

A STUDY OF THE DIAGNOSTIC AND PROGNOSTIC SIGNIFICANCE OF VENOUS PRESSURE OBSERVATIONS IN CARDIAC DISEASE *

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That high venous pressures occur in cases of cardiac decompensation has been observed by Gaertner,¹ Moritz² and von Tabora,³ Frank and Reh,⁴ Sewall,⁵ Schott,⁶ Hooker and Eyster,⁷ and others. The routine measurement of venous pressure at frequent intervals as a means of following the stages of compensation or decompensation, however, has received little attention. The following investigation was undertaken with the purpose of ascertaining whether there is any diagnostic or prognostic significance in repeated venous pressure observations on cardiac cases.

METHOD

A number of instruments and methods have been devised by various workers to measure venous pressure. The technic and relative value of the methods used until 1914 have been summarized by Austin.⁸ Of these, the direct method of Moritz and von Tabora of introducing a hollow needle directly into the vein, while perhaps giving a greater refinement of accuracy, would manifestly be open to serious objections in cases in which a large number of readings were made at frequent intervals. Probably the most useful clinical method was devised by Hooker⁹ in 1914, and it is his instrument which has been used in this investigation. As shown in Figure 1, it consists essentially of a small glass cup (*B*) 2 cm. in diameter and 1 cm. deep, connected with a water manometer. The chamber is sealed to the skin over a suitable vein on the back of the hand by a rim of collodion. In drying, the collodion draws the skin slightly inward, so removing possible error due to superficial tissue tension. The manometer is connected

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* From the Medical Clinic of the Johns Hopkins Hospital.

1. Gaertner: *München. med. Wchnschr.*, 1903, 1, 2038.

2. Moritz and Von Tabora: *Deutsch. Arch. f. klin. Med.*, 1910, xcvi, 475.

3. Von Tabora: *München. med. Wchnschr.*, 1910, lvii, 1265.

4. Frank and Reh: *Ztschr. f. exper. Path. u. Therap.*, 1912, x, 241; 1913, xiii, 37.

5. Sewall: *Jour. Am. Med. Assn.*, 1906, xlvii, 1279.

6. Schott: *Deutsch. Arch. f. klin. Med.*, 1912, cviii, 537.

7. Hooker and Eyster: *Johns Hopkins Hosp. Bull.*, xix, 274.

8. Austin: *Blood Pressure: Its Clinical Application*, G. W. Norris, 1914, 129.

9. Hooker: *Am. Jour. Physiol.*, 1914, xxxv, 73.

to the cup by a rubber tube, and by compressing the manometer bulb (C) the air pressure in the cup is increased. If the vein be observed by oblique illumination the point at which it definitely begins to collapse can be read in centimeters of water on the manometer scale. Hooker finds that "the most consistent results are obtained when the reading is made at the point where slight oscillations of pressure cause the vein shadow to come and go promptly just before the vessel is completely collapsed." This point has been used throughout this investigation. In all readings the same section of vein was constantly observed.

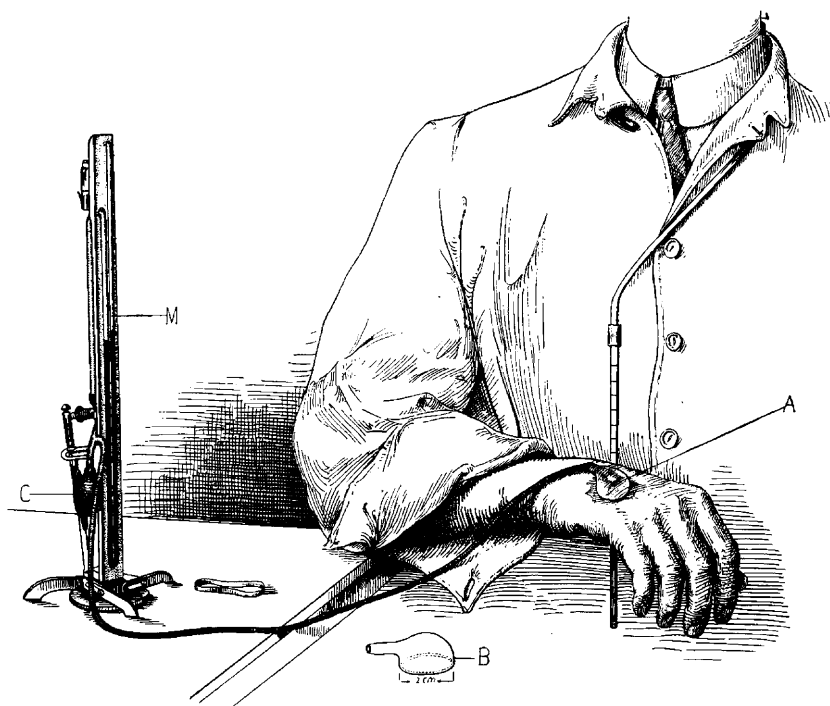


Fig. 1.—Hooker's venous pressure apparatus. A small glass chamber (B) measuring 1 by 2 cm. is held temporarily by a rubber band over a suitable vein on the back of the hand, as shown at (A). A rim of collodion is applied and in drying it seals the chamber to the skin. The rubber band is then removed and the chamber connected by a rubber tube to the water manometer (M). By pressing on the manometer bulb (C) the air pressure in the chamber is raised and a reading is made "at the point where slight oscillations of pressure cause the vein shadow to come and go promptly just before the vessel is completely collapsed." This pressure is recorded directly by the water manometer. The hand is held at the level of the mid point of the anterior-posterior diameter of the body at the costal angle.

