9]. The purpose of this study is to confirm experimentally the validity of this equation and to establish a noninvasive method of measuring Max(dP/dt) of the left ventricle.

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Methods

Theory

In considering the pulse wave propagation in the aorta, one-dimensional motion of the blood is assumed. The vessel is modeled as a uniform elastic tube and the blood treated as an incompressible and nonviscous fluid. The equation of continuity is:

$$\partial A/\partial t + (\partial/\partial x)(Au) = 0 \quad (1)$$

where x is the distance along the axis of the tube, t the time, u the velocity of the fluid, and A the cross-sectional area of the tube. The momentum equation is:

$$\partial u/\partial t + u(\partial u/\partial x) + (1/\rho)(\partial p/\partial x) = 0 \quad (2)$$

where ρ is the density of the fluid and p the pressure in the tube. The elastic properties of the tube wall determine the relation between p and A:

$$p = p(A) \quad (3)$$

The pulse wave velocity c is given by:

$$c^2 = (A/\rho)(dp/dA) \quad (4)$$

In general, pressure and velocity waves measured in a tube consist of forward and reflected waves. The forward wave runs into the tube and is reflected from the periphery. However, before the front of the wave reflected from the periphery, which travels backward, reaches the region concerned, the waves measured in that region consist of forward waves only. In this case, u can be expressed as a function of p. According to Eq. (3), u can also be expressed as a function of A. Based on this, Eqs. (1) and (2) can be written as:

$$(\partial A/\partial t) + [d(Au)/dA](\partial A/\partial x) = 0 \quad (5)$$

$$(\partial u/\partial t) + [u + (1/\rho)(dp/du)](\partial u/\partial x) = 0 \quad (6)$$

Since:

$$(\partial A/\partial t)/(\partial A/\partial x) = -(\partial x/\partial t)_A,$$

Eq. (5) becomes:

$$(\partial x/\partial t)_A = [d(Au)/dA] = u + A(du/dA),$$

and similarly Eq. (6) becomes:

$$(\partial x/\partial t)_u = u + (1/\rho)(dp/du).$$

Since the value of A uniquely determines that of u, the derivatives for constant A and constant u are the same, i.e.:

$$(\partial x/\partial t)_A = (\partial x/\partial t)_u,$$

so that:

$$A(du/dA) = (1/\rho)(dp/du) \quad (7)$$

From Eq. (4):

$$(du/dp) = (du/dA)/(dp/dA)$$
$$= (A/\rho c^2)(du/dA) \quad (8)$$

Combination of Eqs. (7) and (8) yields:

$$(du/dp)^2 = (1/\rho^2 c^2),$$

whence:

$$(du/dp) = \pm (1/\rho c).$$

Since ρc = constant, integration of the above equation gives:

$$u = \pm (1/\rho c) \int dp.$$

Putting $p = p_0$ when $u = 0$, we have:

$$p - p_0 = \pm \rho c u \quad (9)$$

The two signs in Eq. (9) correspond to the forward wave and reflected wave.

In the very early part of the ejection period, pressure and velocity waves in the aorta consist of forward waves alone. Therefore, we can apply Eq. (9) to the blood flow in the aorta in early systole. We obtain:

$$PA - PO = \rho c u \quad (10)$$

Where PA is the instantaneous pressure at time t in the ascending aorta, PO the end-diastolic pressure in the ascending aorta, c the velocity of the pulse wave in the aorta, ρ the density of blood, and u the velocity of blood at time t in the ascending aorta. Differentiation of Eq. (10) with respect to time t yields:

$$dPA/dt = \rho c\,(du/dt) \quad (11)$$

If there is no aortic stenosis, and if the pressure gradient due to the inertia of the blood during acceleration is neglected, the left ventricular pressure P is nearly equal to PA during the ejection period. Although dP/dt takes its maximum value just before aortic valve opening and dPA/dt takes its maximum value just after aortic valve opening, the time difference between them can be considered negligible. Therefore, the following equation applies:

$$\mathrm{Max}(dP/dt) \fallingdotseq \mathrm{Max}(dPA/dt) \quad (12)$$

where Max signifies the maximum value of a derivative. Equation (11) reduces to:

$$\mathrm{Max}(dPA/dt) = \rho c\ \mathrm{Max}(du/dt) \quad (13)$$

Substitution of Eq. (13) into Eq. (12) yields:

$$\mathrm{Max}(dP/dt) \fallingdotseq \rho c\ \mathrm{Max}(du/dt) \quad (14)$$

Therefore, if Max(du/dt) and c are measured, Max(dP/dt) can be obtained from Eq. (14) [9].

