On the Changes in the Minute Volume and the Stroke Volume of the Heart following the Respiration of Compressed Air.

By

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Various changes in organisms provoked by the respiration of compressed air have already been investigated by some experimentators.

In the course of an experiment with the respiration of compressed air, Sommerbrodt¹⁾ observed a decrease of blood pressure, and attributed this phenomenon to the lessening of the volume of blood in the arterial system, on account of the disturbance in circulation from the venous system to the arterial side which was aroused by the compression of capillaries in the lungs due to the elevated gas pressure. Tiegel²⁾ has found that during exposure at an excessive pressure the blood pressure in the endothoracic veins is elevated, a stasis takes place in the peripheral veins and the blood-flow into the endothoracic veins is disturbed, in consequence of the excessive pressure in the thoracic cavity. On observing also that the blood pressure is augmented in the pulmonary arteries, he attributed this occurrence to the extensive resistance in the blood-flow, which is induced by the compression of the lung capillaries due to the high pressure in the alveoli.

Recently, Huggett³⁾ investigated the heart output during respiratory obstruction. He used urethanised cats for the experiments. The obstruction used was threefold:—namely, simple inspiratory, simple expiratory and combined inspiratory and expiratory obstruction. Into the trachea a cannula of Y-shape was inserted, one limb of which was connected by wide rubber tubing to a wash-bottle containing water so that the air which was to be breathed in passed through the bottle. The pressure was regulated by the depth of the inflow tube below the water. A similar but reversed bottle gave the expiratory resistance. In the case of an inspiratory obstruction of 5 cm. of water, the animal would have to lower the pressure inside the bottle to 5 cm. below the atmospheric pressure by expanding the chest, in order that air might pass down the entry tube of the bottle. Similarly, in the reversed (expiratory) bottle the animal would have to create positive pressure in order to drive the air out, thus giving rise to a forced expiration. Under such conditions the mi-

1) Sommerbrodt, cited by Full and Friedrich. *Klin. Wochenschr.*, 1923, 69.

2) Tiegel, *Brun's Beiträge z. klin. Chirurg.*, 1911, 76, 160.

3) Huggett, *Journ. Physiol.*, 1924, 59, 373.

minute volume and the stroke volume were measured by Fick's formula. Inspiratory obstruction increased the minute volume by 37.5 p.c. and the stroke volume by 43 p.c. Expiratory obstruction decreased both minute volume and the stroke volume by 33 p.c. Combined inspiratory and expiratory obstruction caused a decrease in the minute volume by 31 p.c. and in the stroke volume by 23.5 p.c.

Moteki⁴⁾ of our Laboratory using rabbits investigated the changes in respiration, pulse rate, blood gases, blood elements and total blood volume, due to the respiration of air under an excess of pressure, which amounted to from 10 to 15 cm. of water. He summarized his experimental results as follows: The oxygen unsaturation of arterial blood decreased markedly by the inhalation of the compressed air for one hour, and increased thereafter without reaching the initial value. After the cessation of the inhalation it showed in general a tendency to decrease a little again. The oxygen capacity of the blood was already at the end of the first hour of the inhalation markedly diminished, and as a rule further decrease was observed still after the decompression, with a few exceptions when there was an increase. The content of carbon dioxide in the blood indicated also a decrease during the respiration of compressed air in the majority of cases, and decreased after the cessation of it more and more. The haemoglobin and serum protein showed a remarkable decrease during the exposure at excess pressure, and a tendency to recover after the decompression; but serum sodium chloride showed a slight decrease one hour after the exposure, and an increase two hours after it, and this tendency to increase continued even after the cessation of the exposure. The total blood volume increased remarkably during the inhalation of compressed air, and recovered after returning to normal respiration. During the respiration of compressed air, the blood pressure and respiration rate fell and the pulse rate increased, but recovered when respiration was resumed under normal conditions.