As to the so-called "heart level," an arbitrary level in relation to the heart which must be chosen in order to give a constant point for making readings, the level originally defined by von Recklinghausen

and subsequently adopted by Hooker, has been used. This was chosen as the midpoint of the anterior-posterior diameter of the body at the costal angle. The hand, in each observation in this series, was placed on a pillow or other support at this level and was maintained quietly for at least three minutes to allow the hydrostatic factors to become adjusted and constant. As most patients with cardiac decompensation lie in bed with the body at an angle of about 45 degrees to the horizontal, this position was also adopted as the position in which to make the readings. In those cases with a venous pressure higher than the manometer could record, the hand was raised above the "heart level" until the venous pressure was within the range of the manometer. This hydrostatic column in centimeters was then added to the venous pressure.

It was further necessary to adopt the following three conditions: First, the vein must stand out sufficiently from the surrounding skin level to give a distinct shadow by oblique illumination. Second, the vein wall must be collapsible. Third, the patient must be lying quietly and undisturbed in bed.

Obviously not every cardiac case could be studied. Old patients with phlebosclerosis, patients with exceedingly edematous or fat hands, and patients with continuously small veins, had to be passed by. Nevertheless, excluding the rare cases of patients with hardened vessels there were few cases that did not show satisfactory veins on the back of the hand at some time during the day.

MATERIAL

Two hundred and seventy-six venous pressure observations have been made on fourteen cases at various stages of cardiac decompensation. The largest number of readings on a single case was seventy-three (Case 1), covering a period from November 5 to March 7. The lowest number was five (Case 14). In addition to the venous pressure, the systolic and diastolic arterial pressures by the auscultatory method, the pulse rate, the treatment, and the clinical condition of the patient were noted. The fluid intake and output for each twenty-four-hour period was recorded in the majority of cases. At each observation the venous pressure was taken before any of the other data in order to disturb the patient as little as possible. The highest venous pressure recorded was 50 cm. of water (Case 2) which exceeds any record found in the literature. This reading was made by two observers. A case was followed only so long as the venous pressure remained high or there was any doubt as to compensation. Five (Cases 2-6) of the fourteen patients died. In addition to these fourteen cases, eight cases were studied to determine the diurnal variation in venous pressure.

Before giving the results which were found, it would be well to state the factors which alter venous pressure, as observed by previous workers, especially in their relation to the present investigation. Barach and Marks¹⁰ in a series of forty-eight normal cases noted a fall in venous pressure on passively changing from the erect to the horizontal position, and a rise of pressure in returning to the erect posture. Hooker,¹¹ Elpers,¹² and Schott⁶ found that muscular exertion

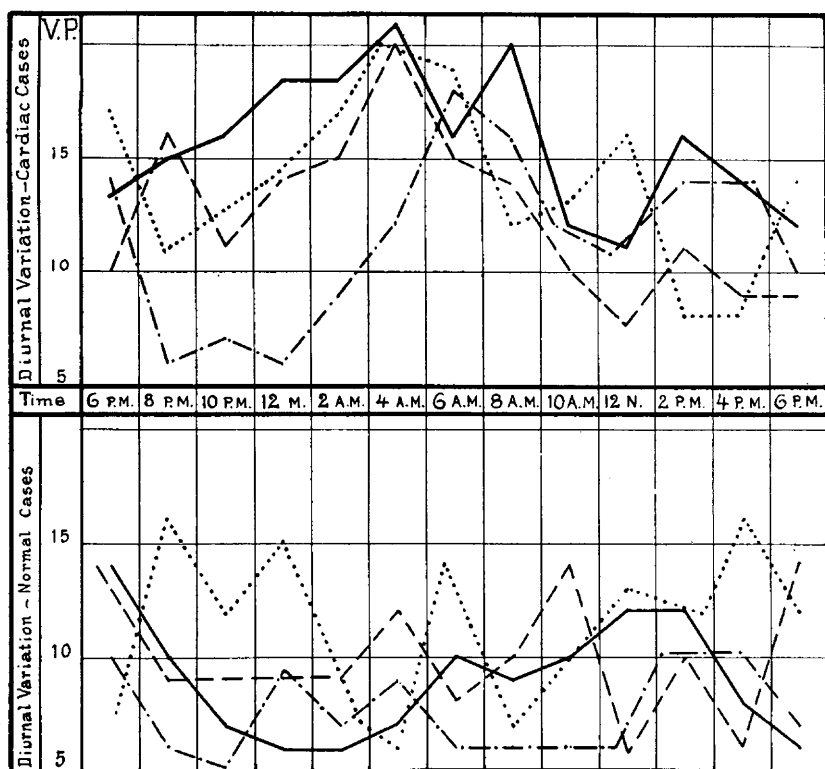


Fig. 2.—Curves showing the diurnal variation in four cardiac and four normal cases. The patients were all in bed under similar ward conditions. Note that in the cardiac cases the highest venous pressures occur at night, while in the normal cases there is an almost complete reversal of the picture, the lowest venous pressures tending to occur during the sleeping hours.

increases the venous pressure. The work of Elpers¹² would indicate that heat and cold increase the venous pressure, and finally, Hooker⁹ has demonstrated a distinct diurnal variation in the venous pressure.

