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SOME EFFECTS OF POSITIVE PRESSURE RESPIRATION DURING ANESTHESIA *

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POSITIVE pressure respiration has been used in inhalation anesthesia (1) and in the treatment of pulmonary edema (2). The influence of this procedure on intravascular pressures (2–6) and on cardiac output (7–10) has been described, but the pressures used were generally high and applied for a short time. It appeared desirable to study the influence of lower pressure applied for a longer time. Since it seemed possible that the increased venous pressure and reduced blood flow accompanying positive pressure respiration might lead to a reduction in plasma volume, this has also been investigated.

EXPERIMENTS

Twenty experiments were done on dogs anesthetized with ether long enough to permit the intravenous injection of sodium barbital, 250 mg. per kilogram of body weight. A tracheal cannula was inserted and connected either with a spirometer of the Benedict-Roth type or with a chamber through which air was passed. The pressure was increased in the spirometer by adding a weight to the moving bell; the pressure in the chamber was increased by letting the air escape from the chamber through a water valve. In some of the experiments of the latter type, the dog breathed in and out of this chamber; in the remainder, the dog inspired from the chamber and expired through a water valve, set at the desired pressure. The pressure used in all experiments was 7 mm. of mercury. Nine experiments with determination of cardiac output by the Fick procedure were done. In eight of these, oxygen consumption was measured with the spirometer; in one experiment the expired air was collected and analyzed, its volume measured, and the oxygen consumption calculated.

Blood for analysis of oxygen and carbon dioxide contents was drawn without exposure to air over mercury (11) and analyzed, usually in duplicate, by the manometric method of Van Slyke and Neill (12). Mixed venous blood was taken from the right auricle by cannula, or from the right ventricle by cannula or needle puncture. The cannulas were introduced by way of the right external jugular vein.

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TABLE 1

Exp. No.	Weight of Dog, Kg.	Time of Observation,† minutes	Source of Venous Blood	Oxygen Content cc. per 100 cc.		A–V Oxygen Difference, cc. per 100 cc.	Oxygen Consumption, cc. per min.	Cardiac Output, l per min.	Arterial Pressure, mm. Hg	Venous Pressure, cm. H_2O	Total Peripheral Resistance‡	Pulse Rate per min.	Hematocrit, per cent red cells
				Arterial	Venous								
1		Control	auricle,	21.49	17.81	3.68	111.4	3.03				206	
		+ P 180	cannula	23.03	16.92	6.11	131.2	2.15				163	
2		Control	auricle,	13.91	9.49	4.45	75.1	1.66					32.6
		+ P 180	cannula	14.88	6.52	8.36	69.7	0.83					30.3
3		Control	auricle,	19.53	17.95	1.58	82.2	5.20	116		1.34	140	44.8
		+ P 180	cannula	20.76	15.17	5.59	120.0	2.15	138		3.84	200	45.6
4		Control	auricle,	22.12	20.50	1.62	88.4	5.46	108	6.0	1.18		49.5
		+ P 180	cannula	23.22	19.29	3.93	108.7	2.74	110	8.9	2.41		43.7
5	20.5	Control 210	ventricle,	16.06	11.50	4.56	116.6	2.56	120		2.82	156	34.4
		+ P 180	needle	16.17	10.01	6.16	114.4	1.86	94	9.1–9.4	3.03	156	36.0
		Control 30		16.41	11.11	5.30	131.2	2.48	103	5.8–6.0	2.49	162	36.4
6	14.9	Control 240	ventricle,	20.73	16.78	3.95	77.2	1.95	150		4.61	150	44.1
		+ P 180	needle	25.23	17.08	8.15	76.2	0.93	115	12.6–12.9	7.43	174	54.1
		Control 44		25.14	18.06	6.08	76.6	1.26	122	7.8–7.9	5.80	156	52.7
7*	12.4	Control 210	ventricle,	23.99	20.70	3.29	52.9	1.61					
		+ P 58	cannula	23.51	19.30	4.21	58.1	1.38					
		Control 50		23.67	18.30	5.37	50.8	0.95					
8	17.8	Control 240	ventricle,	28.12	20.60	7.52	128	1.70	170		6.00	96	
		+ P 18	cannula	27.89	19.95	7.94	145	1.82	160		5.28	102	
		Control 15		27.73	19.08	8.65	228	2.63	150		3.42	108	
9	15.2	Control 180	ventricle,	25.48			86.4		144	6.9		129	
		+ P 9	needle	25.96	20.19	5.77	80.0	1.39	146	7.7–7.9	6.30	129	
		Control 27		25.44	21.62	3.82	85.0	3.82	150	7.2–7.3	2.36	150	

