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ON THE CAUSES OF DYSPNŒA AND CARDIAC FAILURE IN HIGH ALTITUDES.

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IN the course of some experiments during the past winter, upon the physiological effects of rarefied and compressed air, and reported to the American Climatological Association in May,[1] I was often struck with the fact that sudden and considerable rarefaction of the air produced not only extreme dyspnœa but also a rapid, irregular, and intermittent pulse in the animals experimented upon.

The apparatus used by Prof. Martin and myself was the pneumatic cabinet, at present so much employed for the application of rarefied and compressed air to respiratory diseases, and which is used somewhat indiscriminately, without proper regard to the possible danger of subjecting patients with cardiac disease to treatment by this method. The animals, be it said, were placed in the cabinet in the same manner and subjected to the same changes of pressure as are patients.

[1] Preliminary Account of Experiments in regard to the Circulatory and Respiratory Changes observed in Animals placed in the Pneumatic Cabinet, by H. N. Martin and Frank Donaldson, Jr., New York Medical Journal, May 15, 1886.

In our experiments it was found that:

I. When the animal is breathing air from outside the cabinet, rarefaction of air within the cabinet *causes a marked fall of general arterial pressure*, but has no influence on the pulse-rate.

II. This fall of systemic arterial pressure depends on two factors: greater flow of blood to the skin when the air around the animal is rarefied, and greater accumulation of blood in the lungs when they are distended.

III. Of these two factors, accumulation of blood in the lungs is the more effective; for, if the animal breathes air from the cabinet and not from outside, rarefaction of air within the cabinet (in this case accompanied by no special expansion of the thorax) has but a trivial effect in lowering arterial pressure, unless the rarefaction is very considerable—about what would correspond to three or four thousand feet—when the fall of pressure was more marked.

IV. When the animal is breathing external air, rarefaction of air within the cabinet usually has no effect upon the respiratory rate or the extent of individual respiratory acts, unless the fall of blood-pressure is considerable. *If it is considerable, symptoms of anæmia of the medulla oblongata are seen. In most cases there is more forcible dyspnœic breathing; in some there are dyspnœic convulsions similar to those which occur when an animal is bled to death*, and due to the same cause, viz., deficient blood-flow to the respiratory centre.

So convinced was the writer from these facts, of the danger of this method of treatment that he was led to lay down the following rule, and upon which special stress was laid, viz., before deciding a person to be a proper subject for treatment by pneumatic differentiation, *thorough examination* should be made

of the *heart; and that no person found to have insufficiency or stenosis of the mitral or aortic valve, or the slightest tricuspid regurgitation, or a fatty or weak heart, should, under any condition, be subjected to treatment by rarefied and compressed air.*

I was not aware that this point had been emphasized before as to the danger in heart disease of the rarefied and compressed air treatment by this method, and was therefore much interested in Dr. Loomis's paper read before the same Society and just preceding my own. In this article, entitled "The effects of high altitudes on cardiac disease,"[1] Dr. Loomis gives the history of six cases of cardiac failure directly due to removal to high altitudes. He has records, however, of twenty-two. The extreme importance and novelty of these cases entirely justify the insertion here of two.

CASE I.—"In the summer of 1880, during my vacation at St. Regis Lake, in the Adirondack Mountains, I was requested to visit a gentleman who had just arrived from New York and was thought to be dying. I found a gentleman, forty years of age, sitting at an open window, gasping for breath, and deeply cyanosed. No distinct radial pulse could be detected, his extremities were cold, and his body was covered with a profuse perspiration; he was semi-conscious, unable to speak or swallow, and seemed to be dying. Neither heart-sound could be distinctly heard. After the hypodermatic administration of ten minims each of tinct. digitalis and Magendie's solution of morphia, with the free use of hypodermatics of brandy, he rapidly recovered from his extreme condition, and in a few hours was comparatively comfortable, but unable to lie down, and was only partially relieved of his dyspnœa, the radial pulse continuing rapid, feeble, irregular, and intermitting. The next morning I obtained the following history:

[1] New York Med. Journal, June 12, 1886.

