resulting from abnormal metabolism may have a similar effect. These intoxications produce early only functional disorders chiefly referable to the nervous system. When persistent they induce organic changes affecting the bloodvessels and kidneys.

In a subject so confused as that of hypertension in its relation to arteriosclerosis and nephritis it is best that our ideas remain fluid rather than crystallized into conceptions based as they must be at present on assumptions and speculations. We need first of all a larger range of clinical facts.

LONG-CONTINUED OBSERVATIONS ON THE VITAL CAPACITY IN HEALTH AND HEART DISEASE.*

By Joseph H. Pratt, M. D.,
BOSTON.

Mr. John Hutchinson, surgeon, the inventor of the spirometer, applied the term "vital capacity" of the lungs to the "greatest voluntary expiration following the deepest inspiration." He showed definitely that the vital capacity varied with height, weight, age and disease. As his statistics have not been republished by any recent writer and as his series of observations is much the largest recorded I have prepared two tables from figures given in his original communication.[1]

TABLE I.—INFLUENCE OF HEIGHT ON VITAL CAPACITY HUTCHINSON'S OBSERVATIONS ON 1923 MALES.

Feet and inches.	Centimeters	Vital capacity in centimeters.
5 feet to 5 feet 2 inches	150 to 155 cm.	2900
5 " 2 inches to 5 feet 4 inches	155 to 160	3150
5 " 4 " 5 " 6 "	160 to 165	3400
5 " 6 " 5 " 8 "	165 to 170	3725
5 " 8 " 5 " 10 "	170 to 175	3950
5 " 10 " 6 "	175 to 180	4300

In Table I the effect of increased height in increasing the vital capacity is clearly shown. Hutchinson tested 2130 individuals, of which all but 26 were males. There were 60 cases of disease, chiefly phthisis pulmonum. The persons examined were all sorts and conditions of men including gentlemen, paupers, artisans, sailors, soldiers, policemen, printers and pugilists.

* Read at the Meeting of the Association of American Physicians, Washington, D. C., May, 1922.

[1] Hutchinson, J.: On the capacity of the lungs and on the respiratory functions, with a view of establishing a precise and easy method of detecting disease by the spirometer, Medico-Chirurgical Transactions, London, 1840, 29, 137–252.

In a series of 1775 cases he studied the effect of age on vital capacity. He found the maximum vital capacity between the ages of twenty-five and thirty-five.

TABLE II.—INFLUENCE OF AGE ON VITAL CAPACITY (HUTCHINSON).

Age.	Cases.	Vital capacity in centimeters.
18 to 25	774	3425
28 to 35	589	3500
35 to 45	264	3225
45 to 55	92	3050
55 to 65	56	2850

Between the age of fifteen and twenty-five it was almost as great. If 3500 the average vital capacity between twenty-five and thirty-five be taken as 100 per cent, the average in the preceding period of ten years will be 98 per cent. A marked fall occurred after forty-five years of age according to his observations, and between fifty-five and sixty-five it dropped to 71 per cent. It should be noted that over three-quarters of his cases were under the age of thirty-five. It is important in considering the relation of height to vital capacity to know that most of his determinations were made on young men.

Peabody and Wentworth[2] reported in 1917 the vital capacity in a series of 96 normal men, and 44 normal women. They were chiefly medical students and nurses. The average age was probably between twenty and twenty-five years. Their figures for males were considerably higher than those obtained by Hutchinson (see Table I). The average vital capacity for men 6 feet tall or over was 5100. For men 5 feet 8¼ inches to 6 feet in height it was 4800, while men less than 5 feet 8½ inches tall and over 5 feet 3 inches had an average vital capacity of 4000.

Peabody and Wentworth showed the influence of sex on vital capacity, confirming observations of Arnold, made many years before. It is lower in women than in men of the same height. The average vital capacity in women over 5 feet 6 inches tall was 3275; in women varying in height from 5 feet 4 inches up to and including 5 feet 6 inches it was 3050; in still shorter women, 5 feet 1 inch to 5 feet 4 inches the average was 2825 cc.