Animal experiments

Seven mongrel dogs (13–18 kg) were anesthetized with pentobarbital (30 mg/kg, intravenously) and placed on positive-pressure respiration with a mechanical respirator (Bird, Mark 7). A left thoracotomy was performed through the fourth intercostal space, and the heart was supported in a pericardial sling. An electromagnetic flowmeter transducer (Nihonkoden, FB-140T) was implanted around the ascending aorta, and a catheter-tip micromanometer (Millar Instruments, model PC360) was introduced into the left ventricle from the left atrial appendage through the mitral valve. Two other catheter-tip micromanometers (Millar Instruments, model PC370) were inserted into the left and right femoral arteries; one was advanced to the ascending aorta, and the other to the descending aorta. Continuous recordings of an electrocardiogram, pressure, and flow were made on a magnetic tape recorder (SONY, UFR-71460A). Max(dP/dt) was obtained by differentiating the left ventricular pressure with respect to time t. The cross-sectional area of the ascending aortic lumen A was calculated from the diameter which equaled the internal diameter of the electromagnetic flowmeter transducer minus the thickness of the wall of the ascending aorta. The velocity of blood u was obtained by dividing the blood flow rate of the ascending aorta F by this cross-sectional area A. Max(du/dt) was obtained by dividing Max(dF/dt) by A, where dF/dt is the derivative of F with respect to t. Differentiation of P and F was performed by differentiators which had a time constant of 0.5 ms and which were connected to the pressure and flow-rate signal amplifiers. The foot-to-foot velocity of the pulse wave was calculated from the pressure wave tracings obtained from the catheter-tip micro-

Table 1. Changes in hemodynamic parameters before and after the administration of isoproterenol

Experiment no.	Heart rate (beats/min)		Max LVP (kPa)		Max dP/dt (kPa/s)		Max u (m/s)		Max du/dt (m/s^2)		c (m/s)		ρc Max du/dt (kPa/s)	
	Cont.	Load	Cont.	Load	Cont.	Load	Cont.	Load	Cont.	Load	Cont.	Load	Cont.	Load
1	132	142	15.3	14.0	256	470	0.97	1.38	32.5	60.5	5.11	5.28	175	335
2	103	113	13.7	13.3	346	431	2.36	2.75	96.0	115.2	3.51	4.08	354	492
3	153	172	18.4	18.6	343	792	1.40	1.81	51.2	139.3	6.53	5.29	351	772
4	126	154	14.0	14.6	252	723	1.21	2.14	48.6	125.4	4.79	5.29	244	694
5	107	153	21.0	17.0	297	554	1.82	3.15	64.7	141.3	5.12	4.03	348	597
6	97	115	12.4	13.0	188	565	1.17	2.59	32.4	93.4	6.03	5.76	205	565
7	125	189	21.0	19.3	339	714	1.09	1.93	44.3	110.1	6.50	6.49	302	749
Mean	120	148	16.5	15.7	289	607	1.43	2.39	52.8	112.2	5.37	5.17	283	601
SD	19	28	3.6	2.6	60	137	0.49	0.83	22.1	28.3	1.08	0.88	75	155
P	<0.02		NS		<0.001		<0.005		<0.001		NS		<0.001	

Max maximum, *LVP* left ventricular pressure, *u* velocity of blood, *c* velocity of pulse wave, ρ density of blood, *Cont.* control condition, *Load* loading condition, *SD* standard deviation, *NS* nonsignificant

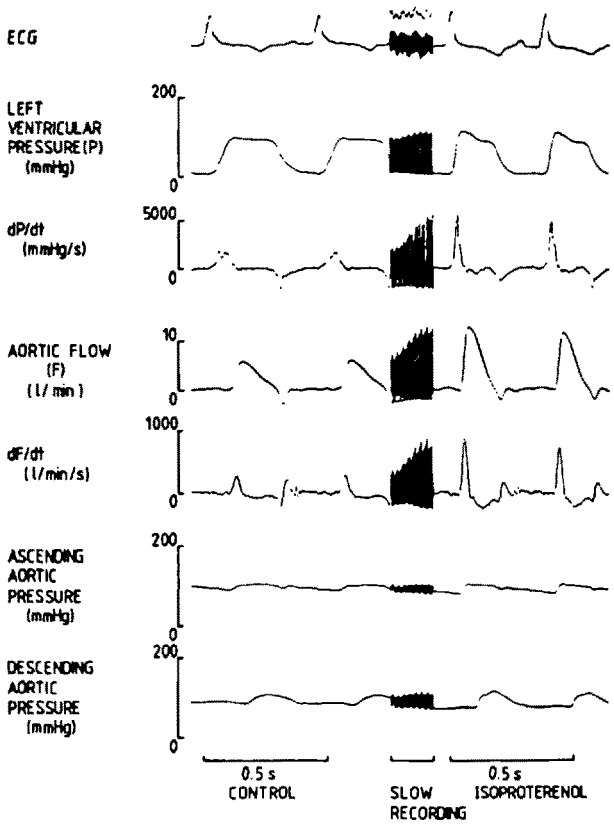

Fig. 1. Representative recordings of the electrocardiogram, left ventricular pressure and its derivative with respect to time, aortic flow and its derivative with respect to time, ascending aortic pressure, and descending aortic pressure before and after the administration of isoproterenol. The tracings were obtained from playback of magnetic tape recording

