A CLINICAL INVESTIGATION OF THE CARBONIC ACID IN THE ALVEOLAR AIR*

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The experiments of Mosso, Haldane and his collaborators, and of Yandell Henderson have shown that the maintenance of a definite percentage of carbon dioxid in the alveolar air of the lungs and in the blood is of prime importance to the organism and is maintained by a definite physiological regulation. They have also shown that overventilation of the lungs by hyperpnea, which reduces the concentration of the CO_2 in the alveolar air below normal, gives rise to a feeling of weakness and giddiness exactly like that of mountain sickness, and when pushed to extremes, also to periodic breathing of the Cheyne-Stokes type.

Yandell Henderson has produced a good deal of evidence to indicate that the clinical manifestations of surgical shock may be brought about by a condition of acapnia; and Porges, Leimdörfer and Marcovici have found acapnia present in cases of acidosis and in certain cases of cardiac dyspnea.

As the subject seemed to warrant further investigation from the clinical side, the following observations were made.

We take great pleasure in expressing our thanks to Drs. Barker, Thayer, Halsted and Williams for the privilege of investigating cases in their respective services in the Johns Hopkins Hospital, as well as to the members of the house staff for their hearty cooperation.

In carrying on a series of investigations, along with other routine duties met with in the wards of a large general hospital, the first essential was the possession of a portable form of apparatus. The appartus to be described below, which possessed all the essentials of a complete Haldane outfit, but which was not much larger than the case of a microscope and could be easily and rapidly transported to different parts of the hospital, was therefore constructed.

EXPERIMENTAL APPARATUS AND TECHNIC

The apparatus consisted essentially of the Haldane long rubber tube and gas buret for collecting samples of air, connected with a Hempel absorption bulb containing a solution of one part by weight of potassium hydroxid in two parts of water.

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The patient took between his lips a mouth-piece made of a slightly flattened piece of glass tubing 1.5 cm. in diameter, which was connected by a thin-walled tube of pure gum rubber with a brass tube 4 cm. long from the side of which a T-tube 0.6 cm. in diameter passed through a hole in the box lid to connect with the gas buret on the inside of the box. The further end of the wide brass tube was connected with a rubber tube of corresponding width 210 cm. long which was coiled up like a trumpet and firmly wired in place on the outside of the lid. A curved Ochsner clamp inserted through a screw-eye just above the soft rubber tube enables the operator to clamp off the latter quickly at the end of the subject's expiration, in order that the air in the long tube may be analyzed at leisure. The capacity of this tube was 370 c.c. and since the mean volume of residual air is about 150 c.c. it can be assumed that at the end of a forced expiration the proximal portion of this tube contained almost pure alveolar air.

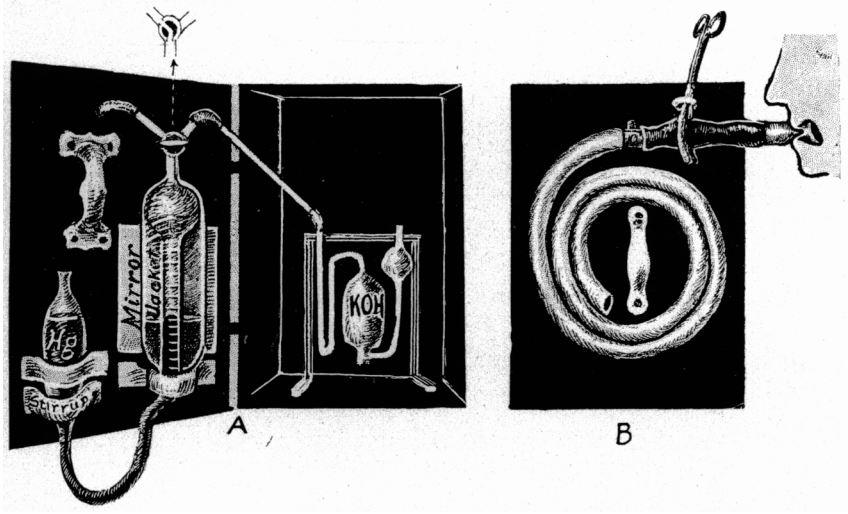

Fig. 1.—Portable apparatus for the determination of carbon dioxid in the alveolar air. A. Inner aspect of the lid and box, showing the three-way cock, the gas buret and water-jacket, the mercury vessel resting in the stirrup and the handle for lifting the lid. Within the box is shown the Hempel absorption bulb (KOH) and its connections. B. External aspect of lid, showing the Ochsner clamp, the mouth-piece, the coiled rubber tube and the second handle for lifting the lid.