Obviously, for a patient lying quietly in bed under the conditions which were observed, the factors of temperature change, of muscular

10. Barach and Marks: *THE ARCHIVES INT. MED.*, 1913, xi, 485.

11. Hooker: *Am. Jour. Physiol.*, 1911, xxviii, 235.

12. Elpers: *Inaug. Diss.*, Kiel, 1911.

exertion, and of posture were practically constant. Under ideal experimental conditions these factors could hardly have been more carefully regulated. As to the complete diurnal variation under these conditions no data could be found, although Hooker⁹ noted a distinct diurnal variation under normal conditions of health and a decided fluctuation in venous pressure during the day in three surgical cases confined to bed.

DIURNAL VARIATION

In view of these observations by Hooker, it was necessary, before it could be decided what constitutes a pathological venous pressure, to see what part the diurnal variation plays in the determinations made on patients confined to bed. To answer this question eight patients were selected on the medical wards. Only those cases which showed

TABLE 1.—CARDIAC CASES

Case	Max. V. P.		Min. V. P.		Diurnal Variation Cm. H ₂ O
	Reading	Time	Reading	Time	
S.	21	4 a. m.	11	12 noon	10
T.	20	4 a. m.	8	12 noon	12
M.	20	4 a. m.	8	2-4 p. m.	12
J.	18	6 a. m.	6	8-12 p. m.	12

TABLE 2.—NORMAL CASES

Case	Max. V. P.		Min. V. P.		Diurnal Variation
	Reading	Time	Reading	Time	
D.	14	6 p. m.	6	12 a. m.	8
..	4 a. m.	..
G.	14	6 p. m.	6	12-4 p. m.	8
..	8	6 a. m.	..
W.	10	6 p. m.	5	10 p. m.	10
L.	16	2 p. m.	6	4 a. m.	9
..	..	8 p. m.

excellent veins for reading were chosen. Four of these cases were at various stages of cardiac decompensation, and four had no cardiac complications. The observations were made at two-hourly intervals over a period of twenty-four hours. The glass cups were left attached during the entire period so that to make a reading it was only necessary to connect the manometer tube. All the patients were in the same ward environment under constant and similar conditions, and during

the twenty-four-hour period they remained in bed. There was a difference, however, between the cardiac and normal cases during the night, in that the normal patients lay flat in bed while the cardiac patients remained propped in a semi-erect posture. During the night it was possible to make the majority of readings without waking the patients, a small pocket electric light being used.

The eight cases are plotted in the curves shown in Figure 2. It is evident from these curves that there is a definite diurnal variation in venous pressure even under the constant conditions of the experiment. In comparing the curves of the normal and cardiac cases certain differences can be seen which are tabulated in Tables 1 and 2.

The first and striking fact is that in the cardiac cases the highest venous pressures occur in the early morning hours, while in the normal cases, as Hooker⁹ found, the lowest venous pressures tend to occur at night. The second fact is that the diurnal variation averages 2.5 cm. more in the cardiac than in the normal cases. This greater change of pressure is interesting in connection with the observations of Schott,⁶ that venous pressure can be altered by exercise in proportion to the degree of cardiac decompensation.

WHAT SHOULD BE CONSIDERED AS A PATHOLOGICAL VENOUS PRESSURE?

Since, therefore, a daily variation of from 8 to 12 cm. in venous pressure has been found in ward cases, the essential question is, what can be regarded as a pathological venous pressure? It is generally admitted that venous pressure observed at a fixed point and level is a fair indication of the feeding pressure of the heart. What, however, is the border-line between an efficient feeding pressure and a pressure due to an incompetent heart that cannot handle the blood with which it is supplied? With the factor of diurnal variation to be considered, an observation made one day might give the lowest and a reading the following day the highest pressure of a normal diurnal variation, and so one might be led to believe that there had been an abnormal increase in the venous pressure.

Various observers, depending on the method used, have given different values as to the upper limits of a normal pressure. Hooker,⁹ by the method here used, gives 20 cm. as the upper limit of the normal diurnal variation under the varying conditions of daily life. To decide this point, the fourteen cases observed in this series have been classified according to the clinical divisions of "compensated or compensating" and "decompensated or decompensating." These divisions were evident from the clinical signs and symptoms. Of the six patients whose cases were classified as "decompensated," five died, and the sixth

(Case 1) recovered, though it was doubtful for three months whether he would live. At the time of writing none of the "compensated" cases have terminated fatally. All the patients have recovered or are on the way to recovery.

