THE DIAGNOSTIC AND PROGNOSTIC VALUE OF BREATH-HOLDING TEST*

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How long can you hold your breath?

This seems an almost absurd question, and yet cumulative experience is showing that the breath-holding capacity and the apneic pause can be made of the most practical diagnostic and prognostic value in the routine practice of medicine. In simplicity, scope and utility, respiratory tests may be classified with the taking of the temperature, pulse, and respiratory rates.

It is nearly two centuries since Valsalva's classical experiment, in 1740, showed the marked and easily recognizable effect of sustained *forced expiration* on the pulse beat, and therefore on cardiac efficiency, when the respiratory passages are forcibly closed. A hundred years later, in 1838, Johannes Mueller of Berlin added the complementary experiment of the effects on the pulse of *forced inspiration* under similar conditions. These two experiments early indicated the intimate relationship of the cardio-respiratory mechanism in health and disease.

In 1902, Sabrazes, of Bordeaux, collected the literature on the voluntary apneic pause, and in an investigation of his own, by means of a split-second watch, he found the average *normal voluntary apneic interval* (between breaths) to be from 20 to 25 seconds in duration; while an interval of 30 to 35 seconds was exceptional. His further observations showed that the voluntary apneic pause varied in different pathological states, and in *mitral insufficiency with asystole,* it was not in excess of from 5 to 10 seconds.

In making his test, Sabrazes used no preliminary forced inspiration, for apnea, under such circumstances, has it own laws. In mitral insufficiency, after compensation had been re-established by digitalis, Sabrazes found the apneic interval prolonged to from 10 to 15 seconds. Sabrazes also reported that the blood pressure rose during the apneic pause, and that the sign of Muset—oscillation of the head—appeared more noticeable during the pause. Of all pathological conditions studied, tuberculosis showed the least response to the test, as even in advanced cases of pulmonary tuberculosis the apneic interval was not so noticeably shortened. In the study of miscellaneous individuals, Sabrazes found that *deep inspiration* or *involuntary expiration,* might lengthen the pause to from 40 to 50 seconds.

More recently, Binet and Bourgeois of Paris have fixed the average normal interval after deep inspiration at 50 seconds. In the French Aviation Service tests, candidates whose pause was under 45 seconds were deemed unsuited for respiration at high altitudes. Unfortunately, Binet and Bourgeois do not allude to the relation of voluntary apnea to disease, tolerance of anesthesia, or to the prolonged interval in special individuals, such as divers.

In 1914, Stange, of Petrograd, recommended the so-called *respiratory test* as the best indication of the patient's cardiac condition for withstanding operation under general anesthesia. Stange found that average healthy persons could hold the breath for from 45 to 50 seconds, while patients with weak heart muscles could do so but for from 10 to 20 seconds. While Stange seemed to have had no suspicion that the breath-holding test was based on an apnea due to *decreased alkaline reserve,* nevertheless he reported observations on a number of chronic diseases, in which he found the duration of the apneic pause shortened in about the degree in which so-called *acidosis* is known to occur from the results of other observers; notably, Yandell Henderson, especially in his studies of altitude sickness.

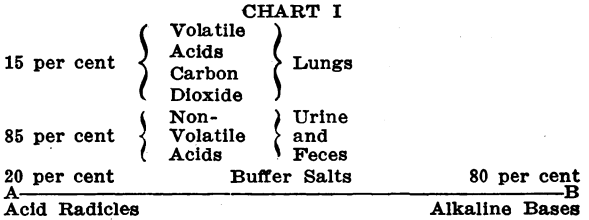
Lewis, Ryffel, Wolf, Cotton, and Barcroft have also shown that the dyspnea of nephritis is due to an acidosis (hypo-alkalinity) essentially like that developed in normal people at high altitudes. Kenway, Pembrey, and Poulton have also found that by following the alveolar carbon dioxide content in diabetics, a warning drop in its tension indicates the approach of coma as long as forty-eight hours beforehand, and much sooner than any other available test.

A thorough understanding and proper evaluation of the breath-holding test, as a diagnostic and prognostic indication in abnormal conditions, depends on the realization that the breath-holding capacity and apneic pause involve the oxygen absorption and carbon dioxide elimination phases of pulmonary respiration as well as the balance of blood and tissue reserve alkalinity. This should be emphasized, especially since Yandell Henderson has shown that three-fourths of the actual breathing of the body is tissue, and only one-fourth pulmonary respiration.

