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Research Article

A PRACTICAL STUDY OF THE AIR CHAMBER MODEL OF THE CARDIOVASCULAR SYSTEM

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Use of the air chamber as a simplified model of the cardiovascular system was studied for many years (1, 2). Subsequently, interest in this model declined, since it failed to yield strikingly useful results. Unfortunately, other methods for describing the behavior of the cardiovascular system in terms of such easily measurable quantities as arterial pressure and pulse rate also were unsuccessful (3, 4). The recent development by one of the authors of a more exact mathematical approach to the air chamber spotlighted the strengths and weaknesses of this model anew (5). Its chief strengths were its great simplicity and the fact that its theoretical behavior agreed with a considerable body of cardiovascular physiology such as the general shape of the arterial pressure curve (5). Its weakness, from the practical viewpoint, was that its application required the use of parameters which could not be determined by direct measurement in the body. The present study of the air chamber model had two objectives. In the first place, an attempt was made to evaluate the closeness of mathematically predicted results to measured data. Then, since the mathematical approach appeared to have validity, the practical usefulness of the model itself was evaluated.

EXPERIMENTAL

The source of experimental data was the human cardiac catheterizations performed at the Louisville General Hospital during the past three years. Only patients with cardiovascular abnormalities requiring further investigation were subjected to cardiac catheterization. Not used in the present study were records incomplete either because of technical difficulties or for other reasons, such as the fact that the cardiac output was not determined in patients below the age of 13 years. In addition, a few catheterizations were omitted because of auricular fibrillation. All other 59 records of the three year period were included, regardless of postcatheterization diagnosis. Previously described techniques of cardiac catheterization were used (6). Data adequate for this study included the output from the left ventricle into the aorta, the average arterial pressure and the arterial pressure tracing.

The quantities necessary for a mathematical description of the chamber model were as follows:

t = time
$P(t)$ = pressure in the aorta near the aortic valve. In the present study, this was approximated by the brachial artery pressure (7)
$V(t)$ = volume equivalent to the aorta
$F(t)$ = effective rate of peripheral blood flow
P_D = end-diastolic pressure
P_S = maximum systolic pressure
$PP = P_S - P_D$ = pulse pressure
$K = \Delta P/\Delta V$ = elasticity of the aorta
$R = P(t)/F(t)$ = effective peripheral resistance
i = seconds between the onset of systole and the occurrence of P_S
P_i = decrease in pressure in the i seconds after the onset of diastole
CO = number of liters of blood passing the aortic valve per minute
P_{av} = average aortic pressure
a = duration of the cardiac cycle in seconds.

The values which were determined by use of the tracing of the brachial artery pressure included P_D, P_S, PP, i, P_i, a, and the ratio, $-K/R$ ($= G$) (Figure 1). P_i and G were determined from plots of the natural logarithms of the diastolic pressures in the tracings *vs.* time. The onset of a rapid drop in aortic pressure was arbitrarily taken as the beginning of diastole. Ordinarily, the time of this rapid drop could be determined within 0.02 second without difficulty. In nearly every case, a straight line represented the relationship between ln P(t) and t during diastole very closely (Figure 2). The value of G was the slope of this line. P_i was also determined from this line, since the initial portion of the actual pressure tracing during diastole

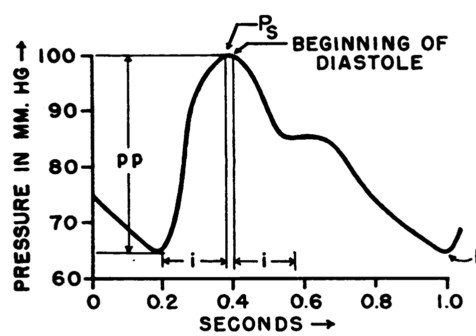

FIG. 1. A PATTERN OF BRACHIAL ARTERY PRESSURE
For this curve, a equals 0.8 second.

