

EFFECT OF AORTIC INSUFFICIENCY ON ARTERIAL BLOOD PRESSURE, CORONARY BLOOD FLOW AND CARDIAC OXYGEN CONSUMPTION¹

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In patients with aortic insufficiency and without disease of the coronary ostia or arteries, the occurrence of chest pain having some or many of the characteristics of cardiac pain due to myocardial ischemia has been ascribed classically to a decrease in the coronary blood flow (1, 2); the aortic insufficiency presumably decreasing the coronary blood flow by lowering the mean aortic diastolic pressure. However, the data available on the effect of aortic insufficiency on the coronary blood flow are contradictory. From acute experiments on the anesthetized dog, Green (3), as well as Green and Gregg (4), concluded that when the aortic insufficiency is marked enough to lower the aortic diastolic pressure, the increase in coronary blood flow occurring during systole does not compensate for the decrease in flow during diastole and the mean coronary flow decreases. On the other hand, Foltz, Wendel, and West (5), from measurements made on anesthetized dogs in which one or two aortic cusps had been torn three or more days previously, concluded that the coronary blood flow was increased over levels usually observed in normal dogs while oxygen consumption was greatly increased over normal levels. Because of the discrepancy in the opinions about the effect of aortic insufficiency on the coronary blood flow, it was thought advisable to reinvestigate this problem.

METHODS

Eight dogs weighing between 17 and 40 kilograms were anesthetized by the intravenous administration of

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10 ml. per kilogram of weight of a 1 per cent chloralose solution. In the first four dogs the trachea, both common carotid arteries and both external jugular veins were exposed through a midline incision and a Y-shaped glass tube inserted into the trachea. Under artificial respiration, the left hemithorax was opened, the pericardium incised and the heart suspended in a pericardial cradle. The method used to measure and continuously record the output of the left ventricle was a modification of that previously described in this laboratory (6). The proximal ends of the left subclavian artery and brachiocephalic trunk were cannulated. Then the aorta was clamped distally to the subclavian artery so that the blood ejected by the left ventricle was directed into an electromagnetic rotameter (7) and returned to the circulation via both carotid arteries cannulated distally as well as both femoral arteries cannulated proximally and distally. Since the coronary sinus drains blood essentially only from the left coronary artery (8, 9) and since, in a given animal, the coronary sinus blood flow represents a constant portion of the left coronary artery flow (8), measurement of the coronary sinus blood flow was used as a means of studying the left coronary artery blood flow. For that purpose, a cannula was introduced via the left external jugular vein into the coronary sinus and tied in place by a string passed around the coronary sinus. All the blood emerging from the coronary sinus through this cannula was directed into a second rotameter by means of which the coronary sinus blood flow was measured and continuously recorded. The coronary sinus blood was returned to the dog through a cannula introduced via the right external jugular vein down to the junction of the right auricle and the superior vena cava. Mean arterial blood pressure was recorded from the root of the aorta with a dampened Gregg manometer. Left ventricular output, mean aortic blood pressure and coronary sinus blood flow were continuously recorded on photographic paper. The heart rate was obtained by direct timing, from the blood pressure tracing or from an electrocardiographic tracing. In the other four dogs the output of the left ventricle was not measured and the coronary sinus flow was measured as in the first four dogs. The mean femoral arterial blood pressure was recorded with a dampened Gregg manometer and the aortic pressure curve was recorded from the root of the aorta with another optical manometer. The instrument used to produce acute aortic insufficiency was the um-

TABLE I

Effect of aortic insufficiency on heart rate, arterial blood pressure, cardiac output, coronary blood flow, coronary blood arteriovenous oxygen difference and cardiac oxygen consumption