The gas buret in which the expired air was collected was specially constructed for the purpose and consisted of an unmarked bulb of about 75 c.c. (75.7 c.c.) capacity above which there was fused a 3-way cock whose tubes connected on the one hand with the T-branch of the collecting tube and on the other by means of small bore (1 mm.) barometer tubing with the Hempel absorption bulbs. On the lower end of the bulb tube there was fused a small tube of 10 to 15 c.c. capacity graduated in 0.1 c.c.[1] The bulb and the buret are inclosed in a water-jacket 6 cm. in diameter to keep the air within at exactly room temperature. The lower end of the gas buret is connected by a heavy rubber tube with a glass vessel of about 100 c.c. capacity, preferably the bulb of a

1. In the first experiments a bulb of 35.7 c.c. capacity was used without the water-jacket, and the connections between the parts were made with rubber tubing. The results obtained with this small instrument did not differ materially from those obtained with the larger water-cooled bulb.

100 c.c. pipet which can be left to rest on a padded stirrup of metal placed on the door of the box a little below the level of the buret and a few inches to the left of the latter. The air is drawn into the buret by lowering this mercury vessel, or more conveniently by having it rest in the stirrup when the 3-way cock is opened. When the latter is closed the exact volume at atmospheric pressure is ascertained by raising or lowering this vessel until the top of the mercury menisci in both vessels are at exactly the same level. The leveling is somewhat facilitated by fixing a small mirror on the door behind the buret and raising and lowering the mercury vessel until the two menisci and their images are seen at the same level (Fig. 1). In order that the air within the bulb should remain saturated with moisture, 0.5 c.c. of water slightly acidulated with dilute acetic acid is left floating above the mercury and the volume of the air is read from the lower edge of this water meniscus rather than from the upper edge of the mercury meniscus. A stock bottle of this acidulated water is kept on hand and is colored pink by the addition of dimethylaminoazobenzol in order to show always that no potassium hydroxid has been carried back into the bulb. After the volume of expired air has been measured, the CO_2 content may be determined by connecting the free arm of the 3-way tube with the Hempel absorption bulbs, preferably by means of an intermediary arm of fine-bored glass tubing (barometer tubing) with short rubber connections. The 3-way cock is then turned to open into this arm and the air forced completely out of buret and bulb and into the Hempel bulb by raising the mercury vessel. When all of the air has been forced out of the bulb, the stop-cock is again turned off full so that no air can return and the mercury vessel returned to the stirrup. The expired air is left in the absorbing bulb for one minute which is sufficient to absorb the CO_2 and then the stopcock is turned back and the air drawn back again into the bulb and buret until the KOH returns to its former level in the arm of the Hempel tube. The stop-cock is once more turned off, the mercury leveled again, and the buret read. The difference between the two readings represents the CO_2 in the expired alveolar air. The percentage of CO_2 is calculated according to the equation

$$\text{Per cent } CO_2 = \frac{\text{First reading minus second reading}}{\text{Volume of bulb plus first reading.}}$$

No correction need be made for temperature and pressure since they are the same for both readings, and therefore do not affect the percentage.

In collecting the expired air the subject is first made to breathe naturally, then to put the mouth-piece in his mouth and at the end of a quiet expiration expire as forcibly as possible, or give a violent cough, through the tube; and at the end of this forced expiration the tube is quickly clamped off. Several readings are taken; those which are most divergent are discarded and the average of the more or less coincident ones recorded. A few preliminary determinations must be made with each patient to accustom him to the use of the apparatus.

In investigating the more or less heterogeneous series of cases presenting themselves in the wards several points were kept uppermost in mind: First, to determine if possible whether the feeling of asthenia in convalescent patients and especially in patients who have gotten out of bed for the first time, is associated with the presence of acapnia. Second, whether the latter phenomenon is responsible for these symptoms in patients with enteroptosis. Third, the extent to which acapnia develops in cardiac dyspnea, and its relation to the mechanism of the latter.