West's studies show strikingly the lower vital capacity in women. In 75 out of 85 normal men he examined the vital capacity was 4000 or more; while 42 in a series of 44 women had a vital capacity below 4000 cc.

Hewlett and Jackson,[4] in their examination of 400 healthy male students of Leland Stanford, Jr., University found the average vital capacity to be the same as that obtained by West in his study

[2] Arch. Int. Med., 1917, 20, 443.
[3] Ueber die Athmungsgrösse des Menschen, Heidelberg, 1855.
[4] Arch. Int. Med., 1922, 29, 515.

of Harvard Medical students. Oxford students, as Schuster[5] showed had an average vital capacity about 7 per cent lower and Hutchinson's subjects more than 20 per cent lower.

METHODS EMPLOYED IN THE PRESENT STUDY. I have used the wet spirometer made by the Narragansett Machine Company of Providence, R. I. This has been tested and found to give trustworthy readings.

The observations recorded have been made by me personally. I have taken pains to instruct each person examined in the use of the instrument. Three or more trials were made. Each one was encouraged to inhale and exhale the maximum amount of air. The highest reading was taken as the vital capacity. Each subject was watched carefully during the test and any error noted was corrected. Toward the end of expiration some persons draw in a little air, and blow the spirometer to a higher level than is possible for them with a single inspiration. This error can be eliminated by rejecting all readings in which the upward movement of the spirometer is not continuous.

Most of the heights recorded in this paper were taken without removing the shoes. This increases the height of men usually from ¾ of an inch to a full inch, but with women the shoes may increase the height from 1 inch to 2½ inches. As the heels vary so much in height I now take this measurement in stocking feet. The heights given by Hutchinson include the shoes, while Peabody and Wentworth's are for bare feet. The weight of my subjects include the clothes. I allow for this one-eighteenth of the weight for males and one-twentieth for females.

As a standard I have taken the one recommended by West[6] as he found a more constant relationship existed between the vital capacity and the body surface area than between the vital capacity and the height, weight or chest volume. It is determined by dividing the vital capacity by the surface area of the body. The latter is calculated by Du Bois' formula. By means of the graphic chart published by Du Bois and Du Bois[7] the surface area is easily determined when the height and weight are known.

According to West the normal vital capacity for men is 2.5 liters per square meter of body surface, for women 2 liters. Such a standard, as Hewlett and Jackson point out, is based on the assumption that there is a simple relation between the vital capacity and some body measurement. Hewlett and Jackson have obtained a formula for calculating the vital capacity from the height and surface area of the body. It introduces an additional constant as is usual in statistical formulas but is applicable only to college students. I have studied the effect of age on vital capacity in a

[5] Biometrika, 1911, 8, 40.
[6] Arch. Int. Med., 1920, 25, 306.
[7] Ibid., 1916, 17, 863.

series of 100 men who showed no evidence of disease of the heart or other organs at the time of their examination. The results are presented in Table III.

TABLE III.—PERSONAL OBSERVATIONS ON THE INFLUENCE OF AGE ON VITAL CAPACITY IN 100 NORMAL MEN.

Age.	No. of persons.	Average height in cm.	Weight in kgm.	Body surface area in square meters.	Average vital capacity in cc.	Average per cent of West's standard.
10 to 20	0	157	47.0	1.47	3125	82
20 to 30	14	173	58.6	1.72	4500	105
30 to 40	23	166	67.1	1.75	3950	00
40 to 50	20	171	69.0	1.81	3775	83
50 to 60	20	170	73.9	1.85	3825	83
60 to 70	11	169	69.8	1.80	3300	73
70 to 80	3	172	69.9	1.82	2525	56