manometers located in the ascending and descending aortas. Measurements were carried out during changing cardiac contractility, preload, and afterload. Cardiac contractility was altered by the intravenous administration of isoproterenol (2–5 μg) or propranolol (10–15 mg). Preload was increased by the rapid infusion of lactate Ringer's solution (80–120 ml/min). Afterload was altered by the intravenous administration of methoxamine (2–3 mg) or phentolamine (2–3 mg). Since the purpose of these interventions was not to investigate their effects on the hemodynamic state but to change the functioning state of the heart over a wide range, we did not pay special attention to whether the effects of previous interventions had completely worn off before the following intervention.

At the end of these measurements, the thickness of the wall of the ascending aorta in situ was measured by collapsing it with a vernier caliper. The distance between the catheter-tip micromanometers located in the ascending and descending aortas was also measured. For this purpose, threads were tied around the catheters where they entered the femoral arteries to mark how far they had been inserted.

All experimental data were expressed as mean values $\pm$ SD, and statistical significance was assessed by the paired Student's *t*-test.

Results

Changes in hemodynamic state due to alterations in cardiac contractility, preload, and afterload

Administration of isoproterenol. Heart rate, Max(dP/dt), and Max(du/dt) increased significantly from 120 $\pm$ 19 to 148 $\pm$ 28 beats/min, from 289 $\pm$ 60 to 607 $\pm$ 137 kPa/s, and from 52.8 $\pm$ 22.1 to 112.2 $\pm$ 28.3 m/s^2, respectively, but the velocity of the pulse wave remained unchanged (Table 1). Figure 1 shows typical recordings before and after the administration of isoproterenol (experiment no.4).

Administration of propranolol. Heart rate, Max(dP/dt), and Max(du/dt) decreased significantly from 131 $\pm$ 25 to 108 $\pm$ 20 beats/min, from 458 $\pm$ 124 to 273 $\pm$ 81 kPa/s, and from 81.4 $\pm$ 27.2 to 52.3 $\pm$ 25.9 m/s^2, respectively, but the velocity of the pulse wave remained unchanged (Table 2). Figure 2 shows typical recordings before and after the administration of propranolol (experiment no.3).

Table 2. Changes in hemodynamic parameters before and after the administration of propranolol

Experiment no.	Heart rate (beats/min)		Max LVP (kPa)		Max dP/dt (kPa/s)		Max u (m/s)		Max du/dt (m/s^2)		c (m/s)		ρc Max du/dt (kPa/s)	
	Cont.	Load	Cont.	Load	Cont.	Load	Cont.	Load	Cont.	Load	Cont.	Load	Cont.	Load
1	127	87	16.0	17.3	284	200	0.97	0.94	38.8	22.8	6.08	5.85	240	140
2	96	91	14.0	13.3	544	375	2.45	2.36	107.0	98.4	4.80	3.35	539	346
3	164	132	18.0	16.6	540	265	1.73	0.85	100.3	44.3	5.63	5.73	591	268
4	124	113	16.6	16.6	416	362	1.64	1.60	84.7	71.8	5.54	4.86	493	366
5	138	106	19.0	18.0	495	198	2.39	1.47	108.2	47.1	4.51	4.51	511	223
6	108	92	12.6	11.7	314	188	1.68	0.89	52.1	28.8	6.03	6.67	330	201
7	160	136	19.0	18.0	612	322	1.65	1.25	78.5	53.0	8.12	6.49	669	361
Mean	131	108	16.5	15.9	458	273	1.78	1.34	81.4	52.3	5.81	5.30	482	272
SD	25	20	2.5	2.5	124	81	0.50	0.54	27.2	25.9	1.17	1.28	149	89
P	<0.005		NS		<0.005		<0.05		<0.02		NS		<0.005	