* This experiment with air chamber; all others with spirometer.
† Time of first control after induction of anesthesia; duration of positive pressure; time after return to atmospheric pressure.
‡ Total peripheral resistance = arterial pressure mm. Hg/cardiac output cc. per sec.

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Arterial blood was obtained by cannulation of a branch of a femoral artery. Arterial pressure was recorded by a mercury manometer connected with a carotid artery, and pulse rate was counted on its tracing. Venous pressure was measured with a saline manometer and needle puncture of a femoral vein, zero pressure referring to the level of the vein, which was at a point below the right auricle. The hematocrit determination was made in Wintrobe tubes. The results of these experiments appear in table 1.

Plasma volume was determined by a modification of the method of Gibson and Evans (13) before and after three hours of positive pressure respiration, in eleven experiments, seven with the air chamber, four with the spirometer. The plasma volume was measured with T-1824, of which 20 mg. was injected for the first determination, and 10 mg. for the second. As a rule, five blood samples were taken for each determination, the first twenty minutes after injection, the remainder at ten minute intervals. The dye concentration of the plasma was measured with a Klett-Summerson photocomparator. The theoretical dye concentration at the time of injection was obtained by extrapolation of a time-log concentration curve. Hematocrit values and venous pressure were determined as in the other experiments. The results of these experiments appear in table 2.

TABLE 2

Exp. No.	Weight of Dog, Kg.	Method	Plasma Volume, cubic centimeters		Percentage Change	Hematocrit, per cent cells		Venous Pressure, cm. H_2O	
			Control	+ P 3 hr.		Control	3 hr.	Control	3 hr.
10	11.5		361	378	+4.7				
11	29.2		1340	1355	+1.1				
12	21.6	Chamber,	758	811	+7.0	41.3	53.3		
13	18.4	air	893	847	−6.7	36.3	43.2		
14	17.0		749	680	−9.2	56.1	55.1		
15	18.4		794	826	+4.0	55.0	53.0		
16	15.6		743	719	−3.2	48.5	45.6	9.5–9.7	13.5–13.9
17	13.0		542	585	+7.9	41.9	43.0		
18	12.5	Spirometer,	583	575	−1.4	51.0	44.9		
19		oxygen	690	690	0.0	49.0	46.1	8.9	12.0–12.2
20	11.7		468	446	−4.7	49.8	50.8	8.2–8.4	12.0–13.0

In four of the experiments on cardiac output, respiratory rate and volumes were measured, and blood carbon dioxide content determined. In two of the experiments on plasma volume, respiratory rate and volumes were measured. These results appear in table 3.

DISCUSSION OF RESULTS

When the dogs were made to respire at a pressure 7 mm. of mercury above atmospheric, a rise in venous pressure, a transient fall in arterial