From Table 3 it will be seen that of 171 observations on six "decompensating" cases the average venous pressure in each case never went below 20 cm. while of 105 readings on "compensating" cases, the average of no case ever went above 20 cm. In this number of observations the factor of diurnal variation must be well equalized, for the readings were made at all hours of the day. The average venous pressure for all the "compensating" cases was about 14 cm. and of the "decompensating" cases 26 cm. It would seem, therefore, from these figures, that by this method, a venous pressure above 20 cm. is pathological, while any pressure below this may be regarded as within the limits of normal variation.

TABLE 3.—COMPENSATED AND DECOMPENSATED CASES

Case No.	Clinical Condition	No. Days Observed	Average Venous Pressure, Cm. Water
1	Decompensated	73	28.0 { first; 29 days
2	Decompensated. Died	33	20.0 { last; 44 days
3	Decompensated. Died	21	29.7
4	Decompensated. Died	8	21.0
5	Decompensated. Died	19	23.2
6	Decompensated. Died	17	29.0
			29.3
		T'l 171	Avg. 26.0
7	Compensated	8	13.0
8	Compensated	30	10.7
9	Compensated	8	17.4
10	Compensated	25	14.0
11	Compensated	5	10.4
12	Compensating	9	15.2
13	Compensating	15	13.8
14	Compensated	5	19.0
		T'l 105	Avg. 14.3

It must be borne in mind, however, that a venous pressure below 20 cm., though indicating that a heart is handling the blood with which it is supplied, nevertheless, does not indicate how narrow the margin is between compensation and decompensation. Thus a heart barely compensating under perfect bodily quiet might give the same venous pressure as a well-compensated heart. Any unusual strain in the former case, however, would be sufficient to throw the heart back into decompensation, while in the latter case decompensation would not occur. It is on this consideration that Schott⁶ based his venous pres-

sure test for the functional capacity of the heart. As shown above, however, the line of immediate danger seems to be at 20 cm. A pressure persisting above this gives a serious, and a pressure below a favorable, outlook.

CLASSIFICATION OF DATA

The deductions which follow are based on the observations stated under "Material" and are illustrated by the curves (Figs. 3-6) which demonstrate the most typical findings in the fourteen cases studied. The clinically interesting facts which they illustrate, follow:

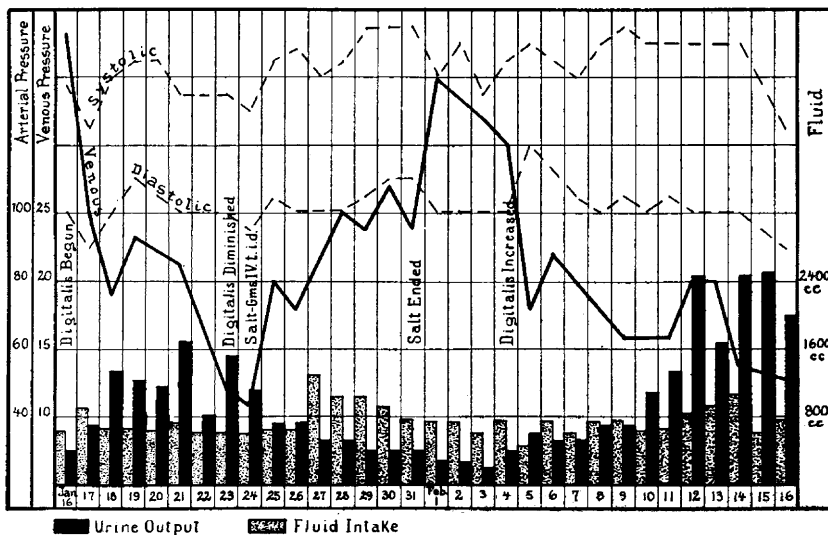


Fig. 3.—From the chart of Case 1, showing the relation of venous pressure, arterial pressure, fluid intake, and urine output. During the first fall in venous pressure the response in urine output is practically coincident, while after the second drop in pressure there is a delayed response of several days. Note also that the venous pressure gives an indication of the effect of therapy and compensation which are shown in no way by the arterial pressure.

Relation of Venous Pressure to Decompensation.—Of the seven cases which showed decompensation (Cases 1-8) all gave a direct relation between venous pressure as followed at frequent intervals and the degree of compensation as indicated by the clinical signs. A pressure continuously above 20 cm. almost invariably gave symptoms which called for treatment.

Rise in Venous Pressure Preceding a Break in Compensation.—In Case 1 (see Figs. 3 and 4), (November 12 to 18, November 23 to 30, December 3 to 11, January 12 to 18, January 26 to February 6), Case 2 (October 15 to 19, November 3 to 8, November 13 to 18, November 23 to 29), Case 3 (see Fig. 5), (November 25 to 27, December 7 to 9, December 12 to 20), Case 4 (see Fig. 6), Case 5 (October 16 to 26,

October 27 to November 9), and Case 6 (October 16 to November 7, November 10 to 19) a steady rise in venous pressure definitely preceded a break in compensation. When the venous pressure reached a high point the usual clinical signs of decompensation generally became apparent enough to call for treatment. If such treatment had been instituted before the venous pressure had risen to such a height the break in compensation might have been averted and the heart spared.