It would have been particularly interesting for us to have determined the alveolar air in clinical cases of surgical shock, but those

TABLE 1.—EXPERIMENTS WITH SMALL INSTRUMENT. RESPIRATION NORMAL

No.	Date	Name	Race	Sex	Age	Diagnosis	Symptoms and General Condition	Resp. Rate	Form of Chest	Per Cent CO_2
50	5/23/12	Tol.	W	28	M	Inflam. verumontanum and utricle.	Walking around. Looks healthy. Much pain, however.	16 to 20	Very well formed	4.9
56	5/25/12	Mey	W	30?	M	Paramyoclonus multiplex.	In bed for a few days. Healthy except for jerking of muscles which makes patient very nervous.	16 to 20	Fairly well formed	4.0
64	6/ 1/12	Dad.	W	24?	M	Perfectly healthy.	Leading natural life as orderly.	16	Well formed	5.3
74	5/20/12	Hir.	W	35?	M	In good health now. Recently sick in bed.	Not up to usual standard as yet.	16	Rather deep chest. Heavy built	5.4
75	7/21/12	E. G.	W	27	M	In fairly good health.	Not robust. Somewhat frail build.	16	Well formed but small.	4.0
76	5/28/12	E. G.	W	27	M	In fairly good health.	Not robust. Somewhat frail build.	16	Well formed but small.	4.1

Ammonium sulphate..... 6.05 Total ammonia 3.01

TABLE 2.—EXPERIMENTS WITH LARGE INSTRUMENT. RESPIRATION NORMAL

No.	Date	Name	Race	Age	Sex	Diagnosis	Symptoms and General Condition	Resp. Rate	Form of Chest	Per Cent CO_2
1	7/17/12	Mary Bartens	W	30	F	Typhoid.	In bed 1 month. Not up yet. Convalescent.	20 to 24	Long and narrow	5.4
2	7/17/12	A. R.	W	11	F	Typhoid.	In bed 14 days. At height of fever. Not extremely sick.	20	Slender child. Medium sized chest	5.2
3	8/10/12	A. R.	W	11	F	Typhoid.	At home. Has been walking about for 1½ weeks. General condition good. Improving rapidly.	20	Slender child. Medium sized chest	5.0
4	7/19/12	W. R.	C	25	M	Typhoid.	In bed 35 days in hospital. Still in bed. Beginning convalescence. Tired out from trials.	24	Well built	4.9

TABLE 4.—EXPERIMENTS WITH LARGE INSTRUMENT IN CARDIOVASCULAR DISEASE

No.	Date	Name	Race	Sex	Age	Diagnosis	Symptoms and General Condition	Resp. Rate	Form of Chest	Per Cent. CO_2
7	7/17/12	C. Z.	W	45	M	Aortic insufficiency. Arteriosclerosis.	In bed in hospital 3 weeks. Up yesterday and to-day for 2 hours. Now in bed. Nocturnal dyspnea previously. Practically none now. Pretty well compensated.	22	Somewhat barrel-formed.	3.9
8	7/17/12	C. H.	W	51	M	Aortic insufficiency. Arteriosclerosis. Tabes?	Five weeks in bed. Good compensation now. Formerly nocturnal dyspnea and myocardial insufficiency. In bed now. Has not been up.	22	Rather deep	6.3
9	7/18/12	P. J.	W	22	M	Aortic insufficiency. Mitral insuff. ac. rheum. fever 1 month ago.	Walking around for 1 week. Up now. No nocturnal dyspnea or cardiac asthma for some time.	20	.....	5.3
10	7/18/12	Mrs. O.	W	54	F	Complete heart-block.	Pulse 50 (digitalis). In bed 9 days. Complains of strange feeling over heart and stomach.	20 to 24	Small. Somewhat flat.	5.9
13	7/20/12	C. N.	C	64	M	Myocardial degeneration. Arteriosclerosis. Slight myocard. insuff. now.	Gets up each day for several hours. Has been in hospital for 5 months. Came in in great decompensation. Never fully compensated since.	28	Barrel	5.3
15	7/21/12	J. W.	C	33	M	Aortic aneurysm.	Has been up in chair for 11 days. Walks some. No dyspnea.	20	Well formed	4.5
16	7/21/12	F. R.	C	49	M	Arteriosclerosis. Emphysema. Angina pectoris.	In bed for 3 days. Up for past 9 days in chair. Very little walking. No dyspnea. Somewhat feeble.	20 to 22	Fairly well formed	4.5
17	7/21/12	W. S.	C	15	M	Mitral insufficiency. Myocardial insufficiency.	Decompensated now. Slight dyspnea standing still. Walking a little to-day. Coughs violently after each trial.	30 to 32	Fairly well formed	4.2
29	7/25/12	W. S.	W	17?	M	Mitral insuff. — stenosis. Marked hypertrophy. Pericardial effusion?	Sitting up in bed now. Subjective dyspnea only. In bed 3 days. No nocturnal dyspnea now.	24 to 28	Fairly well formed. A little flat.	3.6
35	7/26/12	C.	W	56	M	Aortic insufficiency. Arteriosclerosis.	Discovered heart trouble only 4 months ago. Fair amount of dyspnea now.	20 to 24	Deep	4.5
36	7/27/12	J. S.	C	60?	M	Myocardial insuff. Arteriosclerosis. Emphysema.	Venesection yesterday on admission. No dyspnea now.	20	Deep and barrel	5.4
43	8/24/12	I. G.	C	27?	F	Aortic insufficiency. Decompensated now.	Considerable dyspnea. Varies in extent at times. Trials made at different times.	32 to 36	Very fat deep chest	4.1
								36		3.9
								36 to 40		3.3
						.....	On leaving hospital no dyspnea.	24	.....	3.58