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TABLE 3

Exp. No.	Observation and Time in minutes	Respiration			Blood Carbon Dioxide, cc. per 100 cc.	
		Rate per min.	Volume, 1 per min.	Tidal Air, cc.	Arterial	Venous
5	Control	33.5	6.41	191	50.08	54.63
	+ P 180	24.5	10.53	426	41.99	49.00
	Control	38.5	16.40	425	45.51	50.38
6	Control	6.5	1.35	208	45.83	48.64
	+ P 180	38.0	4.56	120	36.58	45.25
	Control	47.5	4.51	99	36.56	45.00
7	Control	45.0	5.40	120	37.84	38.93
	+ P 58	52.0	5.84	112	40.68	43.15
	Control	74.0	8.75	118	37.92	42.30
9	Control	19.5	1.70	142	45.96	
	+ P 9	12.5	2.13	185	43.96	48.07
	Control	20.3	3.10	153	38.95	44.28
14	Control	40.0	6.03	151		
	Control	60.0	6.49	108		
	+ P 60	11.0	3.12	284		
	+ P 120	18.0	6.78	377		
	+ P 180	12.3	5.72	465		
	Control	14.0	7.54	539		
15	Control	37.0	6.70	181		
	Control	28.0	6.41	229		
	+ P 60	19.0	5.46	288		
	+ P 180	100.0	13.0	133		

pressure, and frequently a short period of apnea, were seen, in confirmation of others (3–7). Of the two dogs (Nos. 8, 9) in which cardiac output was determined a few minutes after application of the positive pressure, one showed a decrease of 64 per cent, the other a questionable decrease. The dog in which cardiac output was measured after fifty-eight minutes of positive pressure respiration showed no decrease in output. At the end of three hours of positive pressure respiration, six dogs showed an average decrease of 41 per cent in cardiac output.

Experiments 1 to 4 can be criticized on the basis that the venous blood was taken from the right auricle, which we have shown (14) may not give a representative sample, and on the basis that an inadequate time for equilibration following induction of anesthesia was allowed (14). The remaining experiments are free from these criticisms.

It appears that respiration at this pressure may or may not reduce cardiac output within one hour. Beecher *et al.* (15) have reported a reduction in blood flow in the carotid, femoral, and mesenteric arteries of anesthetized dogs on the application of approximately this same pressure. A reduction of 33 per cent in cardiac output after

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thirty minutes of breathing against an expiratory resistance of 5 cm. of water (3.7 mm. of mercury) was seen in urethanized cats by Huggett (8). In barbitalized dogs, Holt (7) saw a reduction of 33 per cent in output after fifteen minutes' respiration at 16 cm. of water (11.8 mm. of mercury), and a reduction of 61 per cent in output at 20–30 mm. of mercury was found by Humphreys *et al.* (10). In all of these experiments, control observations were made both before and after the period of positive pressure respiration.

It seems likely that the reduction in cardiac output results from interference with filling of the right heart. Beck and Isaac (17) observed such a decrease when pressure was applied only in the pericardium, and Moore *et al.* (18) saw no change in cardiac output on ligation of one pulmonary artery.

The average value for plasma volume was unaltered by the positive pressure respiration. A decrease in plasma volume might be expected from the tendency of the increased venous pressure to raise capillary pressure, while the reduced blood flow with constant arterial pressure would tend to reduce capillary pressure and increase the plasma volume. Landis and Gibbon (16) found that, in the human arm, venous pressure must be elevated to a value above 12–15 cm. of water to produce a net loss of fluid from the circulation.

In only three of the fourteen experiments in which it was measured was there an increase in hematocrit cell percentage. This is presumably a result of expulsion of cells from reservoirs, but it is not clear why it happened in only these experiments.

Concerning the observations on respiration, conclusions as to the influence of positive pressure are difficult to draw because of the instability of the animals. There appears to have been a spontaneous progressive increase in ventilation, with a resulting decrease in arterial carbon dioxide content.

SUMMARY

Respiration at a pressure of 7 mm. of mercury above atmospheric pressure for three hours leads to a reduction of cardiac output by 41 per cent in dogs under barbital anesthesia. Respiration at this pressure for one hour or less may or may not lead to a reduction in cardiac output.

Venous pressure is greater than normal, but arterial pressure, except for a transient fall, is unaltered with respiration at this pressure. Total peripheral resistance is increased, pulse rate is unchanged.

Plasma volume is unchanged after three hours' respiration at this pressure. The hematocrit cell percentage is unchanged, except for an increase in a few experiments.

Respiratory volume, oxygen consumption, and blood carbon dioxide content show no changes attributable to the positive pressure respiration.

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