"Three days before, Mr. C. had left New York City, feeling well, although for two or three months before he had noticed that he became easily fatigued, had dyspeptic symptoms, lost flesh, and spent restless nights. When he reached Plattsburg the evening before (the altitude of which is 150 feet above sea-level) he felt better than when he left New York, slept unusually well during the night, and started at eight in the morning for St. Regis Lake. When he reached an altitude of about 1000 feet his breathing became difficult, and, as he reached higher altitudes, the difficulty increased, and was accompanied by cardiac palpitation and a sense of oppression in the epigastrium. The dyspnœa and oppression became so urgent before he reached St. Regis (the altitude of which is about 2000 feet) that his friends had to support him, and, when taken from the carriage on his arrival, he was thought to be dying. A careful physical examination twenty-four hours after showed a diffused, indistinct cardiac impulse; the area of præcordial dulness was greatly increased both to the right and left. The first sound of the heart could not be made out; its time was occupied by a loud systolic murmur. The pulse was rapid, feeble, irregular in force, and at times intermittent; fine râles were heard at the base of both lungs. One examination of his urine gave negative results. After treating him for three days, with little apparent improvement in his cardiac symptoms, I advised that he should be taken to a lower altitude.

"When he reached Plattsburg on his return his dyspnœa was markedly relieved; when he reached his country home on the Hudson he had so far improved that he was able to walk on a level, and to lie down with his head and shoulders slightly elevated, although his dyspnœa, epigastric oppression, and irregular heart-action continued; his feet soon became œdematous, and six weeks later he died with general anasarca and heart insufficiency. No post-mortem examination was made. His family physician told me, some time after, *that he had never detected signs of cardiac disease until his return from the mountains, although he carefully*

examined his heart two weeks before he started on his summer trip."

CASE II.—"Mr. M., aged fifty-two, with recognized aortic insufficiency, which had never given him serious trouble (*in fact, he was not aware that he had cardiac disease*), on the 10th of August, 1882, started with his family on a pleasure trip through the Adirondacks. When he reached an altitude of about 1000 feet he began to suffer with dyspnœa and cardiac palpitation; when he reached St. Regis Lake he was markedly cyanosed, his respiration was gasping in character, and his pulse could not be counted. His apex impulse was diffused and indistinct, and he was having quite profuse pulmonary hemorrhage. I ordered a calomel purge, and ten drops of tinct. digitalis every three hours, which was followed by marked relief within twenty-four hours, although his dyspnœa and cardiac symptoms were not sufficiently relieved to allow him to lie down. A physical examination showed extensive cardiac enlargement, a diffused and indistinct apex beat, a feeble and irregular heart-action, an absence of the muscular element of the first sound, but no murmur could be detected. The urine contained albumen, but no casts; crepitating râles were heard over the base of both lungs. I advised that he return immediately to his home in Brooklyn, which he reached, the third day after, very much improved. His dyspnœa was so much relieved that he was able to lie down; his feet and legs soon became œdematous, general anasarca followed, and in two months he died suddenly. No autopsy was made, and I could get no intelligent account of the immediate cause of his death."

Dr. Loomis then goes on to say,

"In these cases which I have quoted, as well as similar ones which have come under my observation, *the ventricular dilatation*, which was unquestionably the cause of the sudden development of the distressing symptoms, and from which dated the commencement of the fatal issue, seemed to be directly due to the effects, on the *cardiac circulation, of the change from a low to a*

high altitude. I do not maintain that sudden cardiac dilatation might not have developed in any one of these cases under other conditions, but I do maintain that change in atmospheric pressure is a very important cause of sudden ventricular dilatation when any degree of heart insufficiency preëxists."

Finally, he declares that only with danger can a person whose heart is weak, fatty, or diseased, pass rapidly from a low to a high altitude, and, indeed, I gathered from his subsequent remarks that a person with cardiac disease should under no circumstances be sent to high altitudes.

Dr. Loomis then, had arrived at the same conclusion from clinical experience as the author had experimentally, and it seems that we may rest assured that there is great danger for the diseased heart either in high altitudes or in any form of treatment by rarefied and compressed air. This fact being established, our purpose is to inquire into the cause of dyspnœa and cardiac failure under these conditions.

To do this it will be necessary, first, to give the latest accepted explanation of the circulatory changes which take place in ordinary quiet respiration of air at normal pressure—750 millimetres of mercury—and then to see how high altitudes may affect these normal respiratory and circulatory phenomena.