Exp. and dog no.	Duration of insufficiency	Heart rate beat	Arterial blood pressure			Cardiac output	Coronary flow	Coronary A-V O ₂ difference	Oxygen consumption
			Mean	Syst.	Diast.				
	sec.	min.	mm. Hg			ml./min.	ml./min.	vol. %	ml./min.
			Aortic						
1. C.*		136	150			1,220	59	15.5	9.2
A.I.†	235	160	135			830	80	15.4	12.4
R.‡		152	140			1,000	57	15.9	9.1
2. C.		160	130			1,100	94	12.7	11.8
A.I.	316	160	110			960	113	13.4	15.1
R.		160	128			1,060	87	12.5	10.9
3. C.		167	97			810	49	15.8	7.7
A.I.	340	167	76			600	55	15.6	8.6
R.		176	94			675	50	15.5	7.7
4. C.		166	119			940	65	12.2	7.9
A.I.	214	170	105			940	78	12.7	9.9
R.		170	120			1,050	74	12.8	9.5
			Femoral						
5. C.		160	120				38	17.3	6.6
A.I.	304	162	98				45	17.4	7.8
R.		162	110				40	17.9	7.2
			Femoral						
			Aortic						
A C ₁ §		144	132	157	116		68	16.2	11.0
A.I.‖	208	150	125	150	98		76	16.1	12.0
R ₁		146	132	148	114		68	16.2	11.0
6. C ₂ ¶		148	128	154	115		67		
A.I. ₂	80	148	122	144	88		79		
A.I. ₃	155	150	117	145	80		80		
A.I. ₄	210	150	118	148	76		85		
A.I. ₅	277	150	108	141	63		87		
R ₂		150	128	156	115		67		
A C ₁		183	128	140	105		61	14.9	9.1
A.I. ₁	244	182	105	140	48		70	16.4	11.5
R ₁		185	128	140	105		61	14.9	9.1
7. C ₂		186	122	140	105		57		
A.I. ₂	85	188	104	142	52		72		
A.I. ₃	243	188	104	145	64		72		
A.I. ₄	348	188	104	142	64		70		
R ₂		188	122	140	112		61		
A C ₁		112	160	183	132		65	21.1	13.7
A.I. ₁	126	140	128	154	55		83	21.5	17.8
R ₁		112	160	178	134		74	21.3	15.8
8. C ₂		112	158	182	125		62		
A.I. ₂	30	120	139	172	86		81		
A.I. ₃	83	122	156	172	95		72		
A.I. ₄	130	132	138	176	96		77		
A.I. ₅	204	138	139	185	70		86		
A.I. ₆	251	138	130	164	42		89		
A.I. ₇	288	140	126	165	38		91		
R ₂		138	160	180	120		68		

* C., control.

† A.I., aortic insufficiency.

‡ R., recovery.

§ C₁, control for part A.‖ A.I._{1, 2, 3, etc.}, aortic insufficiency of severity increasing or decreasing stepwise.¶ C₂, control for part B.

brella type valve spreader previously described by Wiggers and Maltby (10). While blood pressure, coronary sinus blood flow and in some experiments left ventricular output were being continuously recorded, the valve spreader was slipped into the aortic ring through the left ventricular wall. The introduction of the instrument into the aortic ring was without effect on blood pressure, left ventricular output and coronary sinus flow. Proper positioning of the instrument was judged by palpation and the correctness of the position of the instrument was checked by direct visualization at the end of each experiment.

In all eight experiments, after a control period an aortic insufficiency severe enough to lower definitely the mean and/or diastolic arterial blood pressure was induced and maintained from 126 to 340 seconds, after which a period of recovery was observed. Arterial and coronary sinus blood samples were drawn simultaneously at the end of the control period, the period of aortic insufficiency and during the recovery period. The oxygen content of the arterial and venous bloods was determined in duplicate by the method of Van Slyke and Neill (11). The oxygen consumption of that portion of the myocardium drained by the coronary sinus was calculated according to the following formula:

Oxygen consumption (expressed in ml. per minute) equals coronary sinus blood flow (expressed in ml. per minute) times coronary oxygen arteriovenous difference (expressed in ml. per cent) times 10^{-3} .

In three of the eight experiments, after the completion of that part of the experiment in which the effect of aortic insufficiency on oxygen consumption of the myocardium, arterial blood pressure and coronary blood flow was studied, an attempt was made to induce aortic insufficiency, then to increase (or decrease) the severity of the aortic insufficiency stepwise in order to establish the effect of aortic insufficiency of progressively increasing (or decreasing) severity on arterial blood pressure and coronary blood flow.