In the present investigation I have undertaken to observe the effects of the respiration of compressed air on the minute volume and the stroke volume of the heart.

Method of Experiment:—The animals used for the experiments were rabbits. Without anaesthetics the animal was fixed in the dorsal position on an animal holder, which was suitably warmed. The cannulae were inserted into the trachea and the femoral artery, and the skin was resected from the cardiac area. The experiment was not begun until the respiration became quiet, after the animal had been lying still for one hour. The excess pressure was made by putting a weight of 3 kilogrammes on the bell of Sanborn-Benedict's respiration apparatus with which a water manometer was connected, indicating the pressure to be higher by about 14 cm. of water than the atmospheric pressure. The determination of the minute volume was carried out by Fick's⁵⁾ principle. The oxygen consumption was measured with Sanborn-Benedict's respiration apparatus. The arterial blood was drawn from the femoral artery. The mixed venous

4) Moteki, Nihon-Naika-Gakkai-Zasshi, 1925, 13, 121.

5) Fick, Verhand. d. physik. med. Gesellsch. in Würzburg, 1872, 2, 16, cited by Lundsgaard, Deutsch. Arch. f. kl. Med., 1916, 118, 36.

blood was taken from the right ventricle by cardiac puncture through the thorax wall. The 2 c.c. syringe used for the purpose was graduated exactly, containing 3.5 p.c. sodium citrate in the dead space of the barrel and the needle, which was 5.5 cm. long and of 0.5 mm. calibre. The blood obtained was immediately transferred to a Barcroft's bottle and the arteriovenous difference of oxygen contents was measured. These measurements were made five times, i. e. before the respiration of compressed air, one and two hours after the exposure at excess pressure and one and two hours after the cessation of the pressure. All care was taken to keep the animals quiet during the experiments, because the disquiet of the animals changes the gaseous metabolism and the circulation.

RESULTS OF THE EXPERIMENTS.

Eleven experiments were made, the results of which are shown in Table I.

TABLE I.

Changes in the Heart Output by Respiration of Compressed Air.

No.	Body weight (kg.) & sex	Time of observation	Respiration per min.	Pulse rate per min.	Oxygen con- sumption per min. (c.c.)	Arteriovenous difference of oxygen contents (c.c.)	Minute volume		Stroke volume	
							c.c.	p.c. diff.	c.c.	p.c. diff.
1	1.87 ♂	Before compression	48	270	29.89	0.09900	302		1.12	
		1 hr. after compression	48	252	18.94	0.10427	182	-40	0.72	-36
		2 hrs. after compression	46	240	18.94	0.11129	170	-44	0.70	-38
		1 hr. after decompression	44	240	22.41	0.10356	216	-28	0.90	-20
		2 hrs. after decompression	44	270	23.35	0.08566	273	-10	1.01	-10
2	2.12 ♀	Before compression	62	222	27.51	0.06045	455		2.05	
		1 hr. after compression	56	216	30.27	0.10906	278	-31	1.29	-42
		2 hrs. after compression	56	218	28.35	0.08597	330	-27	1.51	-26
		1 hr. after decompression	64	210	20.48	0.04678	438	-4	2.09	+2
		2 hrs. after decompression	66	216	20.50	0.04475	458	+1	2.12	+3
3	1.77 ♀	Before compression	88	258	21.72	0.07858	276		1.07	
		1 hr. after compression	95	270	22.10	0.11408	194	-30	0.72	-33
		2 hrs. after compression								
		1 hr. after decompression	83	198	17.70	0.06992	253	-8	1.28	+20
		2 hrs. after decompression	116	270	17.67	0.07068	250	-10	0.93	-13
4	2.40 ♂	Before compression	70	288	25.68	0.08789	292		1.01	
		1 hr. after compression	88	288	19.49	0.09744	200	-32	0.69	-32
		2 hrs. after compression	74	258	19.54	0.10715	182	-37	0.71	-30
		1 hr. after decompression	72	282	27.84	0.11337	246	-16	0.87	-14
		2 hrs. after decompression	80	270	22.85	0.08423	271	-7	1.00	-1