TABLE 5.—EXPERIMENTS WITH SMALL INSTRUMENT IN A NEURASTHENIC

No.	Date	Name	Race	Age	Sex	Diagnosis	Symptoms and General Condition	Resp. Rate	Form of Chest	Per Cent. CO_2
53	5/22/12	Stan.	W	35?	M	Neurasthenia. Constipation.	Occupation (tailor), race, and home life account for condition. No dyspnea.	16 to 20	Fairly well formed	4.3

TABLE 6.—EXPERIMENTS WITH LARGE INSTRUMENT IN NEURASTHENICS

No.	Date	Name	Race	Age	Sex	Diagnosis	Symptoms and General Condition	Resp. Rate	Form of Chest	Per Cent CO_2
11	7/19/12	Mrs. K.	W	35	F	Neurasthenia.	In bed 17 days. Remarkable cure of neurasthenia. Nothing but home life and race to account for state. Complains of headache and weakness from blowing.	22	Heavy set. Fairly deep.	5.0
6	7/17/12	C. P.†	..	..	..	Mucous colitis.	.....	..	.....	...
18	7/21/12	D. S.	W	38	F	Neurasthenia.	First day in bed. Race and home life explain condition.	20	Well formed	4.9
31	7/26/12	A. W.	W	33	M	Gastric neurosis. Looks neurotic.	In bed 3 days. Not very sick.	20	Fairly well formed	5.4
33	7/26/12	T. T.	W	30	M	Psychoneurosis?	In bed 3 days. No dyspnea. Sick 11 years off and on.	20	Fairly well formed	4.5
39	7/27/12	R. F.	W	42	F	Mucous colitis. Secondary neurasthenia.	No hyperthyroidism. In bed 3 days. No dyspnea. Not ill, but looks worn.	20	Fairly well formed	4.3
40	7/27/12	J. D.	W	46	F	Colitis. Neurasthenia. Slight enteroptosis.	No dyspnea. In bed now. Up during day.	20	Slightly flat	5.3

†See under Resp. normals.