RESULTS AND DISCUSSION

The results observed are summarized in Table I and Figure 1. The effect of aortic insufficiency on coronary blood flow and cardiac oxygen consumption was studied in eight experiments on eight dogs. In all eight experiments, aortic insufficiency lowered the mean arterial blood pressure, the decrease ranging from 7 to 32 mm. of mercury, and despite the sudden drop in mean arterial pressure the coronary flow rose in all eight experiments, the increase amounting from 12 to 36 per cent. The oxygen consumption rose in all eight experiments and this rise amounted from 9 to 35 per cent. In three experiments, this increase in oxygen consumption was due to the increase in coronary flow alone, the coronary blood oxygen ar-

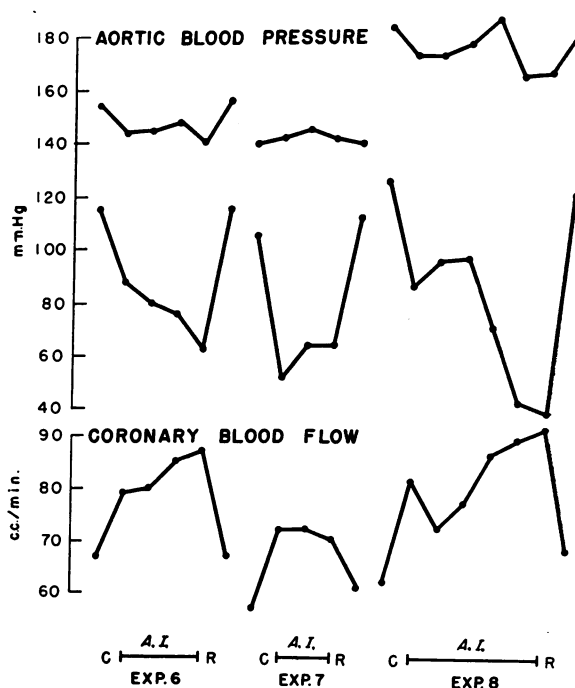


FIG. 1. PLOT OF PART B OF EXPERIMENTS 6, 7 AND 8, ILLUSTRATING THE EFFECT OF ACUTE AORTIC INSUFFICIENCY OF SEVERITY INCREASING (OR DECREASING) STEPWISE

Upper tracings, systolic and diastolic aortic blood pressure; scale in mm. of mercury. Lower tracing, coronary sinus blood flow; scale in ml. per minute.

C, control; A.I., aortic insufficiency of severity increasing (or decreasing) stepwise; R, recovery.

teriovenous difference remaining unchanged. In the other five experiments, most of the increase in oxygen consumption was contributed by the increase in coronary flow, although the coronary blood oxygen arteriovenous difference increased somewhat. The output of the left ventricle, *i.e.*, the effective cardiac output, was decreased in three of the four experiments in which it was measured and unchanged in one experiment. The heart rate was not at all or only slightly affected by the aortic insufficiency, except in two experiments in which it rose significantly. These latter two experiments were otherwise similar to the rest of the experiments. In Part A of Experiments 6, 7 and 8, in which the aortic pressure curve as well as the mean arterial femoral pressure was recorded, the mean arterial blood pressure was observed to fall from 132, 128 and 160 mm. of mercury to 125, 105 and 128 mm. of mercury, re-

spectively, and the diastolic pressure in the same experiments fell from 116, 105 and 132 to 98, 48 and 55 mm. of mercury, respectively. In Part B of Experiments 6, 7 and 8, illustrated in Figure 1, the stepwise *increase* in the severity of the aortic insufficiency resulted in a stepwise *decrease* in the mean arterial blood pressure as well as aortic diastolic pressure, and in a stepwise *increase* in the coronary flow. Part B of these three experiments shows that although the mean arterial blood pressure fell from 128, 122 and 158 mm. of mercury to minima of 108, 104 and 126 mm. of mercury, respectively, and although the diastolic pressure fell from 115, 105 and 125 to minima of 63, 52 and 38 mm. of mercury, respectively, the coronary flow rose stepwise to values 28, 15 and 47 per cent above control level, respectively. From these data there seems to be little doubt that acute aortic insufficiency increases the cardiac oxygen consumption and the coronary blood flow, even when the aortic insufficiency is severe enough to produce a marked decrease in mean arterial blood pressure and aortic diastolic pressure. As a matter of fact, within the limits of the present experiments, the more severe the aortic insufficiency was and the lower the mean arterial and aortic diastolic pressure fell, the more the coronary blood flow increased, as demonstrated by the second part of Experiments 6, 7 and 8.