Abbreviations as in Table 1

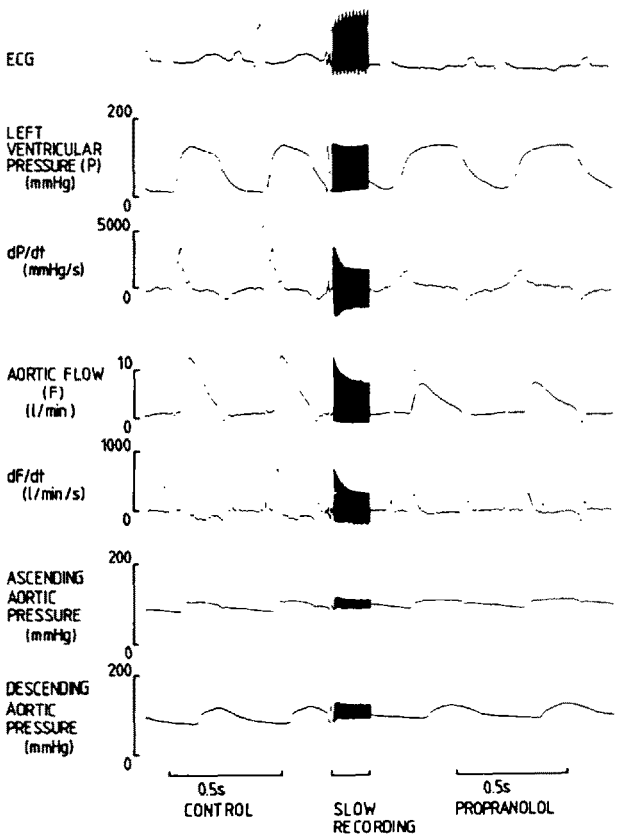

Fig. 2. Representative recordings before and after the administration of propranolol. Arranged as Fig. 1. The tracings were obtained from playback of magnetic tape recording

Infusion of lactate Ringer's solution. The maximum value of the left ventricular pressure, the left ventricular end-diastolic pressure, and the maximum value of the velocity of blood increased significantly from 15.6 ± 2.9 to 18.1 ± 3.9 kPa, from 0.91 ± 0.44 to 1.39 ± 0.52 kPa, and from 1.33 ± 0.31 to 1.67 ± 0.49 m/s, respectively, but Max(dP/dt) and the velocity of the pulse wave remained unchanged (Table 3). Figure 3 shows typical recordings before and after the rapid infusion of lactate Ringer's solution (experiment no.3).

Administration of methoxamine. The maximum value of the left ventricular pressure and the velocity of the pulse wave increased significantly from 16.3 ± 1.9 to 21.4 ± 2.7 kPa and from 5.64 ± 1.13 to 8.06 ± 2.69 m/s, respectively, and Max(du/dt) decreased significantly from 68.1 ± 27.6 to 52.7 ± 30.0 m/s^2. Max(dP/dt) remained unchanged (Table 4). Figure 4 shows typical recordings before and after the administration of methoxamine (experiment no.4).

Administration of phentolamine. The maximum value of the left ventricular pressure and the velocity of the pulse wave decreased significantly from 18.8 ± 2.5 to 15.1 ± 2.7 kPa and from 6.19 ± 1.13 to 5.35 ± 0.94 m/s, respectively, and Max(du/dt) increased significantly from 51.0 ± 30.1 to 61.9 ± 33.5 m/s^2. Max(dP/dt) remained unchanged (Table 5). Figure 5 shows typical recordings before and after the administration of phentolamine (experiment no.3).

Relation between Max(dP/dt) *and* ρc Max(du/dt)

Figure 6 shows the relation between Max(dP/dt) and ρc Max(du/dt) in seven dogs. There was a good linear correlation (Y = 1.01X–2, r = 0.97) between these two quantities irrespective of the dogs and interventions employed to change the hemodynamic state.

Discussion

Equation (10) has been quoted in several books [10–12] and is sometimes termed the "water-

Table 3. Changes in hemodynamic parameters before and after the rapid infusion of lactate Ringer's solution

Experiment no.	Max LVP (kPa)		LVEDP (kPa)		Max dP/dt (kPa/s)		Max u (m/s)		Max du/dt (m/s^2)		c (m/s)		ρc Max du/dt (kPa/s)	
	Cont.	Load	Cont.	Load	Cont.	Load	Cont.	Load	Cont.	Load	Cont.	Load	Cont.	Load
1	18.0	19.7	1.00	1.60	339	405	1.14	1.44	47.3	54.1	6.95	5.96	345	339
2	12.0	14.2	0.33	0.83	282	363	1.88	2.55	61.9	59.8	4.35	5.94	283	373
3	17.3	19.3	1.33	1.33	488	599	1.45	1.60	88.8	99.5	5.58	5.73	520	599
4	13.6	17.0	0.50	0.67	416	421	1.42	1.92	66.2	96.2	5.54	4.84	385	489
5	17.7	21.0	1.33	2.00	231	297	1.38	1.84	46.3	65.1	4.91	4.91	239	336
6	12.0	12.4	0.53	1.33	241	184	0.90	1.08	34.2	33.1	6.67	6.60	240	229
7	18.4	23.7	1.33	2.00	382	416	1.16	1.25	49.7	46.4	7.01	8.27	366	403
Mean	15.6	18.1	0.91	1.39	340	384	1.33	1.67	56.3	64.9	5.85	6.04	340	395
SD	2.9	3.9	0.44	0.52	95	127	0.31	0.49	17.8	24.7	1.04	1.16	99	119
P	<0.005		<0.005		NS		<0.01		NS		NS		<0.05	