Respiratory alterations, due primarily to disturbances of oxygen absorption, should be classified under the term anoxemia; while those dependent on carbon dioxide elimination should be considered under acidemia; while certain conditions affecting both phases of the respiratory cycle as well as blood and tissue alkaline reserve, should be considered under acapnia. The breath-holding test is all the more valuable, however, because it represents in its ultimate analysis the response of the body to the oxygen-carbon dioxide balance in the alveolar air, blood, and tissues under any and all conditions of health and disease.

Mercur, of Pittsburgh, has recently presented a diagrammatic concept of hypo-alkalinity in relation to disease and the breath-holding test that is very illuminating. He uses the accompanying diagram (Chart I), suggested by blood chemistry studies, to illustrate his therapeutic concept:

CHART I



Imagine all the blood of the body to be represented by the straight line A-B. The blood is a

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fluid approximately of 80 per cent alkaline bases and 20 per cent acid radicles—a proportion held in remarkable balance, according to Rowntree, by the *buffer salts* of the blood. In *health* the buffer, irrespective of metabolic indiscretions, promptly takes up an excess on either side, and the delicate balance is not materially changed. When *disease conditions* affect the buffer mechanism, there is an acidosis on the one hand, or an alkalosis on the other—either of which may precipitate serious conditions, especially if either is prolonged or severe.

The 20 per cent of acid radicles represent 15 per cent of volatile acids, thrown off principally through the lungs as carbon dioxide and 85 per cent of non-volatile acids, excreted principally through the urine and very slightly through the feces.

According to Mercur, this diagrammatic concept makes it quite evident that if the non-volatile acids, as a result of *deficient elimination,* accumulate in the blood, the volatile acids will be thereby compressed by the lungs and will have to be increased, and thus the breath-holding capacity will be more or less decreased. This is the rational explanation of the clinical value of the breath-holding test.

It also explains why the breath-holding test may be utilized to *anticipate* the diagnostic and prognostic indications of blood and urine analyses, when retention products are being thrown into the blood stream.

Means further illustrates the acid-base equilibrium in disease by the accompanying formula and diagram, originally suggested by L. J. Henderson, H. W. Haggard, and Yandell Henderson.

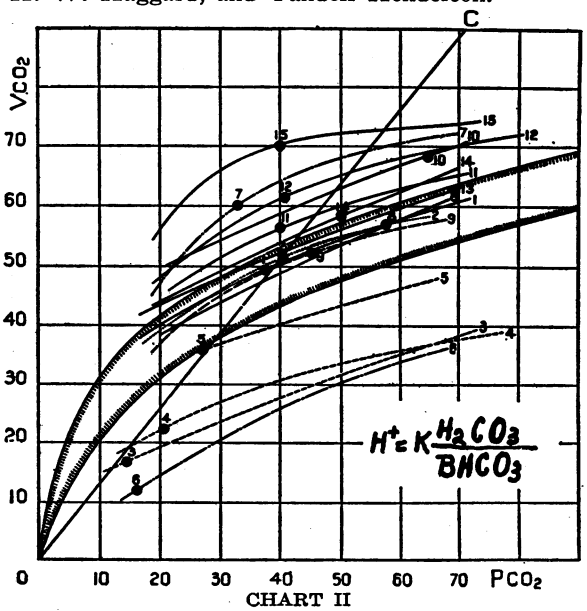

CHART II

The formula is based on the principle that the reaction of the blood is dependent chiefly on the ratio of free carbonic acid to bicarbonate. H† is the concentration of ionized hydrogen, H_2CO_3 the concentration of the free carbonic acid, and the $BHCO_3$ the concentration of bicarbonate, and K a constant. The fraction $H_2CO_3/BHCO_3$ ordinarily has a value of approximately 1/20.

Changes in this ratio denote changes in H†, that is in the blood reaction, while changes in the actual magnitude of the denominator denote changes in available alkali of the blood, and in the numerator changes in the carbon dioxide tension. Knowledge of the two terms of the ratio in any given blood reveal the two most fundamental facts of acidosis.

For clinical purposes Means considers the simplest way of arriving at the value of the ratio, and the two terms of the fraction is to secure data for plotting the so-called carbon dioxide dissociation curve by the method of Christansen, Douglas, and Haldane. Means has found the carbon dioxide dissociation curves of normal human blood to be within the shaded area. When both are normal they cross the line O-C at the points A^1 and A^2, respectively. Any marked change from health to disease would be indicated by a deviation to the alkaline or acid side of the line O-C, or as a response to therapy, and both would be accompanied by diagnostic and prognostic alterations in the breath-holding test, as governed by the oxygen-carbon dioxide reaction of the respiratory mechanism.