5	7/19/12	C. J.	C	19	M	Typhoid.	In bed in hospital 19 days. Beginning convalescence.	24	Well built	4.3
6	7/17/12	C. P.	W	32	M	Mucous colitis (?)	Has been up and about until a few hours ago. No respiratory trouble of any sort.	20	Fairly well formed	5.8
12	7/20/12	Z. T.	C	24	M	Ulcus ventriculi.	In bed 5 days. Does not appear very sick.	24	Well formed	4.3
26	7/24/12	K. F.	W	28	F	Congenital dislocation of hips. Healthy otherwise.	In bed 4 weeks. Operation 4 weeks ago. Has laid flat on back constantly. Now on back.	20	Fairly well formed	4.8
27	7/24/12	B. P.	W	64	F	Carcinoma of breast. Otherwise well.	Operation 16 days ago. In bed since. Recently sits up in bed.	20	Very slender small woman. Small chest.	4.9
28	8/ 5/12	B. P.	W	64	F	Carcinoma of breast. Otherwise well.	Up and walking about for 4 days. No dyspnea.	20	.....	5.3
30	7/25/12	H. B.	W	19	M	Uncinariasis.	Only slight hookworm symptoms. In bed 24 hours only.	20	Well formed.	4.9
32	7/26/12	L. J.	W	40	M	C. N. S. lesion.	No dyspnea. Difficulty in speech and gait.	20	Well formed.	4.4
34	7/26/12	A. R.	W	54	M	Gall-stones (operation).	In bed 2 weeks. No operation as yet. No dyspnea. In no pain at present.	20	Deep and barrel	4.9
37	7/27/12	T. B.	C	41	M	Direct inguinal hernia. Perineal fistula. Pulm. tuberc. (?) Lues. Ascites.	In bed 2 months. Has never been up since admission. Now on back.	20	Rather flat.	4.7
38	7/27/12	H. C.	C	25	M	Pleurisy with effusion. Rt. lung now collapsed.	Operation on chest 8 days ago. In bed on back since.	20	Right side depressed	4.6
41	8/10/12	A. E. H	W	42	M	Double hernia. Otherwise well.	Fourteenth day after operation. In bed since.	20	Good average size	5.2
42	8/12/12	A. E. H	W	42	M	Double hernia. Otherwise well.	Up for first time. Standing beside bed. Some slight light headedness. Weakness and trembling in legs. No dyspnea.	20	Good average size	4.4
44	8/21/12	J. W.	W	36	M	Adhesions of colon. Chr. appendicitis.	In bed now, 13 days after exploratory laparotomy. Looks neurotic.	20	Good average size	5.0
45	8/23/12	J. W.	W	36	M	Adhesions of colon. Chr. appendicitis.	Walking slowly. Has just gotten up for first time. Weakness of legs. Light headedness.	20	Good average size	5.2
46	8/12/12	J. P.	W	35	M	Gastric ulcer. Pyloric stenosis. Marked general enteroptosis.	Emaciated. Stomach below navel. Very weak. Before operation. In bed now.	20	Phthisical. Long, flat, narrow. Lungs clear.	5.5
47	8/14/12	J. P.	W	35	M	Gastric ulcer. Pyloric stenosis. Marked general enteroptosis.	Before operation. Standing at bedside for some time. Records taken on rising and some time later.	20	Phthisical. Long, flat, narrow. Lungs clear.	5.2
48	8/11/12	J. R.	W	29	M	Appendicitis. Operation.	Gets up q. day. Up early this a. m. Now in bed for 2 hours. No dyspnea.	20	Fairly well formed	5.5
49	8/12/12	J. R.	W	29	M	Appendicitis. Operation.	Has been walking around—active for 3 hours. Respiration normal.	20	Fairly well formed	5.0
80	8/24/12	Hir.	W	35?	M	Healthy.	In prime condition.	16	Large chest. Rather deep.	5.8
81	8/19/12	E. G.	W	27	M	Healthy.	Not in absolute robust health.	16	Well formed but small.	4.3