The vital capacity is definitely influenced by the age of the subject. It reaches its maximum in the third decade. The table shows clearly that in the fourth decade the average vital capacity falls slightly. Hutchinson found that the decline began between the age of thirty-five and forty-five years. My averages at all ages are higher than those obtained by Hutchinson. For this I have no explanation to offer. A marked lowering in the vital capacity was not noted in my series until after the age of sixty. The 11 men who were between the age of sixty and seventy when tested showed an average vital capacity of only 73 per cent of West's standard. Above the age of seventy I have examined only 3 men with apparently normal hearts. They had no shortness of breath on exertion, but their vital capacity was much reduced, being only 56 per cent of the normal. The number of cases in this decade is too small to permit of drawing any conclusions from the figures given. When more persons above seventy are examined the average percentage may be lower or higher. I think it will probably be higher, in light of the following observations. One of the men included in the seventh decade in this table has now reached the age of seventy and his percentage of the normal is 65. I have seen a patient of seventy-two with definite cardiosclerosis whose vital capacity was 3050 cc which was 56 per cent of the standard. I know of a vigorous man of eighty whose vital capacity was 3200, which was 70 per cent of his normal.

Individuals may vary considerably from the average vital capacity for their age, height and weight, and yet no disease be present. Hutchinson thought from his study that a reduction from the normal was a valuable sign in the diagnosis of early phthisis. Subsequent investigators found that the individual variations were too great to make the vital capacity determination of value in the recognition of the early stage of this disease. Furthermore the

vital capacity test of the same person may vary from time to time. I have recorded the amount of variation of the vital capacity in 9 persons with apparently healthy hearts and lungs over periods ranging from fourteen months to three years and four months (Table IV). Although the 7 men in this group were urged to make a maximum exertion at each test, and they apparently did so, the variation was considerable, ranging from 100 to 550 cc.

TABLE IV.—VARIATION OF THE VITAL CAPACITY DETERMINATIONS OVER PERIODS RANGING FROM ONE TO THREE YEARS.

Name.	Duration of observation.	No. of tests.	Vital capacity.		Variation. cc.
			Maximum cc.	Minimum. cc.	
Mr. H.	3 years 5 months (Nov. 17, 1918 Apr. 5, 1922)	8	2825	2500	325
Mr. L. D.	3 years 4 months (Dec. 3, 1918 Apr. 10, 1922)	3	3650	3100	550
Mr. A. K.	2 years 8 months (Sept. 5, 1918 May 20, 1921)	3	4500	4300	200
Mr. F. D.	2 years 4 months (Dec. 30, 1910 May 8, 1922)	8	3300	3000	300
Dr. R.	2 years 3 months (Dec. 13, 1919 Apr. 6, 1922)	9	3700	3300	400
Miss E. L.	2 years 2 months (July 1, 1919 Aug. 30, 1921)	3	1800	1775	25
Mrs. T.	2 years 2 months (Apr. 15, 1920 June 7, 1922)	8	1450	1400	50
Mr. F. F.	1 year 7 months (Nov. 11, 1920 June 2, 1922)	3	3350	2900	450
Mr. R. A.	1 year 2 months (Jan. 20, 1919 Mar. 25, 1920)	2	2650	4500	150

In the period covered by these tests there was no decrease that could be attributed to increasing age. The age of 1 of the men was sixty-six when his vital capacity was first determined. It was then 2800 cc, at sixty-eight it was 2550, at sixty-nine it had risen to 2700. A year later when just seventy years of age for the first time in the series of eight tests he was able to expel 2825 cc of air into the spirometer. In none of these people was there either a steady fall or rise in the vital capacity. The change as shown by plotting the curve was an irregular fluctuation. The vital capacity in the different tests made on the 2 women showed very little variation. In 1 of these the range was 50 cc and in the other only 25 cc.