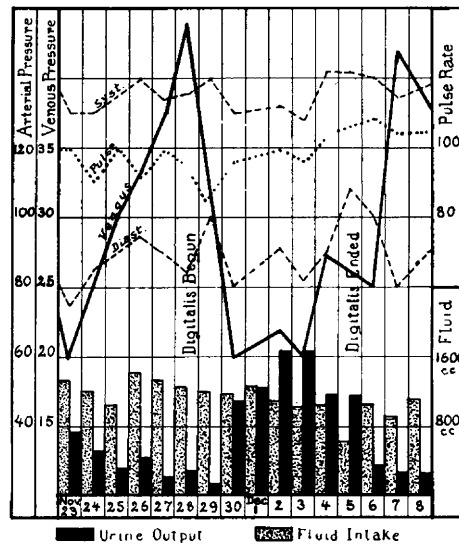


Fig. 4.—From the chart of Case 1, showing the relation of venous pressure, arterial pressure, and fluid intake and output. A rise in venous pressure and a fall in urine output preceded the clinical signs of decompensation which called for digitalis therapy. Note that neither the systolic nor the diastolic arterial pressures gave any indication of the degree of compensation. Only after the venous pressure had reached a high point did the clinical signs and symptoms become apparent enough to call for treatment.

Relation of Digitalis and Strophanthin to Venous Pressure.—All the patients in this series received digitalis or strophanthin either once or oftener during their stay in the hospital. Table 4 groups the results which were found.

TABLE 4.—RESULTS OF USE OF DIGITALIS SERIES

Venous Pressure	Died	Compensating *	Decompensating
Definitely lowered by digitalis series.....	2,	10	1, 7
Not definitely lowered by digitalis series	3, 4 5, 7	8, 9, 11, 12, 13, 14	

* Average venous pressure, 14.

This table indicates that of the five cases which terminated fatally (See Figs. 5 and 6 as examples) only one showed lowering of the venous pressure as a result of cardiac tonics, while of the "compensated" cases also, only one showed a lowering of the venous pressure. In this latter case the variation was in the limits of normal. The lack of effect of digitalis or strophanthin on compensated cases would seem to be in accord with the experimental evidence of Capps and Mathews,¹³ that the digitalis group does not materially alter the venous pressure in normal animals. The two decompensating cases in which the patients did not die gave a striking response to the digitalis group (See Figs. 3, 4, and 6).

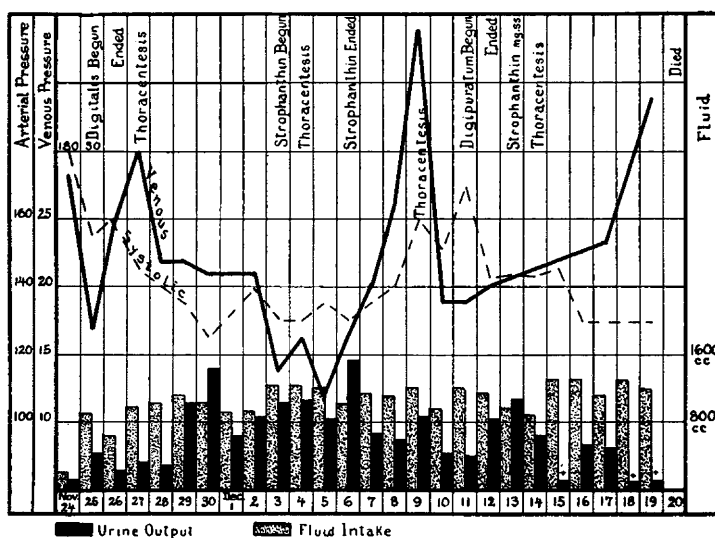


Fig. 5.—Case 3. Chart showing the relation of venous pressure, systolic arterial pressure, and fluid intake and output from admission to the hospital until death. The diastolic pressure could not be determined because of the extreme degree of aortic insufficiency. Note that from December 8 to 12 there was a coincident rise of venous and arterial pressures, but that before death the arterial pressure fell as the venous pressure rose.

If any conclusions can be drawn they would seem to be, first, that with the venous pressure continuously below 20 cm. the effect of digitalis cannot be followed by the venous pressure, and second, a venous pressure continuously above 20 cm., which is not lowered by digitalis, is an indication of grave cardiac involvement.

High Venous Pressure Before Death.—Of the five patients who died (2, 3, 4, 5, 6) all gave high venous pressure readings before death. Case 4 gave a reading of 38 cm. six hours before, while Case 3

13. Capps and Mathews: Jour. Am. Med. Assn., 1913, lxi, 388.

showed a pressure of 34 cm. fourteen hours before exitus. It was impossible to make readings on Cases 2 and 5 immediately before death, but so long as observations were possible, each showed, over a period of two weeks, average venous pressures of 37 cm. and 30 cm. respectively. Case 6, a typical cardiorenal, was continuously on the verge of severe decompensation. While under observation the patient's venous pressure averaged 29 cm. over a period of five weeks, and he died shortly after leaving the hospital. A rapid rise of venous pressure, however, in all the cases observed, did not invariably end unfavor-