These results are in disagreement with those of Green (3) and Green and Gregg (4), who, in acute experiments on anesthetized dogs, found that aortic insufficiency always decreased the coronary blood flow. Because partial occlusion of the aorta during aortic insufficiency could raise the coronary blood flow to or above control level by raising the mean aortic pressure, these authors postulated the possibility that, if aortic insufficiency induced sufficient peripheral vasoconstriction to restore the mean arterial pressure to its control level, aortic insufficiency could raise the coronary blood flow. However, they never actually observed such an occurrence, and in their experiments aortic insufficiency alone always lowered the coronary flow. It must be pointed out that the control arterial blood pressure of their animals was quite low and a decrease in the head of pressure at those very low levels of aortic pressure may have more of an effect on the coronary flow than the same decrease in the head of pressure at

higher levels of aortic pressure. However, this would hardly seem to be the only factor responsible for the discrepancy between Green and Gregg's observations and the present study. Indeed, it should be kept in mind that the condition of dogs with as low a control arterial blood pressure as reported in the observations published by Green and Gregg is such that these dogs most probably respond to the induction of aortic insufficiency differently from dogs with a higher and more normal control arterial blood pressure as well as cardiovascular system in general. On the other hand, Foltz, Wendel, and West (5) observed that during aortic insufficiency "coronary blood flow was increased over levels usually observed in normal dogs, while oxygen consumption was greatly increased over normal values." Unfortunately, no control values for the blood pressure were given by these authors and it was therefore difficult to estimate the severity of the aortic insufficiency. Furthermore, the effect of the aortic insufficiency was appraised by comparison of the values of coronary blood flow found in a group of normal dogs with the values observed in the group of dogs with aortic insufficiency. Hence, no strict control values for coronary blood flow were available. The observations made on patients with aortic insufficiency are sparse and variable in their conclusions. Bing and his co-workers (12) reported coronary blood flow and cardiac oxygen consumption to be definitely increased in one case of aortic insufficiency as compared to a group of normal persons. Regan, Talmers, Christensen, Wada, and Hellems (13) reported that in three patients with severe aortic insufficiency the coronary flow was 91, 100 and 107 ml. per 100 grams of left ventricle per minute, and the oxygen consumption averaged 12.7 ml. per 100 grams of left ventricle per minute. In two patients with aortic insufficiency and angina pectoris the coronary blood flow was 66 and 68 ml., and the oxygen consumption 8 ml. per 100 grams of left ventricle per minute. These data were compared with data obtained on a group of 8 normal persons in whom the coronary blood flow was 103 plus or minus 20 ml. per 100 grams of left ventricle per minute, and the oxygen consumption was 10.5 plus or minus 2.6 ml. per 100 grams of left ventricle per minute. Such data are difficult to assess and hazardous to interpret

because of the small number of observations, the lack of adequate control data and the impossibility of ever ruling out the presence of complicating factors such as diseases of the coronary ostia and/or arteries.

In conclusion, it would seem that an acute aortic insufficiency severe enough to lower markedly the mean arterial blood pressure, as well as the aortic diastolic pressure, resulted in an increase in cardiac oxygen consumption and coronary blood flow. This increase in coronary flow must be due to a decrease in the resistance of the coronary bed, since the coronary flow rose despite a marked drop of the aortic pressure. This decrease in the resistance of the coronary bed was probably induced by the increase in the work of the left ventricle (14). Whether the increase in coronary flow was sufficient to meet the demands made upon the left ventricle by the aortic insufficiency, and thereby prevent myocardial ischemia, cannot be determined from these experiments.

SUMMARY AND CONCLUSIONS

In the anesthetized dog acute aortic insufficiency, sufficient to lower markedly the mean arterial blood pressure and the aortic diastolic pressure, resulted regularly in an increase in coronary sinus blood flow and myocardial oxygen consumption.

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