On 1 person my observations of the vital capacity span a period of sixteen years. The tests were all made on the same spirometer. The patient, S. R., had early active pulmonary tuberculosis in 1905. At the time the first determination of the vital capacity was made there was dulness to the second rib of the right lung

anteriorly and rales were present over this area. His vital capacity was then 74 per cent of the normal. He was thirty-four years of age. The average vital capacity in the fourth decade in my series of 23 was 90 per cent. The lowest percentage recorded by West among his 85 normal men was 82 per cent, but West's man with this low percentage was only twenty-three years of age. It is impossible to decide whether the vital capacity was normal for my patient or reduced by tuberculous infiltration of the lungs. In April, 1906,

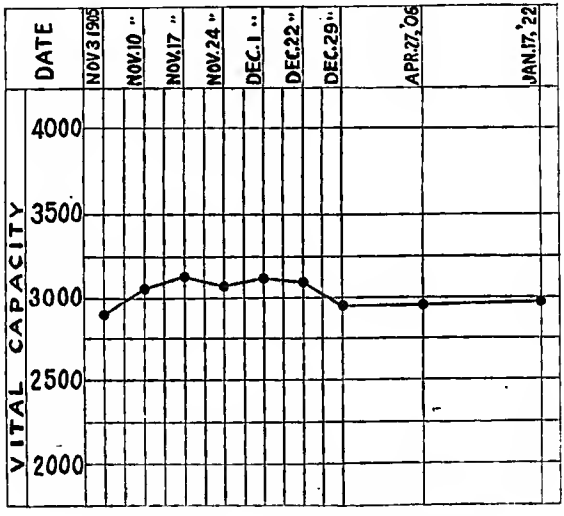

CHART I.—Vital capacity record covering a span of sixteen years.

the symptoms and signs of active pulmonary tuberculosis had disappeared, but his vital capacity was practically the same, 75 per cent. Sixteen years later it was 72 per cent of the normal. At his present age of fifty it is still low for his years. He has been well and working since his recovery in the spring of 1906 to the time the last test was made, January, 1922. The nine observations made on this man showed a variation in the vital capacity of only 125 cc (Chart I).

Peabody[8] showed that the vital capacity was low in cardiac insufficiency because of the inability to increase the depth of breath-

[8] Arch. Int. Med., 1917, 20, 433.

ing in the normal manner. He and his associates[2] found that when the patients improved under treatment there was a rapid and marked increase in the vital capacity, and also that an increase of the cardiac weakness was accompanied by a further fall in the vital capacity.

I have compared the vital capacity in 100 persons, both men and women who had organic heart disease with 100 other persons who had normal hearts. The results of this analysis are presented graphically in Chart II.

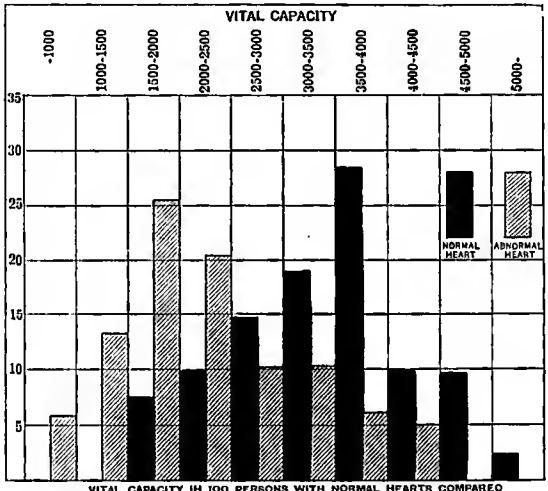

CHART II

The mean vital capacity of the 100 persons with normal hearts was between 3500 cc and 4000 cc, while the mean for these with abnormal hearts was between 1500 cc and 2000 cc. No one with a healthy heart had a vital capacity below 1500 while 18 per cent of those with heart disease had a vital capacity below this level. No one with a diseased heart had a vital capacity above 4500 cc and only 5 of the 100 above 4000 cc; 11 per cent of those with healthy hearts had a vital capacity above 4500.

Seventy-seven of the 100 persons with abnormal hearts had cardiac insufficiency of the congestive type. Dyspnea on exertion or edema

[2] McClure, C. W. and Peabody, F. W.: Jour. Am. Med. Assn., 1917, 69, 1954.

was present. Sixteen presented no evidence of cardiac weakness. The average vital capacity of the males in the first group was between 2500 and 3000, of the women in this group between 1500 and 2000 cc. In the second group, without evidence of cardiac weakness, the average vital capacity among males was 1000 cc more than in the first group, but among the females the average was just as low, 1500 cc to 2000 cc, as in group one. Seven patients, all males, had angina without breathlessness. Their average vital capacity was between 3500 cc and 4000 cc.