LVEDP left ventricular end-diastolic pressure; other abbreviations as in Table 1

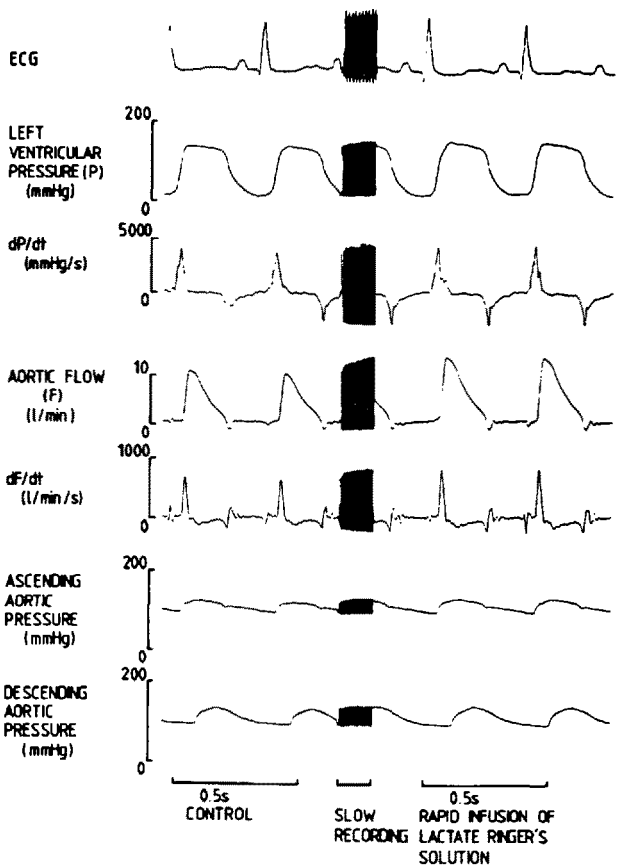

Fig. 3. Representative recordings before and after the rapid infusion of lactate Ringer's solution. Arrange as in Fig. 1. The tracings were obtained from playback of magnetic tape recording

hammer" formula [13]. However, no explicit description of the method of deriving this equation from the nonlinear basic Eqs. (1)–(4) has been given. We derived Eq. (10) by following the method of Landau and Lifshitz [14] for obtaining the fundamental characteristics of one-dimensional traveling waves in a compressible gas. The pressure and flow wave tracings published by van den Bos et al. [15] indicated a good linear relation between PA and u during their upstroke and would lend support to Eq. (10). Two assumptions were made in order to derive Eq. (12). The first one is that the pressure in the left ventricle is equal to that in the ascending aorta during the ejection period. Strictly speaking, there is a difference between these two pressures due to inertia, i.e., there is a pressure gradient which forces the blood to accelerate and decelerate [16]. However, within the accuracy of practical pressure measurements, it is rather difficult to detect this pressure difference and so it was not considered. The second assumption is related to the difference between the times at which dP/dt and dPA/dt reach their maximum values. Although both dP/dt and dPA/dt take their maximum values at times close to the time of aortic valve opening, dP/dt reaches its maximum value slightly earlier than dPA/dt [1]. However, around the time of aortic valve opening, the left ventricular pressure P increases almost linearly. Therefore, dP/dt retains nearly the same value as its maximum until dPA/dt reaches its maximum.

In Figs. 1–5, it seems that there is a considerable difference between the times when dP/dt and dF/dt (i.e., du/dt) take their maximum values. However, in the electromagnetic flowmeter, there is a delay of 20–30 ms between the input and output signals, while there is little delay in the pressure-measuring system. If the time delay is adjusted, the difference between the times when dP/dt and dF/dt take their maximum values is not so large.

The blood flow rate (i.e., the velocity of blood) was measured with an electromagnetic flowmeter transducer, which tethered a periodic expanding motion of the ascending aorta caused by pulsating pressure. This tethering might have altered the velocity of the pulse wave in the ascending aorta. Strictly speaking, the pulse wave velocity used in

Table 4. Changes in hemodynamic parameters before and after the administration of methoxamine