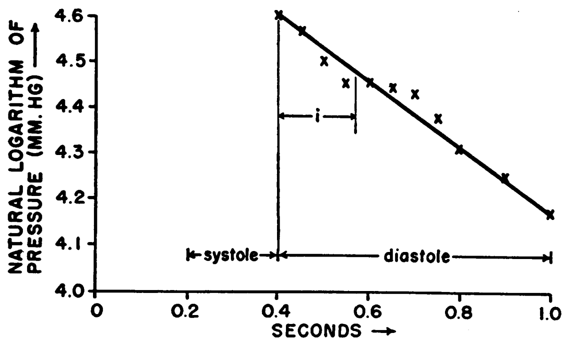

FIG. 2. A PLOT OF THE NATURAL LOGARITHM OF P *vs.* T FOR THE DIASTOLIC PRESSURE OF FIGURE 1

P_i equals 12 mm. Hg; $-K/R$ equals 0.72 second^{-1}.

was distorted somewhat from the theoretically anticipated logarithmic decline as a result of the closure of the aortic valve. In each arterial pressure tracing, three consecutive, nearly identical cycles were averaged. Quantities which were independently measured included P_{av} and CO.

Only one equation which could be tested in a practical way was derived from the mathematical formulation. This was

$$P_{av} = \frac{PP + P_i}{aG}. \qquad 1)$$

All of the quantities on the right hand side of Equation *1* were determined from the arterial pressure tracing alone. The value of P_{av} calculated from Equation *1* was then compared with the measured value, and the closeness of the two values provided an indication of the overall reasonableness of the theory. R was determined from the relationship, $R = \frac{60\ P_{av}}{CO}$, and substitution of this value of R into $-K/R = G$ yielded K.

RESULTS

The results of the 59 catheterizations were compiled in Table I. The ages of the patients ranged from 14 to 58 years, the average age being 31.5, with a standard deviation of 13 years. There were only six normal catheterization studies. Diagnoses included aortic insufficiency, mitral stenosis, mitral insufficiency, patent ductus arteriosus, peripheral arteriovenous fistula, anomolous pulmonary vein, interauricular septal defect and coarctation of the aorta. The wide range of each of the measured parameters was consistent with the great variety of pathology present.

On the average, the calculated value of P_{av} ($= P_{av-c}$) was 1.07 ± 0.07 times the measured value ($= P_{av-m}$), while, if P_i were omitted, P_{av-c} became 0.86 ± 0.09 times P_{av-m}. In each case, the number after the $\pm$ sign was the standard deviation. For the six patients with normal catheterization findings, P_{av-c} was 1.04 ± 0.07 times P_{av-m}. On the average, P_i was 25 ± 8 per cent of the corresponding pulse pressure. With use of the directly measured value of P_{av}, the mean value of R was $1,620 \pm 760$ dyne-seconds per cm.^{5}, and the average value of K was $1,470 \pm 770$ dynes per cm.^{5}, while the average value of K/R was 0.92 ± 0.33 seconds^{-1}. When the calculated value of P_{av} was used, the mean value of both R and K increased by 7 per cent, a change not statistically significant.

DISCUSSION

In the present study, P_{av-c} was nearly equal to P_{av-m} for a wide variety of cardiovascular pathology, as well as for normal hearts, over a range of ages from 14 to 58 years. The closeness of the theoretical and measured values of P_{av} indicated the practical feasibility of the air chamber model, insofar as this could be tested at present. The demonstration of the general validity of the theoretical approach was of considerably greater importance than was mere development of another method for the determination of P_{av}. From an empirical point of view, indeed, P_{av} might be more closely and easily approximated by such relationships as the sum of the diastolic pressure and one-third the pulse pressure than by Equation *1*. The significance of Equation *1*, however, lay in the fact that it was derived from application of the mathematical theory to the chamber model and could have no other origin, pragmatic or theoretical.