Since the spring of 1917 I have followed the variation in the vital capacity of all cases of heart disease that have been under my care. When cardiac insufficiency of the congestive type has existed the vital capacity has followed closely the change in the clinical condition. It has proved to be a good index of the amount of reserve power possessed by the heart.

In 4 cases the vital capacity has been determined over periods ranging from nineteen months to nearly five years. The clinical condition in cardiac insufficiency is more clearly shown by charts of the vital capacity readings than by the graphic representation of any other measurement, or any other record such as a statement of the amount of effort that can be made without dyspnea.

CASE I.—James M., aged forty-four years in 1917. Seen in the Cardiac Clinic of the Massachusetts General Hospital. He had mitral stenosis of rheumatic origin with auricular fibrillation. When the vital capacity was first determined in 1917 he could not walk 100 yards without stopping for breath. Apex rate 125, radial rate 105. His vital capacity was 1300 cc. which was 32 per cent of the normal (West's standard). The next week he returned to the out-patient department. He had taken 2.1 grams of active digitalis leaf. He felt better. Two days after beginning to take the digitalis (0.1 gram, 3 daily) he had a free diuresis. After this occurred he had less dyspnea. His vital capacity had increased to 41 per cent.

March 1, 1917. Patient has felt quite well the past few days. Heart rate 80, deficit 10. Vital capacity 48 per cent. Resumed work today as assistant agent at railroad station, March 17. Has worked steadily for two weeks. Can now walk a quarter of a mile without stopping. Heart rate: Apex 52, radial 52.

Vital capacity continued to rise under digitalis treatment as shown by the chart until on April 7, 1917, it was 54 per cent. On May 19, 1917, the vital capacity was 52 per cent. He stated at this time that he could walk a mile without shortness of breath. Pulse 66. No deficit.

When next seen over two years later on November 31, 1919, he presented evidence of severe cardiac weakness. He said he was unable to walk 200 yards without stopping. For two months his

exercise had been restricted by breathlessness. He coughed a good deal and he said that he slept poorly and complained of pain in the region of the liver. Slight swelling of the ankles had been noticed several days previously. The liver was swollen and tender and there was fluid in the chest (hydrothorax). The vital capacity had fallen to 1000 cc or 24 per cent. The pulse was 72, no deficit. During the following week he took 3.3 grams of digitalis leaf. His vital capacity rose to 37 per cent. Pulse 56, no deficit. December 29, 1919. Marked dyspnea even at rest. Has taken no digitalis for four weeks as his local doctor said he would be better off without it. Apex rate 108, radial 76.

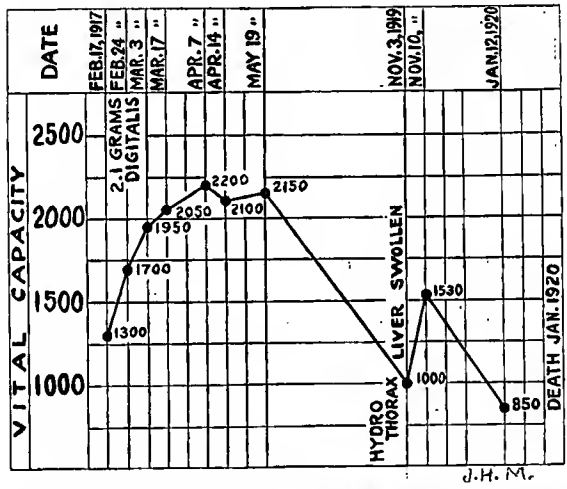

CHART III. Case I.—Mitral stenosis with auricular fibrillation. Improvement from digitalis without bed rest. Record of vital capacity for period of nearly three years.

On January 12, 1920, he returned to the clinic in very bad condition. In spite of having taken 4 grams of digitalis leaf during the previous fortnight the vital capacity had fallen to 850 cc which was 21 per cent. The apex rate was 120, the radial 90. Death occurred a few days later.