The numbered curves in Means' diagram represent plotted carbon dioxide dissociation curves from blood analyses of actual patients, normal (1, 2) or suffering from diabetic acidosis (3, 4, 5), nephritic acidosis (6, 7), pneumonias (8, 9, 10), cebral hemorrhage (11), anemias (12, 13, 15) and tetany (14). The tendency to acidosis and alkalosis is very apparent.

Yandell Henderson's directions for making the breath-holding test are as follows:

1. Sit quiet for five minutes.
2. Take a full, but not too deep breath.
3. Hold it with mouth and nostrils closed.
4. Note time in seconds.

Diagrammatically, and for all practical purposes, a breath-holding test (Stange) of 50 to 70 seconds is above the average normal, while 45 to 50 seconds represents the test of the average healthy person; 35 to 45 seconds indicates beginning acidemia; 25 to 35 seconds, mild acidosis; 15 to 25 seconds, frank acidosis; and 10 to 15 seconds an imperiling acidosis.

According to the apneic pause, split-seconds watch method of Sabrasez, the following intervals represent the same degrees of normality and depletion of alkaline reserve: 30 to 50 seconds, exceptional; 25 to 30 seconds, average normal; 20 to 25 seconds, beginning acidemia; 15 to 20 seconds, mild acidosis; 10 to 15 seconds, frank acidosis; and 5 to 10 seconds, severe acidosis. The difference in time in the two tests is due to the period of time available for the absorption of the deep breath in the Stange test (Chart III).

Involuntary expiration or inspiration during the making of the test, by either method may lengthen the interval, and must be guarded against. The tests may be made more severe by evaluating the average time of several successive tests.

It is of interest also to note, in passing, that

CHART III

Breath-Holding Stange (Seconds)	Breath-Holding Sabrazes (Seconds)	Condition	Vital Capacity	Pulse Rate	Resp Rate	Pulse Press.	Heart Load Per cent	Hemoglobin	Oxygen Need Per cent
45-50	25-30	Normal	3500 cc	72	16	40	50	100	20
35-45	20-25	Acidemia	3000 cc	80-100	24	60		90	30
25-35	15-20	Mild Acidosis	2500 cc	100-120	32	80		80	40
15-25	10-15	Acidosis	2000 cc	120-140	40	100		70	50
10-15	5-10	Severe Acidosis	1500 cc	140-160	48	120	200	60	60

the breath-holding test bears a definite and direct ratio to vital capacity as gauged by the spirometer. Dryer and others, notably Wittich, Peabody, and their co-workers, have noted that, while especial fitness and physical training may *increase* vital capacity by 30 per cent above normal, physical fatigue and weakness *decrease* it by about the same amount; *whereas heart disease and other pathological conditions may show a vital capacity 75 per cent below normal.*

Thus, a normal breath-holding test would predicate a minimum vital capacity of 3500 cc. in the average-sized male, and 3000 cc. in the female; whereas, a breath-holding test of 10 to 15 seconds (Stange) would indicate a vital capacity of 1500 cc. or less. As the diseased condition becomes more severe, the breath-holding capacity becomes shorter and the vital capacity falls; and when the patient improves, both again approach normal. Clinically, vital capacity figures bear an approximate relation of 1 to 100 to the Stange test in the male, and the Sabrasez test in the female.

Patients with cardiac disease can lead a normal life if their vital capacity is above 90 per cent; a vital capacity between 70 and 90 per cent means a restricted life and slight amount of work. Patients whose vital capacity is less than 70 per cent are dyspneic on very moderate exertion, and when the vital capacity falls to 40 per cent, dyspnea is pronounced and decompensation is present, or readily occurs.

At this point it should again be emphasized that all *breathlessness* is not necessarily of cardiac origin. A year ago, while discussing the breath-holding test with the physical director of a well-known athletic club, he informed me that he had a group of some twelve business men in his classes, all over forty-five years of age, who got breathless after brief exercise, but on examination by their doctors had been pronounced cardiacly sound. He was at a loss to account for their dyspnea.