It is very generally stated that the cause of dyspnœa and shortness of breath in high altitudes is the want of oxygen; that the system needing oxygen, more blood has to be sent through the lungs in a given time, thus increasing the amount of work to be done by the heart, which often dilates under the sudden strain. Such explanation, though applicable

for very high altitudes, is at the same time based on a misconception.

1st. The partial pressure of oxygen at normal atmospheric pressure is equal to 152 millimetres of mercury.

2d. So long as the partial pressure of this gas remains above 25 millimetres of mercury (one inch) the amount taken up by the blood will depend upon the amount of hæmoglobin in that liquid, and not on how much oxygen there is in the air.[1]

3d. The partial pressure of oxygen even at so great a height as 10,000 feet would be 101 millimetres of mercury, at which the hæmoglobin could get all the oxygen necessary.

But it may be urged, Paul Bert found an increased amount of hæmoglobin in the blood of animals in high altitudes, which would show a necessity on the part of nature for more oxygen. True, but this change takes place only after a very long time, probably not in one generation. Again, it may be objected that in many of those very cases sent to high altitudes the blood is poor in hæmoglobin. Doubtless this is true, but it is not the sick only, by any means, who suffer from dyspnœa and cardiac failure in high altitudes.

It is not necessary in this article to go into the various theories of the aspiration of the thorax, advanced by Poiseuille, Quincke and Pfeiffer, Funke and Latschenberger, Bowditch and Garland, Mosso, Hegel and Spehl, and others. I shall refer only to the work of de Jager, whose explanation of the respiratory phenomena has, on the whole, the greatest experimental proof to support it.[2]

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[1] Martin, Human Body, p. 384, 1st edition.

[2] For the *résumé* of the work of de Jager, with a critical exami-

As to the influence of the normal respiratory movement upon the circulation. In any given inspiration the chest cavity is enlarged, negative pressure is created in the thorax; not only the lungs, but the right side of the heart and the intrathoracic portions of the systemic veins, are expanded in consequence, and the blood is drawn toward the great veins and right side of the heart. Such being the general effect of negative intrathoracic pressure, what is its effect upon the blood-flow through the lungs, upon the intrathoracic vessels, and finally upon the systemic circulation?

De Jager holds that though the negative pressure is exerted equally on the pulmonary artery and vein, the effect upon the blood in each is very different, for the walls of the pulmonary artery are much thicker than those of the pulmonary vein, and any want of pressure would be transmitted through them upon the blood much less readily. Again, the walls of the pulmonary artery are normally distended by a pressure of from 30–40 millimetres of mercury, and so the slight increase of negative pressure occurring during inspiration would probably have little or no effect in distending the pulmonary artery. On the other hand, the blood in the pulmonary vein is under little, if any, positive pressure, and so diminution of pressure would distend its walls considerably. De Jager, therefore, holds that we would have the normal conditions during life more nearly represented if, in an artificial thorax, the pulmonary vein should be exposed to the same negative pressure as that expanding the lungs, while the pulmonary

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nation of his theory, I am greatly indebted to my friend, Dr. W. H. Howell, of Johns Hopkins University, who kindly allowed me to make use of his lectures on the aspiration of the thorax.

artery was left under a constant pressure of one atmosphere, for instance.

Experimenting in this way, he found that the stream velocity (the amount flowing from the pulmonary vein in fifteen seconds) is markedly greater during *expansion* than during *collapse;* the effect then of normal inspiration, on the whole, is to increase the quantity of blood flowing through the lungs, probably by increasing their capacity, and thus diminishing the resistance to the flow. De Jager, in his second paper, gives the final opinion that "the respiratory waves in the blood pressure in the dog in normal quiet respiration are caused by changes in lung circulation. The first sinking of pressure at the beginning of inspiration is a capacity curve—*i.e.*, due to increased capacity of the lung vessels; the following rise, a stream velocity curve"—*i.e.*, caused by an increased flow through the lungs. In a like way, the rise at the beginning of expiration is a capacity curve—*i.e.*, owing to a diminished capacity; the succeeding fall, a stream velocity curve, due to diminished flow through the lungs.