The chart shows an increase of 750 cc in the vital capacity which must be attributed to digitalis as he was given no other treatment. He took no regular rest and resumed work before the pulse deficit had disappeared. His vital capacity rose in three months to 54 per cent of the normal. Among 19 ambulatory cases of heart disease

in which I followed the change in vital capacity resulting from treatment only 1 other case showed as marked a rise. The effect of bed rest, measured by the increase in vital capacity has been much greater.

When the patient returned over two years later with his heart greatly weakened it will be noted that although he stated that he could walk further than in February, 1917, before he had to stop, the vital capacity was lower. Rapid improvement with a rise of 530 cc in the vital capacity in one week followed the employment of digitalis and rest. Omission of digitalis for one month resulted disastrously.

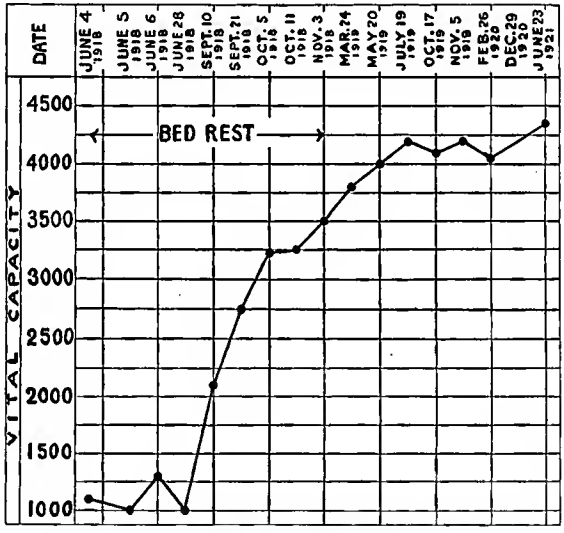

CHART IV. Case II.—Acute myocarditis. Increase of vital capacity from 23 per cent to 74 per cent while in bed.

CASE II.—The clinical history of this case has already been published, and a portion of the vital capacity readings.[10] The patient, a young man of twenty-one, had acute rheumatic myocarditis. He had been ill nearly three months when admitted to the Baptist Hospital, Boston, on June 4, 1918. For five weeks previous to entrance he had been unable to lie down in bed owing to

[10] Pratt, J. H.: Southern Med. Jour., 1920, 13, 481–490.

dyspnea. His vital capacity on admission was reduced to 1100 cc which was 23 per cent of the normal (West's standard). The next day it was 1000 cc or 21 per cent. After he had been at rest in the hospital twenty-four days the vital capacity was still 21 per cent. In September and October there was a marked rise. It increased from 45 per cent on September 10 to 69 per cent on October 11, being an increase of 1125 cc. When he left the hospital on November 3, the vital capacity was 74 per cent. Under rest and graduated exercise it rose slowly until it reached 89 per cent in July, 1919. He was then not short of breath after walking any distance on the level. He took up his work as a clerk in an importing house at this time. Two years later in June, 1921 his vital capacity reached the maximum. It was 4350 cc or 92 per cent of his normal. The chart (Chart IV) covers a period of three years and shows, (1) how low the vital capacity may fall in acute myocarditis and yet recovery occurs, (2) the beneficial effect of long continued rest in restoring the vital capacity, (3) the restitution of the vital capacity nearly to normal in the course of three years. This patient has an enlarged heart with aortic and mitral insufficiency.

CASE III.—The chart (Chart V) gives the vital capacity record over a period of five years, in this case of mitral stenosis with auricular fibrillation. Miss H. P. was forty-nine years of age in 1917, when the vital capacity was first determined. It was then 1600 cc or 52 per cent of the normal. She had pounding of the heart and a slight sense of suffocation and pressure across the sternum if she took even a few steps rapidly. At times when nervous she had these symptoms while at rest. In 1918 she was stronger. On May 29, I noted: "No trouble from heart. Possibly slight breathlessness at times. No palpitation." Her pulse that day was 46 at the apex and the wrist. The vital capacity was 2000 cc which was 65 per cent of her normal. Since 1918 she has been unable to move about freely without discomfort. The cardiac power has been less and the chart shows that the vital capacity has never been so high. Since June, 1919, it has only twice been recorded above 50 per cent.