Such patients should be given Cornell's tests for the acidemia of chronic nephritis as a prelude to a more searching examination. Cornell has shown that in ninety-five out of one hundred cases, in which nephritis has been present under three years, there is a non-cardiac form of dyspnea associated with mild exertion. His charting of the average respiratory and pulse rate response to exercise in one hundred cases of early chronic nephritis is of very pertinent interest (Chart IV). The tardiness with which the respirations approach their rest-rate is readily seen to be in decided contrast with the quicker and normal slowing of the heart rate.

Cornell has found that although not more than 20 per cent of patients make a complaint of this dyspnea, since in mild cases it is not troublesome, 95 per cent of early nephritics will admit having it when questioned, and its presence can be proved by testing the response of the cardio-respiratory rate-ratio to exercise.

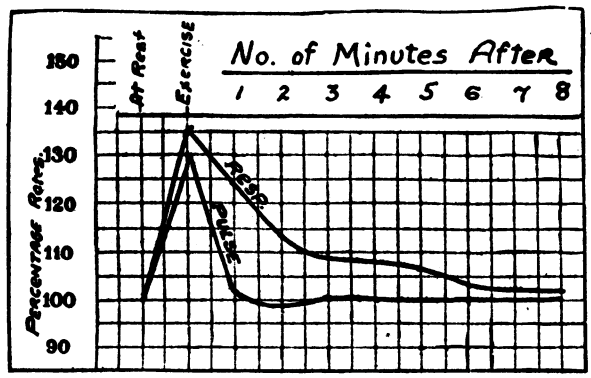

CHART IV

Nephritic dyspnea has two distinguishing characteristics:

1. It has no accompanying cyanosis.
2. It is speedily removed by the oral administration of sodium bicarbonate. Usually, after 4 (20 gr.) doses of bicarbonate, the most extreme type of nephritic dyspnea subsides and disappears.

In fully half the cases studied, Cornell has noted that color changes (red, blue, green or varigated) of the electric bulb are described by the patients during the dyspneic period after exercise, and that alkaline therapy obviates the delusion.

The degree of dyspnea does not always coincide with the *phthalein* output, and seems more dependent on the degree of acidemia and the sensitiveness of the respiratory center. Clinically, this cardio-respiratory test is presumptive evidence of incipient nephritis, even in the absence of albumin, and it also indicates alkaline therapy in nephritics, whose distressing dyspnea fails to respond to digitalis.

Accumulating clinical observations are also showing that the breath-holding test may be charted in relation to pulse rate, pulse pressure, respiratory rate, hemoglobin index, and oxygen need. Some of these relations are briefly shown in Chart V.

CHART V

Case No.	Condition	Breath-holding Test	Pulse Rate	Blood Pressure Sys.	Blood Pressure Dias.	Pulse Pressure	Hemoglobin Index	Oxygen Increase Per cent
1.	Diabetes	16 sec.	108				80	
2.	Thyroid	10 sec.	110	142	95	47	70	50
3.	Thyroid	10 sec.	150	145	67	78	70	100
4.	———	25 sec.	112	123	82	41	70	
5.	Anemia	15 sec.	96	152	74	78	60	
6.	Mitral Regurgitation	40 sec.	92	115	65	50	(Compensating heart)	
7.	Mitral Murmur	14 sec.	110	182	62	120	70	100
8.	Tuberculosis	12 sec.	110	98	78	20	70	
9.	———	15 sec.	100	104	104	40	80	
10.	Pus Diabetes	20 sec.	104	120	62	58	70	
11.	5½ per cent Sugar	35 sec.	(Operation for gangrene)					
12.	Sepsis (Bad teeth; large amounts of pus; giant-cell epulis of lower jaw)	12 sec.	94	148	100	48		
13.	Acute Infective Cellulitis	15 sec.	120 (Temperature 102.4°—For every degree above normal temperature the baso-metabolic rate is increased 10 per cent.)					

At the request of the National Anesthesia Research Society, W. I. Jones, secretary of the Research Committee, subjected the daily run of patients coming to his dental clinic for extractions to the breath-holding test, and ten of the cases charted occurred in the first fifty patients examined, while the other three cases included were operated on at the hospital during the same period.

As patients presenting for extractions are commonly considered to be in average health, an incidence of one seriously handicapped patient in every five in so short a series is certainly provocative of thought.

Note the striking difference in the two diabetic cases (1 and 11): the latter with considerable sugar in the urine, and a breath-holding test of 35 seconds, indicating good renal permeability and fair surgical risk for an amputation; the former showing a breath-holding test of 16 seconds and being a very grave anesthetic risk, although walking into the clinic for a simple extraction.