The influence of respiration upon the heart and great vessels. The first effect of the negative pressure of inspiration is a diminution of pressure on the heart and intrathoracic vessels, and a consequent *sucking* of blood into the right side of the heart, which in turn is sent through the increased area of the lung vessels to the left heart, and thence to the systemic circulation, causing a rise of pressure. Another important element in the respiratory waves of blood pressure is the change which takes place in intra-abdominal pressure in inspiration. Luciani and Schweinberg maintain that the descent of the diaphragm causes increased abdominal pressure, which assists the rise in arterial pressure, 1st, by

driving the blood out of the abdominal veins toward the heart; 2d, by compressing the abdominal arteries (a questionable supposition). Another cause of increased pressure during inspiration is the increase of heart rhythm, due, according to Fredericque (*Archives de Biologie*, 1882), to a rhythmic automatic discharge from the cardio-inhibitory centre—the discharge depending in some way on the rhythmic discharge from the neighboring respiratory centre.

The effects, then, of the negative pressure following inspiration, are an increased blood-flow to the large intrathoracic veins, and to the right heart; an increased heart capacity, especially of the right side; an increase in intra-abdominal pressure, forcing onward the blood; an increase of capacity in the pulmonary veins; an increased blood-flow through the lungs and to the left heart; an acceleration of the heart rhythm, and, finally, increase of systemic arterial pressure.

Such, in brief, is the theory of the circulatory changes which take place in ordinary respiration. It remains for us to inquire how the cycle may be affected in high altitudes.

However much the phenomena of normal quiet respiration may be changed in a highly rarefied atmosphere, the chief and immediate cause of dyspnœa and cardiac failure, may, I think, be traced to *the diminished pressure on the heart walls and their consequent dilatation.* This fact may be made plain as follows:

The external air presses against the interior of the lungs with a pressure equal to that exerted on the same area by a column of mercury 760 millimetres high, it distends them and pushes them against the inside of the chest walls, the heart, and great blood-vessels and contents of the thorax. The pressure

thus exerted is not equal to that of the external air, since some of the total air pressure on the inside of the lungs is used up in overcoming their elasticity (equal to six millimetres of mercury), and it is only the residue, equal to a column of mercury 754 millimetres high, which pushes the lungs against the heart and great vessels. The actual pressure then on the heart walls, normally, is equal to 754 millimetres of mercury.

How is this pressure changed at an altitude, say of 10,000 feet? The total pressure of the air at that altitude would be equal to a column of mercury 501 millimetres high. The pressure, then, upon the lungs and whole body would be lessened. But, as the person is breathing the same air as that around him, the respiratory act would be unchanged. Prof. Martin and the author found that neither the frequency nor depth of the respiratory act were changed in an animal placed in a rarefied atmosphere. There would be (as, again, we found) a slight fall in blood pressure, which would be very temporary, for the circulation being a system of closed tubes, is but little, if any, affected, and the pressure rises at once to the normal, owing to increased vasomotor action. There would be no increase of negative intrathoracic pressure. Finally, the only organ directly affected at moderate altitudes (of course, at very high altitudes, 12,000 to 18,000 feet, the dyspnœa from want of oxygen would be felt), say from 3,000 to 10,000 feet, would be the heart, and for this reason. As we have said, the pressure on the heart usually is equal to 754 millimetres of mercury. At an altitude of 10,000 feet, however, the general pressure being 501 millimetres, the pressure of the heart would be only 495 millimetres of mercury. Now the arterial pressure,

and, therefore, the intracardiac pressure would be the same as when the pressure on the heart walls was 754 millimetres. But one result could follow such conditions, and that would be a stretching and dilatation of the heart walls—especially of the right side—and this is precisely what is found in many of those who go into high altitudes for their health, or for other reasons, and what we found in experiments upon animals. The heart, weak and diseased possibly beforehand, is unable to stand such strain as is put upon its dilated walls, and so we find its contractions weak, intermittent, and irregular—we have the murmurs, the syncope, the dyspnœa, which are often so marked in high altitudes.

Of course, the want of oxygen is a great cause of the dyspnœa in very high altitudes, but we are quite justified, on physiological grounds, in saying that at the altitudes to which persons are ordinarily sent—three, four, six, seven, or even ten thousand feet—the hæmoglobin can take up all the oxygen the system needs.

Finally, then, and speaking purely from an experimental standpoint, I hold that there is danger to the weak and diseased heart, not only in the use of such apparatus as the pneumatic cabinet, but also in high altitudes; in which conclusion I am supported by the clinical experience of Prof. Loomis, and of Dr. Solly, of Colorado Springs.