No.	Body weight (kg.) & sex	Time of observation	Respiration per min.	Pulse rate per min.	Oxygen con- sumption per min. (c.c.)	Arteriovenous difference of oxygen contents (c.c.)	Minute volume		Stroke volume	
							c.c.	p.c. diff.	c.c.	p.c. diff.
5	2.40 ♀	Before compression	48	246	20.55	0.08254	249		1.01	
		1 hr. after compression	58	258	20.83	0.08958	233	- 6	0.90	-11
		2 hrs. after compression	56	270	21.78	0.08223	265	+ 6	0.98	- 3
		1 hr. after decompression	58	246	21.51	0.06929	310	+25	1.26	+25
		2 hrs. after decompression	54	258	24.36	0.07478	326	+31	1.26	+25
6	2.15 ♀	Before compression	120	228	28.47	0.11629	246		1.08	
		1 hr. after compression	62	216	21.25	0.13569	157	-36	0.73	-32
		2 hrs. after compression								
		1 hr. after decompression	76	228	25.65	0.09468	271	+10	1.19	+10
		2 hrs. after decompression	74	228	21.71	0.09550	227	- 8	1.00	- 7
7	2.30 ♀	Before compression	183	246	23.69	0.12132	195		0.79	
		1 hr. after compression	95	216	18.57	0.09646	193	- 1	0.89	+13
		2 hrs. after compression	90	216	18.55	0.09836	189	- 3	0.87	+10
		1 hr. after decompression	111	240	17.36	0.09923	175	-10	0.73	- 8
		2 hrs. after decompression	88	240	17.44	0.07789	224	+15	0.91	+15
8	1.95 ♀	Before compression	47	267	21.35	0.06073	352		1.32	
		1 hr. after compression	53	246	12.37	0.12924	96	-73	0.39	-70
		2 hrs. after compression	63	243	12.06	0.12560	96	-73	0.40	-70
		1 hr. after decompression	49	276	10.96	0.08085	136	-61	0.49	-63
		2 hrs. after decompression	61	264	13.26	0.10136	132	-63	0.50	-62
9	2.16 ♀	Before compression	40	180	24.21	0.06391	379		2.11	
		1 hr. after compression	56	204	19.80	0.08761	227	-40	1.11	-47
		2 hrs. after compression	52	210	16.97	0.08892	191	-50	0.91	-57
		1 hr. after decompression	40	156	17.56	0.07893	222	-41	1.42	-33
		2 hrs. after decompression	40	180	16.72	0.05461	306	-20	1.70	-19
10	2.26 ♀	Before compression	72	228	26.38	0.06471	408		1.83	
		1 hr. after compression	60	237	24.80	0.07695	322	-21	1.36	-26
		2 hrs. after compression	68	228	19.08	0.05813	328	-20	1.44	-21
		1 hr. after decompression	108	225	21.55	0.07497	287	-30	1.28	-30
		2 hrs. after decompression	151	222	23.39	0.06827	343	-16	1.55	-15
11	2.06 ♀	Before compression	45	234	18.66	0.08476	220		0.94	
		1 hr. after compression	49	264	18.27	0.10526	174	-21	0.66	-30
		2 hrs. after compression	48	267	17.65	0.10762	164	-25	0.61	-35
		1 hr. after decompression	50	246	17.81	0.10464	170	-23	0.69	-27
		2 hrs. after decompression								
Average		Before compression	75	242	23.92	0.08365	307		1.30	
		1 hr. after compression	65	242	20.61	0.10415	205	-33	0.86	-34
		2 hrs. after compression	61	235	19.21	0.09614	213	-31	0.90	-31
		1 hr. after decompression	69	243	20.08	0.08511	248	-20	1.11	-15
		2 hrs. after decompression	77	242	20.14	0.08579	281	- 8	1.20	- 8

Fig. 1.