In the late fall of 1920 she had a severe attack of rheumatic myositis, pericarditis and pleurisy. The great fall in vital capacity with its subsequent slow rise is indicative of involvement of the heart muscle by the rheumatic infection. On January 28, the vital capacity reading was only 350 cc or 11 per cent. The following day after the withdrawal of 990 cc of a somewhat turbid serous fluid from the left pleural cavity it was 500 cc or 16 per cent. The dyspnea which had been marked for several days was lessened. Two days later she appeared very sick and weak. An attempt to obtain the vital capacity was unsatisfactory. The amount of air expelled from the lungs was only 150 cc. It is probable that not

only this low reading, but the others obtained while the patient was very ill were less than the true vital capacity. The rise in the curve was slow and was only 925 cc or 30 per cent on March 16 when patient's general condition was much improved. The vital capacity rose until July, 1921, when it reached 1625 cc or 53 per cent. She could then walk slowly twenty-five to fifty yards on the level without breathlessness. In April, 1922, she was unable to lift light objects such as a blanket without producing pounding of the heart and a slight sense of suffocation. The vital capacity on April 18, 1922, was 1250 cc or 41 per cent. (On June 22, it had fallen to 1100 cc or 36 per cent without any definite change in her clinical condition.) The pulse has been kept slow during the past six years by the constant administration of digitalis.

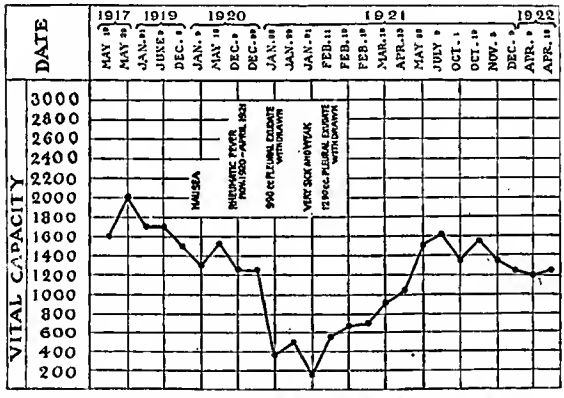

CHART V. Case III.—Vital capacity record over period of five years in a case of severe mitral stenosis. Fall of vital capacity during attack of acute rheumatic pericarditis with slow rise during convalescence.

CASE IV.—F. S., male, aged fifty years, when he came under observation in 1920. For ten months had been troubled with "wheezing" when he laid down at night. Increasing breathlessness on exertion gradually developed. About May, 1920, the effort of walking up a single flight of stairs made him short of breath. In June he had a severe attack of dyspnea which lasted all night. Cheyne-Stokes breathing first noticed then. It occurred every night when asleep. When I saw him he presented evidence of cardiosclerosis. The blood-pressure was high. Systolic pressure 180 mm.; diastolic pressure 120 mm. The pulse was regular, but alternating. His vital capacity was 2475 cc or 51 per cent of the

normal. He was admitted to the Baptist Hospital that day. Under strict bed rest and the Karrel diet the attacks of dyspnea ceased and the vital capacity rose to 3400 cc or 69 per cent on September 28. The administration of 2 grams of digitalis leaf in a single dose was followed by a second and more rapid rise (Chart VI). From October 9 to 11 the vital capacity increased from 3350 cc to 3650 cc. When he left the hospital on October 20, it was 3750 or 76 per cent of the normal. This was an increase of 1275 cc or 21 per cent during the six weeks of hospital treatment. On March 30, 1921, the auricles began to fibrillate. This proved to be a permanent arrhythmia. His heart was digitalized at the time of onset. The pulse remained slow, the rate being 60 to the minute.