TABLE 3.—EXPERIMENTS WITH SMALL INSTRUMENT IN CARDIOVASCULAR DISEASE

No.	Date	Name	Race	Sex	Age	Diagnosis	Symptoms and General Condition	Resp. Rate	Form of Chest	Per Cent CO_2
51	5/22/12	Bod.	W	58	M	Myocardial degeneration.	No dyspnea. In bed for 2 weeks. Decompensation on admission.	20 to 24	Barrel	4.3
52	5/22/12	Som.	W	54	M	Myocardial degeneration. Chronic nephritis.	In bed many weeks. Repeated decompensations. Now compensated. No dyspnea while perfectly quiet.	24	Deep	3.9
54	5/21/12	Nich.	C	64	M	Myocardial insufficiency. Aortic insufficiency.	In bed 10 days. Venesection 2 days before. Very sick now. Dyspnea.	36 to 40	Barrel	3.3
57	5/20/12	Con.	C	62?	M	Mitral stenosis + insufficiency. Arteriosclerosis.	Gets out of bed during day. Some dyspnea —more on exertion.	30 to 36	Barrel	3.6
62	5/20/12	Wat.	C	52?	M	Aortic insufficiency. Slight myocardial insufficiency.	Very slight if any dyspnea. Some last night.	20	Deep	3.6
63	6/ 1/12	Som.*	W	54	M	Myocardial degeneration. Chronic nephritis.	Up during day for 1 week.	20	Deep	4.5
65	6/ 2/12	C. N.	C	64	..	Myocardial degeneration. Arteriosclerosis. Slight myocardial insufficiency.	In bed many weeks. Entered with marked decompensation. See No. 13 under cardiacs with new machine. Same patient.	20	Barrel	3.3
66	6/ 3/12	Bod.	W	73	M	Myocardial degeneration. Chr. nephr. Emphysema.	In bed for many weeks. Entered with decompensation.	24	Barrel	4.4
67	6/ 3/12	Sch.	W	13?	M	Mitral stenosis + insuff.	In bed for weeks. Compensated. Poor cardiac action.	36	Fairly	4.0
72	6/11/12	Jef.	C	22	M	Mitral + aortic insuffic.	In bed 7 days. No dyspnea.	16 to 20	Fairly	5.2
58	5/26/12	Med.	C	58?	M	Myocard. insuff. Chr. nephritis. Arteriosclerosis.	In bed 5 weeks. Very slight decompensation.	36 to 40	Deep	2.6

*See No. 52 above. Same case.

TABLE 7.—EXPERIMENTS WITH LARGE INSTRUMENT IN PREGNANCY

No.	Date	Name	Race	Age	Sex	Diagnosis	Symptoms and General Condition	Resp. Rate	Form of Chest	Per Cent. CO_2
19	7/22/12	D. McK.	W	25	F	Pregnancy at term.	Second child. Walking. Shallow expirations. Nervous. No dyspnea, except on exertion.	32	Somewhat flat	3.9
20	7/22/12	E. D.	W	28	F	Pregnancy at term.	First child. Walking. Deep expirations. Not nervous. Dyspnea only on exertion.	24 to 28	Well formed	4.2
21	7/22/12	M. E. B.	W	23	F	Pregnancy at term.	Fourth child. Shallow expirations. Not nervous. Dyspnea only on exertion.	20	Fairly well formed	5.3
22	7/22/12	E. J.	W	33	F	Pregnancy at term.	Fifth child. Exceedingly short expirations. Hard for patient to follow instructions. Dyspnea on exertion.	24	Fairly well formed	4.3
23	7/24/12	K. W.	W	21	F	Pregnancy at term.	First child. No dyspnea except on rather severe exertion. Excellent patient.	24	Well formed	4.8
24	7/24/12	A. R.	C	26	F	Pregnancy at term.	Second child. Dyspnea on walking.	32	Well formed	3.9
25	7/24/12	J. M.	C	24	F	Pregnancy at term.	Third child. Considerable dyspnea on exertion.	24	Well formed	3.9

TABLE 8.—EXPERIMENTS WITH SMALL INSTRUMENT IN TYPHOID FEVER

No.	Date	Name	Race	Age	Sex	Diagnosis	Symptoms and General Condition	Resp. Rate	Form of Chest	Per Cent. CO_2
60	5/26/12	Wol.	W	20?	M	Typhoid.	Twenty-one days in bed. Beginning convalescence.	20 to 24	Fairly well formed	3.6
68	6/ 7/12	Bar.	W	26	M	Typhoid.	In bed 2 months. Up for 3 days, 1 hour q. d. Frail looking man.	20	Rather flat chest	5.4
69	6/ 7/12	Clem.	W	28	M	Typhoid.	In bed 1½ months. Up 3 days 1 hour q. d.	20	Fairly well formed	5.6
70	6/ 7/12	Wol.	W	20?	M	Typhoid.	See Case 60 above (same case). First day up, ½ hour in chair. Now in bed again.	20	Fairly well formed	4.0
71	6/ 9/12	Strec.	W	46?	M	Typhoid? Clinical but blood culture neg.	In bed for 8 days. No dyspnea. Quite sick.	16	Well formed	3.6