Some of the small deviation of P_{av-c} from P_{av-m} was due to errors in the measurements alone, such as those from the use of the Fick principle, and to the fact that all of the measurements were not recorded simultaneously. In addition, the brachial artery pressure differed from the pressure at the root of the aorta, among the differences being a slightly earlier occurrence and slightly greater magnitude of the peak of systolic pressure in the brachial artery than in the aorta (*7*). The term P_i brought the calculated value of P_{av} considerably closer to the observed value and decreased the deviation of the ratio, P_{av-c}/P_{av-m}, from its average value. For individuals undergoing exercise, with rapid heart rate, vigorous heart action and rapid initial decline of diastolic pressure, the relative

TABLE I

Results of 59 cardiac catheterization studies *

Case	Initials	Age	Sex	Diagnosis	P_S	PP	P_{av-m}	CO	m	a	R†	K†	$\frac{P_i}{PP} \times 100$	$\frac{P_{av-c}}{P_{av-m}} \times 100$
no.					*mm. Hg*	*mm. Hg*	*mm. Hg*			*sec.*	*dyne-sec./cm.⁵*	*dyne/cm.⁵*		
1	C. M.	14	M	Valvular pulmonic stenosis	112	52	75	1.86	7	0.65	3,220	3,870	17	105
2	E. W.	14	M	Coarctation of aorta	149	57	117	7.62	4	0.67	1,230	980	23	112
3	J. B.	15	M	Infundibular pulmonic stenosis	140	60	100	4.75	6	0.70	1,680	1,680	23	106
4	C. T.	15	F	Patent ductus arteriosus	116	47	94	7.31 (L→R 2.29)	4	0.56	1,030	1,160	26	99
5	H. M.	15	M	Patent ductus arteriosus	116	56	80	4.85 (L→R 1.55)	4	0.60	1,310	1,880	38	112
6	M. M.	15	F	Acute idiopathic pericarditis Normal catheterization	121	51	92	6.40	6	0.73	1,150	970	20	108
7	R. M.	17	M	Coarctation of aorta-aortic insufficiency	190	116	130	7.07	6	0.84	1,470	1,860	17	98
8	H. G.	17	M	Normal	130	60	90	7.79	7	0.65	920	1,050	15	103
9	G. S.	17	F	Rheumatic heart disease, mitral stenosis	84	27	65	2.80	5	0.98	1,850	1,000	30	102
10	R. P.	18	M	Patent ductus arteriosus	105	45	80	9.90 (L→R 4.15)	6	0.60	650	760	24	99
11	B. G.	18	F	Pulmonary hypertension of undetermined etiology	134	54	96	6.19	4	0.57	1,240	1,490	30	106
12	C. M.	18	F	Patent ductus arteriosus	98	34	76	2.51 (L→R 0.46)	4	0.96	2,410	1,210	21	112
13	J. M.	19	M	Pulmonic stenosis	109	33	85	7.28	5	0.95	930	400	18	112
14	M. C.	20	F	Normal	94	36	70	3.26	5	0.81	1,720	1,240	22	108
15	A. P.	21	F	Normal	123	70	80	8.10	4	0.80	790	1,030	31	110
16	B. C.	22	F	Rheumatic heart disease, mitral stenosis	90	28	74	4.11	3	0.83	1,440	760	36	117
17	S. H.	22	M	Normal	106	36	94	2.93	5	0.62	2,560	2,300	31	90
18	W. J.	22	F	Rheumatic heart disease, mitral stenosis	99	33	77	4.24	8	0.76	1,450	870	6	100
19	M. W.	23	F	? Interatrial septal defect	97	33	80	3.94	7	0.78	1,620	960	15	103
20	J. W.	23	F	Rheumatic heart disease, mitral stenosis	102	32	78	2.95	6	0.68	2,110	1,370	19	110
21	P. M.	23	F	Coarctation of aorta	230	110	160	11.2	5	0.69	1,150	1,410	24	100
22	B. T.	25	F	Anomalous pulmonary vein	111	41	88	3.55	5	0.80	1,960	1,350	22	104
23	A. M.	25	M	Valvular pulmonic stenosis	126	46	90	5.59	6	0.86	1,280	860	22	108
24	B. H.	25	F	Rheumatic heart disease, mitral stenosis	124	50	85	4.83	4	0.74	1,410	1,270	34	118
25	J. W.	26	M	Rheumatic heart disease, mitral stenosis	158	69	110	3.57	7	0.76	2,460	2,070	13	111