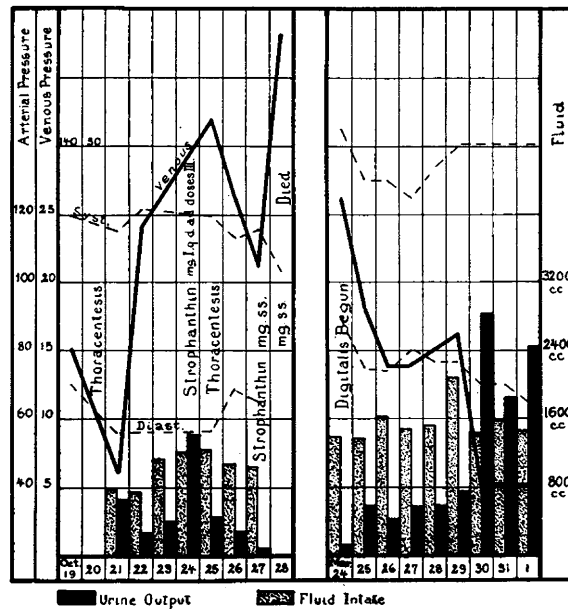


Fig. 6.—Case 4 (left), Case 7 (right). Charts of two cases of severe myocardial insufficiency, one ending in death, the other in recovery, showing the relation of venous pressure, arterial pressure, and fluid output and intake. Note that in the case which ended fatally the venous pressure steadily rose, the urine output diminished, and the systolic pressure fell with a lowering of the pulse pressure in spite of thoracentesis and strophanthin. In the case which ended in recovery the venous pressure fell rapidly, and the urine output and pulse pressure responded four days later to simple digitalis therapy.

ably, as can be seen by glancing at Figures 3 to 6. The proper therapy often lowered a high venous pressure. The interesting facts nevertheless remain, first, that so long as observations were possible no patient died with a low venous pressure, and second, that a rapid elevation of pressure to a high level, or a continuously high average venous pressure, was of serious prognostic significance.

Relation of Arterial to Venous Pressure.—The relation between arterial and venous pressure, as found in these cases, is interesting.

Plumier's¹⁴ experiments on animals support the theoretical conclusion that venous and arterial pressure vary inversely. Elpers,¹² Schott,⁹ and others, in human cases, have found that the height of venous pressure is not passively dependent on the height of arterial pressure. Isolated observations by me on several cases of extreme hypertension in which compensation was perfect, did not give high venous pressure readings. One case brought to the accident department with cerebral hemorrhage showed a systolic pressure of 300 mm., a diastolic of 180 mm. Hg, and a venous pressure of 20 cm. water. The relations which were noted between arterial and venous pressure are tabulated in Table 5.

TABLE 5.—RELATIONS BETWEEN ARTERIAL AND VENOUS PRESSURES

	Venous Pressure Shows No Relation to	Venous Pressure Varies Directly with	Venous Pressure Varies Inversely to
Systolic arterial pressure	1 (at first), 2, 7, 10, 11	3 (at first), 5 (at first), 8, 9, 12	3 (before death), 5 (before death), 4, 6
Diastolic arterial pressure	1, 2, 4, 6, 9, 10, 11	7, 8, 12	
Pulse pressure	1, 2, 3, 4, 5, 6, 8, 9, 10, 11, 14	7, 12
Pulse rate	1, 2, 3, 4, 6, 7, 8, 9, 10, 11, 13	12	5

Thus, of 12 cases on which sufficient observations were made, 5 showed no relation between the systolic and the venous pressures; in 3, the venous and systolic pressures varied directly; 2 gave an inverse ratio between the pressures; while in 2 cases the venous and arterial pressure varied directly at first, and before death varied inversely. In 4 of the 5 cases which terminated fatally, the inverse ratio was noted, that is, the venous pressure rose and the systolic pressure fell before death.

Satisfactory diastolic pressure readings could be made in ten cases. Seven indicated no relation between diastolic pressure changes and venous pressure changes, while in three there was a fall in venous pressure with a fall in diastolic pressure. From the tables, however, it can be seen that this fall in diastolic pressure in Cases 8 and 12 was synchronous with a fall in systolic pressure but that in Cases 7 and 12 there was an increased pulse pressure due to the fall in diastolic pressure.

As to the relation of pulse pressure to venous pressure it was found that in eleven cases no relation was noticeable, while in two

14. Plumier: Arch. internat. de Physiol., 1909, viii, 1.

(See Fig. 6, Case 7 as example) there was a fall in venous pressure with an increased pulse pressure.

In this connection it is interesting to note the relation of the so-called "amplitude-frequency product" of v. Recklinghausen.¹⁵ It was suggested by him that the product of the pulse pressure and pulse rate might be a criterion of the condition of the greater circulation. Fellner¹⁶ thought he found a relation between this amplitude-frequency factor, and the venous pressure, in cardiac cases. Ten cases in this series could be compared as to this point. Of these, six, based on 173 observations, showed no definite relation, while four cases, based on 74 observations, showed only an occasional relation between the venous pressure and the amplitude-frequency product. In this series, therefore, neither the pulse pressure nor the product of the pulse pressure and the pulse rate seemed to be associated in any definite way with venous pressure.