Experiment No. 1, Table I.

Before the breathing of compressed air the minute volume amounted to from 455 to 195 c.c. with an average of 307 c.c. Being exposed for one hour at the excess pressure, it decreased markedly, amounting to from 322 to 96 c.c., with an average of 205 c.c., in two hours to from 330 to 96 c.c. on an average to 213 c.c. After the cessation of the exposure at the excess pressure it increased gradually:—namely one hour after the cessation it was raised to from 438 to 136 c.c. with an average of 248 c.c., two hours thereafter returning to from 458 to 132 c.c. with an average of 281 c.c. In brief, it can be clearly seen that the minute volume which has decreased during the respiration of compressed air begins to increase after the cessation of this respiration, but does not yet reach the former value.

The stroke volume also showed a tendency resembling that of the minute volume. Before the compressed air was inhaled, the stroke volume amounted to from 2.11 to 0.79 c.c. with an average of 1.30 c.c., but during the respiration of compressed air it decreased markedly, i. e. one hour after the commencement of inhalation of the compressed air it was reduced to

from 1.36 to 0.39 c.c. with an average of 0.86 c.c., in two hours to from 1.51 to 0.40 c.c. with an average of 0.90 c.c. After removing the excess pressure, the stroke volume increased :—namely, one hour after the normal respiration it amounted to from 2.09 to 0.49 c.c. with an average of 1.11 c.c.; two hours thereafter, returning to from 2.12 to 0.50 c.c. on an average to 1.20 c.c. These facts agree with the results which Huggett³⁾ obtained in his investigation, using the expiratory obstruction.

The oxygen consumption per minute amounted to from 29.89 to 18.66 c.c. with an average of 23.92 c.c. before the inhalation of the compressed air. During the inhalation of compressed air it was lessened in one hour to from 30.27 to 12.37 c.c. with an average of 20.61 c.c. and in two hours to from 28.35 to 12.06 c.c. on an average to 19.21 c.c. One hour after removing the compressed air, it was raised a little showing values fluctuating from 27.84 to 10.96 c.c. with an average of 20.08 c.c., but even after two hours it could not completely recover, amounting to from 24.36 to 13.36 c.c. with an average of 20.14 c.c.

The arteriovenous difference of oxygen contents per c.c. of blood, i. e. oxygen utilisation, which amounted to from 0.12132 to 0.06045 c.c. with an average of 0.08365 c.c. before the compressed air was inhaled, increased markedly during the inhalation of it, being augmented in one hour to from 0.13569 to 0.07695 c.c. with an average of 0.10415 c.c., in two hours to from 0.12560 to 0.05813 c.c. with an average of 0.09614 c.c. Respiration at normal pressure brought it to decrease more or less :—namely in one hour it was lowered to from 0.11337 to 0.04678 c.c. with an average of 0.08511 c.c., in two hours keeping the value from 0.10136 to 0.04475 c.c. with an average of 0.08579 c.c.

To ascertain the results above mentioned, in an experiment compression was given once more two hours after the decompression, as is shown

TABLE II.

Body weight (kg.) & sex	Time of observation	Respiration per min.		Pulse rate per min.	Oxygen con- sumption per min. (c.c.)	Arteriovenous difference of oxygen contents (c.c.)	Minute volume		Stroke volume	
							c.c.	p.c. diff.	c.c.	p.c. diff.
2.15 ♀	Before compression	120	228	28.47	0.11629	246			1.08	
	1 hr. after 1st compression	62	216	21.25	0.13569	157	-36		0.73	-32
	1 hr. after decompression	76	228	25.65	0.09468	271	+10		1.19	+10
	2 hrs. after decompression	74	228	21.71	0.09550	227	-8		1.00	-7
	1 hr. after 2nd compression	74	210	17.28	0.13829	125	-49		0.60	-44
	1 hr. after decompression	100	222	16.01	0.09640	165	-33		0.73	-32

in Table II. In this second examination it was found that the decreases of the minute volume, the stroke volume and oxygen consumption as well as the increase of the oxygen utilisation have the same tendency as the results shown in Table I, but in adding the second compression in the respiration apparatus the grade of the variations was greater than that in the first observation.