* Abbreviations are as follows: P_S, maximum systolic pressure; P_D, end-diastolic pressure; PP ($= P_S - P_D$), pulse pressure; P_{av-m}, average aortic pressure, measured value; CO, number of liters of blood passing aortic valve per minute; m, integer nearest to the actual value of the ratio, $\frac{a}{\text{duration of systole in seconds}}$; a, duration of cardiac cycle in seconds; R, effective peripheral resistance; K, elasticity of the aorta; P_i, decrease in pressure in the i seconds after the onset of diastole; i, seconds between the onset of systole and the occurrence of P_S; P_{av-c}, average aortic pressure, calculated value.

† K and R were determined with the use of P_{av-m}.

TABLE I—*Continued*

Case	Initials	Age	Sex	Diagnosis	Ps	PP	P_{av-m}	CO	m	a	R†	K†	$\frac{P_1}{PP} \times 100$	$\frac{P_{av-c}}{P_{av-m}} \times 100$
no.					*mm. Hg*	*mm. Hg*	*mm. Hg*			*sec.*	*dyne-sec./cm.*[5]	*dyne/cm.*[5]		
26	W. W.	26	M	? Interatrial septal defect	100	45	80	5.73	5	0.79	1,110	890	20	107
27	R. M.	26	F	Rheumatic heart disease, mitral insufficiency	96	36	74	2.75	3	0.69	2,140	1,710	31	115
28	H. T.	26	M	Coarctation of aorta, aortic insufficiency	192	108	117	11.40	5	0.64	820	1,310	24	112
29	C. B.	27	F	Rheumatic heart disease, mitral stenosis	100	30	82	4.20	4	0.56	1,560	1,450	47	103
30	H. J.	27	F	Normal	124	54	88	4.85	7	0.67	1,460	1,460	13	103
31	B. M.	29	M	Rheumatic heart disease, aortic stenosis and insufficiency, mitral stenosis	140	88	90	4.98	4	0.78	1,440	2,380	26	96
32	R. P.	30	F	Pulmonary arteriovenous fistula	110	57	72	9.30	4	0.74	620	720	32	121
33	A. M.	30	F	Rheumatic heart disease, mitral stenosis, post commisurotomy	105	20	92	4.30	3	0.68	1,710	790	30	90
34	A. M.	30	F	Rheumatic heart disease, mitral stenosis, pre-commisurotomy	105	42	83	2.30	4	0.69	2,880	3,180	43	95
35	J. C.	35	M	Rheumatic heart disease, mitral stenosis	93	28	72	4.00	5	0.54	1,440	1,050	21	120
36	N. S.	36	F	Patent ductus arteriosus	130	60	100	6.45	4	0.46	1,240	2,260	40	100
37	L. T.	38	M	Arteriosclerotic heart disease, with left heart failure	118	33	95	1.91	6	0.55	3,970	2,780	18	107
38	L. T.	39	F	Anomalous pulmonary vein	109	42	90	3.14	4	0.69	2,290	2,020	26	97
39	G. S.	40	M	Coarctation of aorta	148	68	100	4.91	5	0.73	1,630	1,650	28	118
40	J. B.	40	M	Hypertensive cardiovascular disease, with left heart failure	168	62	129	5.20	4	0.68	1,980	1,690	26	104
41	J. R.	41	M	Rheumatic heart disease, mitral stenosis	100	30	80	7.71	4	0.65	830	620	37	105
42	G. C.	41	F	Pulmonary hypertension of undetermined etiology	143	48	110	10.80	3	0.77	810	540	35	115
43	G. T.	41	M	Arteriosclerotic heart disease, with right and left ventricular failure	130	53	105	4.15	5	0.73	2,020	1,720	23	100
44	E. O.	42	M	Arteriosclerotic heart disease, with right and left ventricular failure	121	46	88	3.94	5	0.76	1,780	1,250	15	113
45	T. T.	43	M	Traumatic arteriovenous fistula, right thigh	128	60	85	10.9	5	0.97	740	560	28	123
46	G. D.	43	F	Arteriosclerotic heart disease, with left ventricular failure	119	57	82	5.41	4	0.72	1,210	1,410	33	111
47	W. H.	43	M	Pulmonary hypertension of undetermined etiology	138	60	98	11.38	5	0.74	690	690	28	106
48	T. T.	43	M	Arteriovenous fistula, right thigh (80 sec. after fistula closed by pressure)	157	70	108	4.15	6	1.30	2,060	1,110	20	111
49	M. J.	46	F	Normal	120	68	80	5.90	5	0.72	1,080	1,480	25	108
50	H. C.	47	M	Rheumatic heart disease, mitral stenosis, pre-commisurotomy	107	50	75	3.76	8	1.43	1,590	910	12	91