To summarize the relation between the clinical observations of arterial and venous pressures in this series, it may be said that, except for a fall in systolic and a rise in venous pressure before death in four cases, no constant relation could be found between either the systolic, the diastolic, the pulse pressure, or the amplitude-frequency product on the one hand, and the venous pressure on the other; that in general, the venous pressure gave indications of the variations in compensation which were suggested in no way by the data obtainable from the arterial pressure.

Relation of Venous Pressure to Urine Output.—Clinically and experimentally it is well known that the function of a normal kidney depends largely on the circulation. With congestion and slowing of the blood stream the amount of fluid put out by the kidneys is diminished and with a relief of this congestion the kidney function returns to normal. Reference to Figures 3 to 6 shows a distinct relation between the venous pressure and the urinary output. In a total of 203 observations on ten cases in which the fluid intake and output were studied, this inverse ratio of venous pressure to urinary secretion was noted in seven (1, 2, 3, 4, 7, 10, 13) and was not found in two cases (8, 12). In six of these eight patients it was not apparent which was the first indication of the changed circulation, while in two instances (Figs. 3, and 6, Case 7) the drop in venous pressure preceded the polyuria by several days.

We would conclude, therefore, from this series that in the majority of cases the venous pressure and the amount of urine varied inversely and that the variations are coincident. This would add further evi-

15. Von Recklinghausen: Arch. f. exper. Path. u. Pharm., 1906, lvi, 1.

16. Fellner: Deutsch. Arch. f. klin. Med., 1907, lxxxviii, 1.

dence to show that consecutive changes in venous pressure are an indication of the condition of the circulation and heart.

Venous Pressure as Related to Intrathoracic Pressure.—Cases 2, 3 and 4 in this series had sufficient hydrothorax to make thoracentesis necessary. Eight pleural tappings were performed on these three cases. The venous pressure was lowered seven times with an improvement in the clinical condition of the patients. (See Figs. 5 and 6 as examples.) The final thoracentesis in Case 3 did not lower the venous pressure and the patient died. The intrathoracic pressure was measured twice with a water manometer in Case 3 and in each instance there was a change from a positive to a negative pressure during thoracentesis. This is in accord with the results of Hooker,⁹ who found that increased intrathoracic pressure causes a rise in venous pressure which cannot be compensated. It also emphasizes the value of thoracentesis as a therapeutic measure in relieving an overstrained heart.

Effect of Venesection on Venous Pressure.—Table 6 illustrates this point in three cases observed. Venesection, therefore, seems to lower venous pressure, but the subsequent rise in pressure is rapid. The results of von Tabora agree with these observations.

TABLE 6.—EFFECT OF VENESECTION ON VENOUS PRESSURE

Patient	Condition	Amount of Blood Drawn c.c.	Fall in Venous Pressure cm.	Subsequent Effect on V. P.
S. H.	Emphysema, pulmonary congestion	500	28-9	Following day V. P. = 18
P. W.	Myocardial insufficiency. Pulmonary congestion	550	29-14	Following day V. P. = 28
L. M.	Normal, transfusion donor	540	18-9	Three hours later V. P. = 15

SUMMARY

1. The venous pressure has been followed at frequent intervals in fourteen cases of cardiac insufficiency at various stages of decompensation and compensation. Two hundred and seventy-six observations have been made. Hooker's modified method has been used, and the conditions which must be observed in making clinical venous pressure readings by this method have been given.

2. A diurnal variation in venous pressure of from 8 to 12 cm. of water was noted in four normal and four cardiac cases, all the patients

being confined to bed. This variation was based on two-hourly observations over a period of twenty-four hours. It was found that the highest venous pressures occurred during the sleeping hours in the cardiac cases, while in the normal cases, as previously observed by Hooker, the lowest pressures of diurnal variation occurred at night.

3. In the patients who died, either a rapid rise in venous pressure or a continuously high pressure above the 20 cm. level was observed before death.

4. A venous pressure continuously above 20 cm., which was not lowered by the digitalis series, was an indication of grave cardiac involvement.

5. A venous pressure continuously below 20 cm. gave no definite indication as to the effect of digitalis or strophanthin therapy.

6. A fall in systolic arterial pressure and a rise in venous pressure occurred in four cases before death. Otherwise no constant relation could be noted either between the systolic, the diastolic, the pulse pressure, or the amplitude-frequency product on the one hand, and the venous pressure on the other.

7. Venous pressure and urine output generally showed an inverse variation. The variation was usually coincident, but, if coincidence did not occur, the changes in venous pressure tended to precede the changes in kidney function.

8. The venous pressure was lowered in seven out of eight pleural tapings.

9. Venesection lowered the venous pressure in three cases observed, but the subsequent rise in pressure was rapid.

CONCLUSIONS

1. Venous pressure measurements, when made at frequent intervals, give a definite indication of the degree, and changes in the degree of cardiac decompensation.

2. A venous pressure of 20 cm., by this method, marks the danger line between compensation and decompensation.

3. A rise in venous pressure above 20 cm. precedes the clinical signs of decompensation.

4. Above the 20 cm. level a rising venous pressure has an unfavorable, and a fall in venous pressure a favorable prognostic significance.