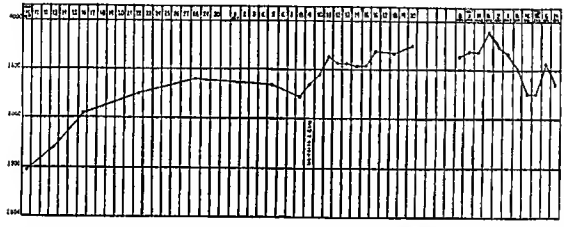

CHART VI. Case IV.—Cardiosclerosis with cardiac insufficiency. Vital capacity record from September, 1920, to April, 1922. Note rise as result of rest treatment followed by a second sharper rise after administration of digitalis. Slow fall in 1921 resulting from increasing cardiac weakness.

Four days later his vital capacity was taken and found to be higher than at any time since he had been under observation. It was 3875 cc or 79 per cent. On April 6, it rose to 80 per cent. With the onset of the abnormal cardiac mechanism, however, Cheyne-Stokes breathing which had disappeared when the patient's condition improved in the hospital, returned and persisted. There was a gradual fall of vital capacity from the height of 80 per cent reached in April, 1921 to 66 per cent in December. In February, 1922, he began to be troubled again with dyspnea when at rest. His sleep was troubled. He would awake during the hyperpnoeic phase of Cheyne-Stokes breathing. Readmitted to the Baptist Hospital. Under the usual treatment, by rest, morphin, digitalis, and Karrel diet, his condition improved and the vital capacity rose to 3575 cc or 73 per cent on April 6.

In this case four points may be emphasized. (1) The steady slow rise of the vital capacity after rest treatment was begun in September 1920, lasting eighteen days, then a stationary condition for a week followed by a slight drop. (2) The sharp sudden secondary rise beginning October 9, 1920, which is attributed to digitalis. (3) The

rise to the maximum on April 6, 1921, after the establishment of the abnormal cardiac mechanism (auricular fibrillation). (4) The slow steady fall from April to December, 1921, accompanied for several months by only slight signs of diminishing cardiac power.

The graphic records on the charts of these and similar cases show the value of long continued observations of the vital capacity repeated over a long period of time in measuring the increase or decrease of cardiac power. They will be found to be a distinct aid in the treatment and prognosis of heart disease.

Conclusion. I am sure of the facts presented. The deductions given are simply my interpretation of them. I would present this work in the same spirit that moved John Hutchinson in 1846 to close the report of his studies with these words. "The deductions I have ventured to draw" (from the facts presented) "I wish to advance with modesty, because, time, with its mutations, may so unfold science as to crush these deductions and demonstrate them as unsound. Nevertheless, the facts themselves can never alter, nor deviate in their bearing upon respiration—one of the most important functions in the animal economy."

EXPERIENCES IN NEW YORK HOSPITAL WITH THE TREATMENT OF LOBAR PNEUMONIA BY A SERUM-FREE SOLUTION OF PNEUMOCOCCUS ANTIBODIES.*

By Lewis A. Conner, M.D.,
NEW YORK.

During the past two years all the cases of lobar pneumonia admitted to the adult wards of the First Medical Division of the New York Hospital have been treated by the intravenous use of a serum-free solution of pneumococcus antibodies prepared according to the method of F. M. Huntoon[1] and supplied to the hospital by the Mulford Biological Laboratories. The study of the effects of this antibody solution has been made with the coöperation with the Second Medical Division of Bellevue Hospital where the same method of treatment has been employed during the same period of time.[2]

The preparation of the solution is based upon the capacity of

* Read at the meeting of the Association of American Physicians, Washington, D. C., May, 1922.

[1] Tr. Philadelphia Path. Soc., 1920, 22, 75. Jour. Immunol., 1921, 6, 117. Huntoon and Etris: Jour. Immunol., 1921, 6, 123. Huntoon and Craig: Jour. Immunol., 1921, 6, 235.

[2] Cecil and Larsen: Jour. Am. Med. Assn., 1922, 79, 343.