Experiment no.	Heart rate (beats/min)		Max LVP (kPa)		Max dP/dt (kPa/s)		Max u (m/s)		Max du/dt (m/s^2)		c (m/s)		ρc Max du/dt (kPa/s)	
	Cont.	Load	Cont.	Load	Cont.	Load	Cont.	Load	Cont.	Load	Cont.	Load	Cont.	Load
1	131	110	16.0	24.3	279	335	0.94	0.75	36.2	22.1	5.28	11.00	191	256
2	100	95	14.0	17.0	378	473	2.36	2.02	96.0	92.7	4.08	5.69	411	553
3	154	148	19.3	22.6	551	608	1.86	1.19	105.3	85.3	5.54	7.22	610	640
4	120	121	16.6	20.0	416	416	1.75	1.57	85.5	68.2	4.88	6.59	438	472
5	153	142	17.0	22.2	363	330	1.41	1.27	58.8	51.1	5.64	6.26	348	336
6	146	124	13.7	19.2	297	251	1.06	0.96	38.8	26.6	6.49	9.04	264	252
7	167	136	17.3	24.3	406	310	1.16	0.60	56.4	22.8	7.59	12.20	449	292
Mean	139	125	16.3	21.4	384	389	1.51	1.19	68.1	52.7	5.64	8.06	387	400
SD	23	19	1.9	2.7	90	120	0.51	0.49	27.6	30.0	1.13	2.69	137	155
P	<0.02		<0.001		NS		<0.01		<0.01		<0.05		NS	

Abbreviations as in Table 1

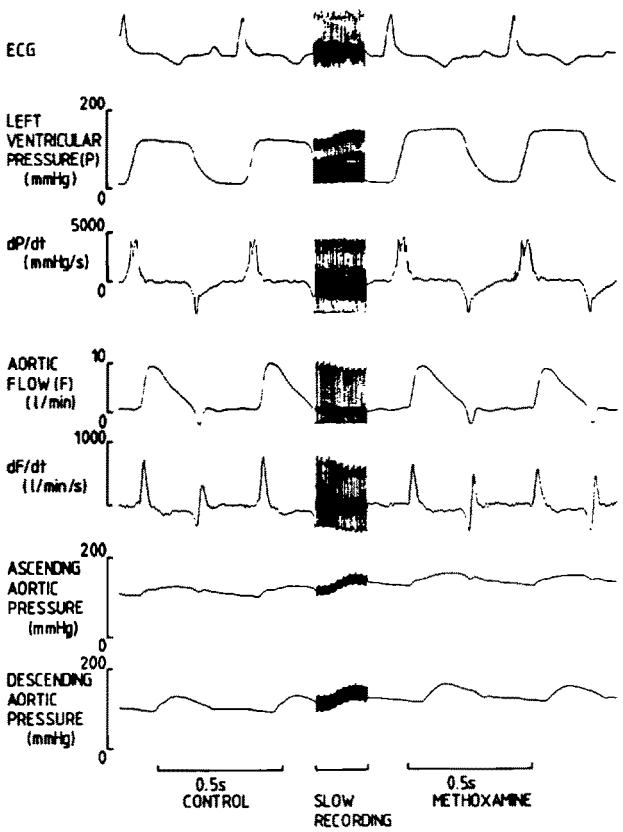

Fig. 4. Representative recordings before and after the administration of methoxamine. Arranged as in Fig. 1. The tracings were obtained from playback of magnetic tape recording

Eq. (10) should be the local pulse wave velocity in the ascending aorta. In our experiments, however, an average pulse wave velocity was used, which was measured over the distance between the ascending and descending aortas. This was to simulate a clinical situation of noninvasive measurement in which only an average pulse wave velocity is measurable. It is considered that the pulse wave velocity averaged over a long distance in the aorta is somewhat greater than the local pulse wave velocity in the ascending aorta. In consideration of such intrinsic inaccuracy of the noninvasive method, the influence of tethering aortic expansion by an electromagnetic flowmeter transducer does not seem to be important for practical measurements of the pulse wave velocity. The so-called foot-to-foot velocity was measured as the velocity of the pulse wave. It was rather difficult to read accurately the time difference between the lowest points of pressure wave tracings from the ascending and descending aortas [17]. It was thought that there was an error of 10% at most in the reading of this time difference.

Although it is difficult to know how the abovementioned factors for the error influence its accuracy, Eq. (14) holds with sufficient precision for practical applications, as shown by the results of our experiments (Fig. 6).

Several attempts have been made to correlate the blood flow in the ascending aorta and the left ventricular contractility. Benett et al. [2] measured the velocity of blood in the ascending aorta using a catheter-tip velocity probe in patients with ischemic heart disease. They reported that the maximum values of the velocity and the acceleration of blood could be correlated with the ejection fraction of the left ventricle, and their maximum values declined according to the severity of the disease. Sabbah et al. [5] obtained similar results using a continuous-wave Doppler velocity meter.