DISCUSSION.

The experiments indicate that in non-anaesthetised rabbits the minute volume is remarkably influenced by a respiration of compressed air up to 14 cm. of water. That one hour after the exposure to compressed air the minute volume of the heart shows a considerable decrease may probably be based on the following fact:—since the pressure of the compressed air acting on the inside surface of pulmonary alveoli has an effect upon the great endothoracic veins as well as on the weak parts of the heart muscles, the blood-flow can not easily stream into the endothoracic veins on account of the disturbed condition of the heart, and in turn stasis is aroused in the peripheric venous system, whereby the blood-flow returning to the right side of the heart is reduced. It is also probable that the high pressure in the pulmonary alveoli affects the pulmonary capillaries, and, accordingly, the passage of blood from the pulmonary arteries to the pulmonary veins is interfered with. Through these two factors the blood-flow to the left ventricle is necessarily decreased, and consequently the output from the ventricle to the aorta also. Two hours after exposure at excess pressure, the minute volume does not differ a great deal from that of the first hour after the inhalation of the compressed air, but on account of the continued forced respiration, the respiration and circulation become so fatigued that in some cases the minute volume shows a tendency to decrease slightly. After the cessation of the respiration of compressed air the general conditions returning to the former state, the causes of the circulatory disturbances are taken away, with the result that the minute volume gradually shows a tendency to recover, augmenting more or less one hour after the normal respiration, but since the respiratory and circulatory system have not yet sufficiently recovered from the fatigue, it has not yet reached the initial value. Two hours afterwards the minute volume increases further. Of course in these variations, anaemia due to the taking of blood several times, and an enfeeblement of the heart owing to the repeated cardiac punctures may fairly take part. The fact that the minute volume does not recover

completely after the cause is removed is presumably related to these accessory changes. A conspicuous decrease in the minute volume in the second observation mentioned above may also occur from the same process.

The variation of the stroke volume can also be explained as occurring for the same reason as that of the minute volume.

It is beyond discussion that a decrease in the minute volume during the respiration of compressed air has a great effect upon gaseous and general metabolisms of tissues. A decrease in the oxygen consumption during the respiration of compressed air may be partly due to the decreased blood-volume passing through the lungs per minute, which leads to a diminished oxygen intake in the lungs. But with this explanation alone the decrease in the oxygen consumption cannot be completely illustrated. A considerable increase in the arteriovenous difference of oxygen contents per c.c., i.e. the oxygen utilisation, during the respiration of compressed air is based on the prolonging of the time in which the blood and tissues are in contact with each other through the capillaries because of the decrease of the minute volume, and on the fact that the tissues take up as much oxygen as possible from the unit volume of the blood in order to fulfill the demands of the tissues. With such a mechanism the obstacle of the gaseous and general metabolisms seems to be mitigated. If the total blood volume increases during the respiration of compressed air, as Moteki¹⁾ of our Laboratory observed, this fact can also relieve more or less the hindrance of gaseous and general metabolisms, which is provoked by the decrease in the minute volume.

CONCLUSIONS.

1. In rabbits the minute volume and the stroke volume of the heart are considerably decreased during the respiration of excessively compressed air up to 14 cm. of water.
2. These alterations bring about a decrease in the oxygen consumption per minute and an increase in the arteriovenous difference of oxygen contents per c.c. i. e. the oxygen utilisation.
3. These alterations cease more or less, after the cessation of the respiration of the compressed air, and the former condition is recovered.