TABLE I—*Continued*

Case	Initials	Age	Sex	Diagnosis	Ps	PP	P_{av-m}	CO	m	a	R†	K†	$\frac{P_i}{PP} \times 100$	$\frac{P_{av-c}}{P_{av-m}} \times 100$
no.					*mm. Hg*	*mm. Hg*	*mm. Hg*			*sec.*	*dyne-sec./cm.⁵*	*dyne/cm.⁵*		
51	H. C.	47	M	Rheumatic heart disease, mitral stenosis, post-commisurotomy	115	48	82	5.31	5	0.88	1,230	990	27	105
52	F. S.	49	M	Rheumatic heart disease, mitral stenosis	99	32	80	5.20	4	0.80	1,230	770	31	105
53	M. M.	49	F	Rheumatic heart disease, mitral insufficiency	137	53	110	4.30	4	0.51	2,040	2,450	26	99
54	G. C.	50	F	Pulmonary hypertension of undetermined etiology	185	100	125	3.99	4	0.87	2,500	2,910	34	105
55	G. W.	52	M	Rheumatic heart disease, mitral stenosis	108	40	82	3.65	5	0.94	1,800	920	13	114
56	H. W.	54	M	Arteriosclerotic heart disease, aortic insufficiency, cor pulmonale	160	102	90	3.66	5	0.72	1,960	3,410	27	115
57	O. B.	54	M	Rheumatic heart disease, mitral stenosis	134	68	92	3.95	4	0.86	1,860	2,010	31	104
58	E. C.	58	F	Interatrial septal defect	154	64	108	4.50 (L→R 6.40)	5	0.60	1,920	2,070	23	113
59	H. K.	58	M	Rheumatic heart disease, mitral stenosis	126	46	90	2.12	4	0.79	3,390	2,440	33	119

value of P_i might be considerably greater than for the resting catheterization subjects.

With this demonstration of the validity of the mathematical model, it appeared reasonable to pursue the practical implications further. Attempts to determine the cardiac output from the arterial pressure tracing alone were unsuccessful. The cardiac output could not be determined from the pressure tracing until the value of K or R was known. However, while the ratio K/R could be derived from the pressure tracing, the determination of individual values of K and R required use of the measured cardiac output. Thus, an insoluble situation existed.

The value of the resistance, R, could be directly derived from measurements of P_{av} and CO. The distribution of R in the present study was broad, with the standard deviation nearly equal to one-half the mean value. Only minimal correlation of R with the diagnosis existed. Thus, for example, the average value of R for the eight patients with patent ductus arteriosus, peripheral arteriovenous fistula or aortic insufficiency, which might be expected to be low, actually was 1,350 ± 560, quite close to the overall mean value. On the other hand, the highest values of R occurred in patients with no obvious aortic or peripheral pathology. Age, sex and body size had no consistent effect upon R. Its value was less a function of gross anatomical abnormalities than of the state of constriction of the arterioles throughout the body. Furthermore, the wide variation of R in different individuals in essentially identical physical surroundings indicated that R was of more value as an index of emotional than of physical adjustment to an environment. R would probably be more useful in evaluation of the circulatory changes within an individual in different circumstances than in comparison of one individual with another.