Jewitt et al. [3] also measured the velocity and the acceleration of blood in the ascending aorta with a catheter-tip velocity probe in 24 patients with ischemic heart disease. They pointed out a linear relationship between the maximum values of velocity and acceleration and suggested that these maximum values are related to left ventricular function. They reported a linear relationship between the

Table 5. Changes in hemodynamic parameters before and after the administration of phentolamine

Experiment no.	Heart rate (beats/min)		Max LVP (kPa)		Max dP/dt (kPa/s)		Max u (m/s)		Max du/dt (m/s^2)		c (m/s)		ρc Max du/dt (kPa/s)	
	Cont.	Load	Cont.	Load	Cont.	Load	Cont.	Load	Cont.	Load	Cont.	Load	Cont.	Load
1	107	125	22.2	15.7	216	277	0.75	1.10	24.3	41.0	6.40	5.76	163	248
2	92	95	15.7	13.6	447	454	2.18	2.47	96.0	98.4	4.35	4.11	438	411
3	141	157	20.0	18.0	352	639	1.41	1.65	76.7	105.0	5.54	5.73	446	632
4	120	120	19.3	17.0	438	399	1.66	1.85	75.5	86.5	6.59	4.62	522	420
5	121	121	16.3	12.0	116	125	0.92	1.03	21.6	24.3	5.64	4.51	128	115
6	123	144	16.9	11.6	241	251	0.98	0.96	28.8	35.3	7.82	6.67	225	247
7	135	142	20.9	17.7	287	294	0.88	1.01	34.3	42.9	7.01	6.07	252	273
Mean	120	129	18.8	15.1	300	348	1.25	1.44	51.0	61.9	6.19	5.35	311	337
SD	16	21	2.5	2.7	121	166	0.52	0.57	30.1	33.5	1.13	0.94	156	169
P	<0.05		<0.005		NS		<0.01		<0.05		<0.02		NS	

Abbreviations as in Table 1

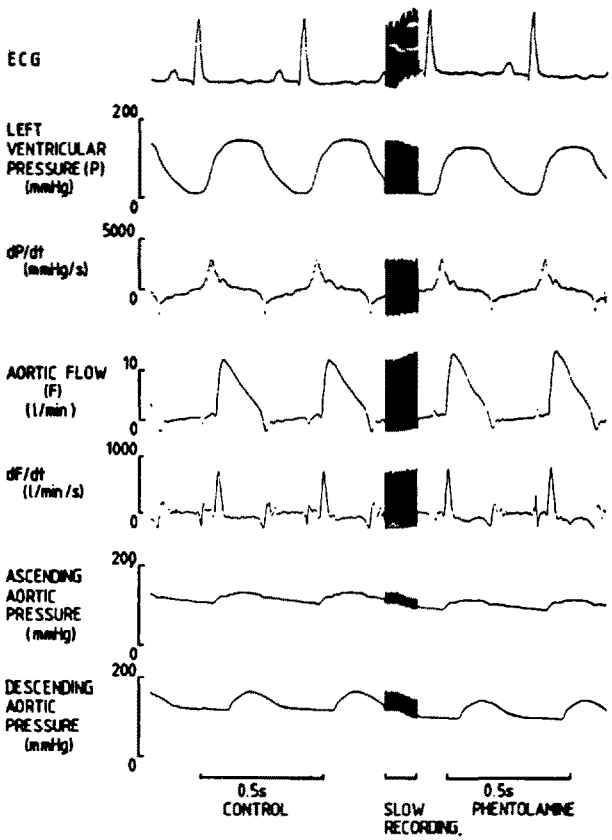

Fig. 5. Representative recordings before and after the administration of phentolamine. Arranged as in Fig. 1. The tracings were obtained from playback of magnetic tape recording

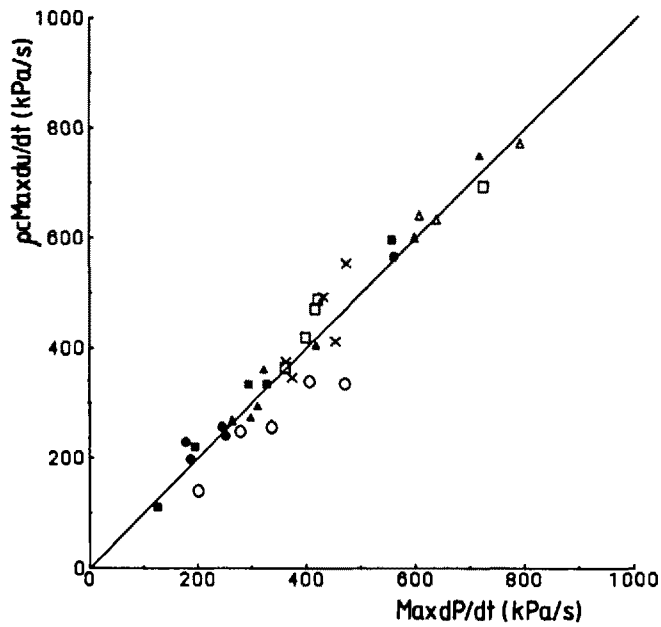

Fig. 6. Relation between Max(dP/dt) and ρcMax(du/dt). Data from seven dogs have been pooled together. The *solid line* is the linear regression line: $Y = 1.01X - 2$ ($r = 0.97$)