TABLE 9.—EXPERIMENTS WITH SMALL INSTRUMENT IN MISCELLANEOUS CASES

No.	Date	Name	Race	Age	Sex	Diagnosis	Symptoms and General Condition	Resp. Rate	Form of Chest	Per Cent CO_2
59	6/26/12	Wil.	C	32?	F	Diabetes. (Formerly intense acidosis.)	In bed for many weeks. Carbohydrate-free diet and $NaHCO_3$. Now up in day for 2 weeks. Shallow respiration.	20 to 24	Fairly well formed	3.7
73	6/14/12	Qk.	W	56	M	Bronchial asthma marked.	In bed for several weeks most of day. Some dyspnea now—attack fairly started.	24	Barrel	4.5
61	5/29/12	Bell	C	29?	M	Bronchial asthma.	Constantly marked expiration. Dyspnea.	20	Deep barrel	6.4

TABLE 10.—EXPERIMENTS WITH LARGE INSTRUMENT IN MISCELLANEOUS CASES

No.	Date	Name	Race	Age	Sex	Diagnosis	Symptoms and General Condition	Resp. Rate	Form of Chest	Per Cent. CO_2
14	7/20/12	T. R.	C	35	M	Septicemia? Nature of infection never definite.	Gets up for a part of each day. Some little dyspnea when up. Now in bed. Appears sick.	28	Fairly well formed	5.4

patients in whom the degree of shock was sufficiently high to be regarded as crucial tests it did not seem safe to subject the patient to the ordeal of this examination.

We therefore tried to determine whether there might be any relation between acapnia and the production of symptoms of asthenia such as are met with in convalescents from long illnesses, typhoid fever, surgical operations and also in persons with enteroptosis. The results of determinations on such patients as shown on the chart falls within normal limits (4.0 to 5.5 per cent.) in most cases, though in a few convalescents from typhoid fever they were a little lower. These results coincide well with the figures obtained on normal individuals by Haldane and his collaborators, especially Mabel Fitzgerald.

We also tried to determine whether the weakness, dizziness and similar sensations experienced by convalescents on first getting out of bed were associated with acapnia. This was studied in four patients. In two of these — a man who had just gotten out of bed after an operation for appendicitis and a patient with gastric ulcer and extreme ptosis of the viscera — there was a slight fall of alveolar CO_2 (from 0.3 to 0.5 per cent.), a little more than the diurnal variation. In one convalescent from an appendix operation there was a rise of 0.2 per cent. in spite of the fact that he complained of weakness and some giddiness and light headedness. One of the four patients, a convalescent from a double herniotomy, who also had these symptoms, showed a marked fall in the CO_2 from 5.3 per cent. down to 4.4 per cent. There was, however, no marked change in rate of respiration.

These figures all represent percentages well above the level of acapnia, although in only one of the cases was the fall sufficiently great to account for the occurrence of any such symptoms.

We also investigated the alveolar CO_2 of a number of heart cases. It was difficult to investigate these in the most severe grades of dyspnea, because the mere act of making a very large forced expiration threw them into violent coughing spells and made them feel so ill that it was not possible always to repeat the observations often enough to secure concordant results. A considerable number of these cases gave results below the lowest level for normals and this was especially marked in those who had rapid respiration rates at the time of making the determination.

After this work was in progress we found a short article in the literature by Porges and Marcovici describing similar findings of low CO_2 in certain but not in all cases of cardiac dyspnea. It would appear that we have in cardiac disease two distinct mechanisms for the production of dyspnea:

1. Stasis and congestion in the pulmonary area, associated with difficulty in aeration of the blood. This may act either by reflex stimulation of the vagus endings in the lung or by increasing the CO_2 in the blood bathing the respiratory center which will give rise to hyperpnea until the CO_2 falls to its normal level.

2. Slow arterial circulation through the medulla, as in arteriosclerosis, aortic insufficiency and general failure of the circulation, which gives rise to symptoms of lack of oxygen in the latter and the formation of acids there quite independent of the CO_2. This acid intoxication overstimulates the respiration and gives rise to over-ventilation of the lungs, acapnia, and probably sometimes Cheyne-Stokes breathing, for Pembrey and his collaborators have shown that the latter can be stopped by inhalations of CO_2. It is probable that such cases can be benefited by rebreathing, but we have not been able to answer this question as yet.

We had one patient with Adams-Stokes disease with complete heart-block, who was not having any attacks or dyspnea at the time of determinations, whose alveolar CO_2 was normal.

A number of observations were made on pregnant women near term who had slight dyspnea, most of whom were a little below the lower limits of normal, which might be easily accounted for on purely mechanical grounds, though the possibility of a mild acidosis such as has been assumed by Porges and his collaborators cannot be excluded.

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