In contrast to the resistance, which could be calculated from readily measurable quantities, the direct determination of K would be impossible in the living body. This direct determination required measurement of changes of aortic pressure as a function of nonperiodic changes in volume. Hence, during life, K would have to be determined by indirect methods. The distribution of K in the present study was broad, with a standard deviation greater than one-half the mean value. K

TABLE II

Variation of the aortic elasticity with age

Age	No. in group	Average K	Standard deviation	Standard deviation of mean
yrs.				
10–19	13	1,410	820	228
20–29	18	1,390	420	99
30–39	7	1,830	920	346
40–49	15	1,170	540	140
50–58	6	2,290	780	318

showed no consistent change with age up to 58 years (Table II). The results in Table II were in remarkably close agreement with values found in direct measurement of the pressure-volume relationship in the aortas of post-traumatic human cadavers (8). The 47 postmortem aortas yielded an average change of 16 ± 6.5 ml. in aortic volume per $M.^2$ of body surface as an accompaniment of an increase in pressure from 80 to 110 mm. Hg. Since 1.78 was the mean value of the body surface area of the cadavers, K was equal, on the average, to 1,400 dyne per $cm.^5$—a mean value only 5 per cent less than, and statistically identical to, the average value of 1,470 (standard deviation of mean = 100) found in the present study, but distinctly different ($p < 0.01$) from the mean value of $1{,}023 \pm 482$ (standard deviation of mean = 58) found by Shock (9). The similarity of the values of K found in the work of Remington, Noback, Hamilton and Gold (8) and in the present study strongly suggested that the volume of the chamber in the mathematical model was essentially equivalent to the volume of the aorta. The deviation of the pressure in the distal aorta from the idealized pressure in the model as a result of the standing wave effect (10) apparently did not decrease the validity of the theoretical representation of the aorta by the chamber. In addition, Remington and his group showed that, up to approximately 115 mm. Hg, the value of K for aortas of individuals younger than 58 years showed no consistent pattern. However, above 58 years, the value of K increased. The similarity between the results found in cadavers and the results of the present study probably arose from the fact that the elasticity of the aorta, residing mainly in the inert elastic fibers, did not depend upon the viability of the body. The significance of K lay in its possible correlation with such factors as the degree of arteriosclerosis of the aorta.

The air chamber model gave evidence in the present study of present and potential usefulness. Its use led to an indirect method for the determination of K. With a known value of D, K and R could be determined by means of the arterial pressure tracing without measurement of P_{av}. If a measured value of D were unavailable, D could be roughly approximated from the arterial pressure tracing by assumption of a value for K. Furthermore, additional work would indicate whether or not the value of K remained reasonably constant in the same individual under various physiological circumstances. If it did remain reasonably constant, relative changes in R and D, though not the absolute values of these parameters, could be determined in a given individual from the arterial pressure tracing alone. Finally, the arterial pressure tracing might, under certain circumstances, be replaced by measurement of the blood pressure at the arm with a cuff. The auscultatory blood pressure would approximate the actual values of P_S and P_D in the brachial artery. From the pulse rate, the value of *a* could be determined, and tables of the average duration of diastole as a function of cardiac rate could be applied. An approximation to the value of K/R could be calculated from $\frac{\ln (P_S/P_D)}{\text{duration of diastole}}$. From this linear relationship, too, P_i could be easily approximated. Thus, approximate determination of relative changes in R and D in a given individual might, under certain conditions, be feasible merely from measurement of the blood pressure at the arm with a cuff.

SUMMARY

The recent development of a more exact mathematical approach to the air chamber model of the cardiovascular system suggested evaluation of the practical validity of the mathematical representation and, more importantly, of the usefulness of the model itself. The source of experimental data was 59 cardiac catheterizations of adults with cardiovascular abnormalities. The mathematical approach proved valid insofar as it could be tested, the 7 per cent difference between the average arterial pressure directly measured and that derived

by means of the theory from the arterial pressure tracing being within the range of reasonable experimental error. The usefulness of the air chamber was indicated by the similarity of the aortic elasticities determined from the model to values previously found in direct measurements upon the aortas of cadavers. This similarity opened new possibilities for future application of the model.

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