maximum value of the acceleration of blood in the ascending aorta, i.e., Max(du/dt), and Max(dP/dt) of the left ventricle in a patient with mitral valve insufficiency. However, this linear relationship does not hold when afterload alters. As shown in Fig. 4, the administration of methoxamine, which is believed not to change the contractility, did not change Max(dP/dt) but clearly decreased the maximum value of the blood flow in the aorta, i.e., the maximum value of the velocity of blood, and Max(dF/dt), i.e., Max(du/dt). In this case, the velocity of the pulse wave was increased, and Eq. (14) still held. Conversely, the administration of phentolamine did not change Max(dP/dt) but increased Max(du/dt) (Table 5). In this case, the velocity of the pulse wave decreased, and Eq. (14) also held. Because it changes significantly due to the alteration in afterload, as mentioned above, Max(du/dt) alone cannot be an index of the contractility of the heart muscle [1].

Nowadays, the maximum acceleration of the blood in the ascending aorta can be measured by the ultrasound Doppler method [5–8], and pulse wave

velocity can be obtained from pulse wave tracings. Therefore, the absolute value of Max(dP/dt) of the left ventricle can be obtained by using Eq. (14) from noninvasive measurements only. Although Max(dP/dt) of the left ventricle is slightly affected by alterations in preload, it is much more markedly affected by changes in the contractile state [1]. Therefore, the feasibility of noninvasive, hence repeatable, measurement of Max(dP/dt) will certainly lead to re-evaluation of its usefulness in the analysis of cardiac function from the clinical viewpoint.

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References

1. Braunwald E, Ross J Jr, Sonnenblick EH (1976) Mechanisms of contraction of the normal and failing heart, 2nd edn. Little, Brown and Company, Boston, pp 130–165
2. Benett ED, Else W, Millar GAH, Sutton GC, Miller HC, Noble MIM (1974) Maximum acceleration of blood from the left ventricle in patients with ischaemic heart disease. Clin Sci Molec Med 46: 49–59
3. Jewitt D, Gabe I, Mills C, Maurer B, Thomas M, Shillingford J (1974) Aortic velocity and acceleration measurements in the assessment of coronary heart disease. Eur J Cardiol 1: 299–305
4. Wallmeyer K, Wann S, Sagar KB, Kalbfleisch J, Klopfenstein HS (1986) The influence of preload and heart rate on Doppler echocardiographic indexes of left ventricular performance: comparison with invasive indexes in an experimental preparation. Circulation 74: 181–186
5. Sabbah HN, Khaja F, Brymer JF, McFarland TM, Albert DE, Synder JE, Goldstein S, Stein PD (1986) Noninvasive evaluation of left ventricular performance based on peak aortic blood acceleration measured with a continuous-wave Doppler velocity meter. Circulation 74: 323–329
6. Light H (1976) Transcutaneous aortovelography: a new window on the circulation? Br Heart J 38: 433–442
7. Sequeira RF, Light LH, Gross G, Raftery EB (1976) Transcutaneous aortovelography: a quantitative evaluation. Br Heart J 38: 443–450
8. Buchtal A, Hanson GC, Peisach AR (1976) Transcutaneous aortovelography: potentially useful technique in management of critically ill patients. Br Heart J 38: 451–456
9. Sugawara M, Nakata S (1985) Relation between the maximum acceleration of blood in the aorta and maximum dP/dt of the left ventricle. Proc 46th Meeting Jpn Soc Ultrason Med: 751–752 (abstract)
10. Jones RT (1972) Fluid dynamics of heart assist devices. In: Fung YC, Perrone N, Anliker M (eds) Biomechanics: its foundation and objectives. Prentice-Hall, Englewood Cliffs, pp 549–565
11. McDonald DA (1974) Blood flow in arteries. Edward Arnold, London, pp 284–285
12. Caro CG, Pedley TJ, Schroter RC, Seed WA (1978) The mechanics of the circulation. Oxford University Press. Oxford, pp 282–283
13. Taylor MG (1964) Wave travel in arteries and the design of the cardiovascular system. In: Attinger EO (ed) Pulsatile blood flow. McGraw-Hill, New York, pp 343–372
14. Landau LD, Lifshitz EM (1959) Fluid mechanics. Pergamon, Oxford, pp 366–367 (translated by Sykes JB Reid WH)
15. van den Bos GC, Westerhof N, Randall OS (1982) Pulse wave reflection: Can it explain the difference between systemic and pulmonary pressure and flow waves? Circ Res 51: 479–485
16. Noble MIM (1968) The contribution of blood momentum to left ventricular ejection in the dog. Circ Res 23: 663–670
17. McDonald DA (1968) Regional pulse-wave velocity in the arterial tree. J Appl Physiol 24: 73–78