Note the three thyroid cases (2, 3, and 4), and see how the breath-holding test predicates the other reactions of a more searching examination to determine surgical risk and prognosis. Consider the cardiac cases (5, 6, and 7), and contrast the mitral regurgitation patient, whose booming murmur seemingly could be heard across the room, with the more serious anemic and the very grave mitral risk with a heart load of 200 per cent. The breath-holding test again points the finger of warning. The tubercular cases (8 and 9) were anything but good risks for operation or anesthesia, and yet these patients had little or no appreciation of the gravity of their condition. It is also interesting to note the effects of pus, sepsis, and cellulitis on the breath-holding capacity. These relations, clinically noted by Jones, may be diagrammatically presented as in Chart III.

In the recent influenza epidemics it has often been very difficult to make a certain prognosis in broncho-pneumonias. In this connection Bié, of Copenhagen, studied one thousand cases of influenzal broncho-pneumonia and published his analyses, and more particularly his conclusions regarding the 234 deaths in the series of cases. This large loss of life was due in some degree to the fact that many of the women were pregnant, for the death rate is 2.5 times as great in the gravid. There were also fifty-five patients with heart disease in which the mortality rate was very heavy. A toxic factor, as shown by the presence of jaundice, albuminuria, and the behavior of the temperature, pulse and respiration, means a bad prognosis. Bié found that half of those who breathed thirty-four and upward on admission died, while if the respiratory rate was below thirty-four recovery was the usual outcome. This difference was not dependent on whether the pneumonia was single or double, but inhered in each form, although naturally the prognosis is much better for single pneumonia. In all pneumonias with a respiratory rate above 40 the prognosis is grave, and the more rapid the breathing the worse the outlook.

Hirschfelder, of Minneapolis, has recently stressed the explanation why influenzal pneumonia is so fatal. In influenzal pneumonia the cyanosis is not due to heart failure, but is produced in much the same way as is the cyanosis of congenital heart disease, by the entrance of unaerated blood into the left side of the heart. W. C. Stadie has shown that, whereas in ordinary lobar pneumonia very little blood flows through the consolidated area, owing to increased resistance, and most of the blood flows through the normal parts of the lungs, where it is well oxidized, in influenzal pneumonia there is little resistance to the blood-flow through the congested areas. So the blood flows through without being oxidized and thus enters the left auricle, where it is mixed with aerated blood; and so a mixture of blue and red blood is pumped to the tissues. Thus, such cyanosis is not an expression of heart failure, as it is in the ordinary lobar pneumonia, but an expression of the kind of mixed blood which is coming into the heart from the lungs. It can be readily seen from this explanation why the respiratory rate becomes prognostic.

Very recently Gilbert Fitz-Patrick has reported on the results of the breath-holding test in 871 obstetrical cases, primiparas and multiparas of all ages, and including the various complications found before and subsequent to pregnancy.

1. The average parturient woman has a breath-holding pause of 25 seconds (Sabrazes).
2. The shortest breath-holding test, 3 seconds, was found in a primipara, 44 years of age, weighing 180 pounds, who had a cardio-renal insufficiency, with general venous stasis, and who gave a history of rose fever in her younger life.
3. The longest breath-holding test, 65 seconds, was found in a primipara, 24 years of age, who holds the hurdling championship for girl athletes in her State.
4. The average cardio-nephritic pregnant woman has a breath-holding test of 11 seconds.
5. The toxemias of pregnancy due to faulty excretion showed a test of 18 seconds, while those due to faulty secretion showed a test of 15 seconds. The average vital capacity in 180 obstetrical cases was 135 cubic inches.

Fitz-Patrick concludes (1) that the average pregnant woman has a breath-holding test (Sabrazes) of at least 25 seconds. Any reduction below this point demands an explanation. (2) And that the pregnant woman with an apneic pause of 15 seconds has an organic lesion, is a poor obstetro-surgical risk, and should be given an anesthesia only by a professional anesthetist, nitrous oxid-oxygen being the anesthetic of choice, and the individual oxygen need being determined and supplied.

In conclusion, the breath-holding test in connection with the patient's response to other tests, when indicated, may be of diagnostic and prognostic significance, and may not only indicate therapeutic measures, but may also serve as a guiding sign to their effectiveness. Used routinely at the bedside, in the office, and in the hospital, it can be made one of the most valuable assets of daily practice.

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