5. Death from cardiac decompensation is preceded by a continuously high venous pressure, or a rapid rise.

6. Venous pressure observations at short intervals give information as to the degree of cardiac decompensation which is not obtainable by observation of the arterial pressure.

7. Frequent venous pressure measurements not only have a valuable diagnostic and prognostic significance, but also furnish an important indication for, and check on therapeutic measures.

Grateful acknowledgment is made to Dr. D. R. Hooker, who supplied the instrument used, and to the clinical staff of the hospital and others who kindly cooperated in this investigation.

PROTOCOLS

CASE 1.—J. S., aged 48, male, colored. Admitted Oct. 28, 1914. Clinical diagnosis: Aortic insufficiency and myocardial insufficiency. Eleven months before admission began having shortness of breath and swelling of the abdomen. Was improved by three weeks' stay in the hospital during April, 1914. Symptoms returned a few weeks before last admission. Admitted with dyspnea, orthopnea, edema of the legs, ascites, moderate hydrothorax, a large tender liver, greatly enlarged area of cardiac dulness and signs of aortic and mitral regurgitation. Under digitalis, purgation, diuretics, and limited fluids the patient wavered between compensation and decompensation for about six weeks, then went on to recovery. The venous pressure from November 5 to February 7 varied from 44 to 10 cm. The average venous pressure for the first twenty-nine days was 28 cm., and for the last forty-four days 20 cm.

CASE 2.—33,167. F. J., aged 49, female, colored. Admitted Oct. 12, 1914. Clinical diagnosis: Aortic insufficiency and myocardial insufficiency; syphilis. Three weeks before admission the patient began having shortness of breath, orthopnea, cough and edema of the ankles and feet. Admitted to the hospital with dyspnea, orthopnea, tachycardia, and enlarged area of cardiac dulness, the signs of aortic insufficiency and fluid at the bases of both lungs. Under limited fluids, purgation, thoracentesis, diuretics, digitalis and strophanthin the patient wavered between compensation and decompensation for five weeks, but finally no longer responded to treatment and died December 15. Necropsy confirmed the clinical diagnosis. The venous pressure from October 15 to December 4, when it could no longer be observed because of the edema of the hands, varied from 9 to 50 cm., with an average pressure of 29.7.

CASE 3.—33,356. S. S., aged 45, female, white. Admitted Nov. 24, 1914. Clinical diagnosis: Aortic insufficiency and myocardial insufficiency; syphilis. Five months before admission patient began having paroxysmal attacks of dyspnea, orthopnea, severe epigastric pain and edema of the ankles. On admission was dyspneic, orthopneic, with ascites, anasarca, hydrothorax and showed a large area of cardiac dulness and the signs of aortic and relative mitral insufficiency. In spite of repeated thoracentesis, limited fluids, purgation, diuretics, digitalis and strophanthin the patient did not improve and died about a month after admission. Necropsy confirmed the clinical diagnosis. The venous pressure observed from admission until death varied from 12 to 39 cm., with an average pressure of 21 cm.

CASE 4.—33,172. E. W., aged 20, female, white. Admitted Oct. 13, 1914. Clinical diagnosis: Acute and chronic endocarditis; mitral stenosis and insufficiency; myocardial insufficiency. At 14 years patient began having palpitation and shortness of breath. Ten months before admission became pregnant and as pregnancy advanced, dyspnea and palpitation became progressively worse. Following delivery on October 6 the patient became no better and in spite of repeated thoracentesis, limited fluids, purgation, diuretics, strophanthin and digitalis, the patient was not improved and died fifteen days after admission. Necropsy confirmed the clinical diagnosis. Venous pressure from October 19 until death varied from 15 to 38 cm. with one reading of 6 cm. immediately after thoracentesis. The highest venous pressure of 38 cm. occurred six hours before death.

CASE 5.—33,181. P. V., aged 52, white, male, admitted Oct. 15, 1914. Clinical diagnosis: Aortic and mitral stenosis and insufficiency; myocardial insufficiency. About a year before admission began noticing shortness of breath and swelling of the ankles. Was compelled to stop work. Was improved by five weeks' stay in the hospital in February, 1914, but soon relapsed into his previous condition. Admitted with dyspnea, orthopnea, anasarca, ascites, a large and tender liver and spleen, and the signs of aortic and mitral stenosis and insufficiency, and myocardial insufficiency. Was not improved by limited fluids, Karell diet, purgation, diuretics, digitalis, or venesection, and died four weeks after admission. Necropsy confirmed the clinical diagnosis. Venous pressure from October 16 to November 10 varied from 16 to 39 cm., with an average pressure of 29 cm.

CASE 6.—33,104. W. H., aged 52, white, male. Admitted Sept. 30, 1914. Clinical diagnosis: Arteriosclerosis; chronic nephritis; hypertension; myocardial insufficiency. For five years had slowly increasing dyspnea, weakness, edema of the ankles and swelling of the abdomen. For almost a year had been able to do no work. Admitted to the hospital with dyspnea, orthopnea, polypnea, ascites, general anasarca, a much enlarged area of cardiac dullness, a blood pressure of 220, right-sided hydrothorax and albuminuria. Phenolsulphonephthalein excretion 15 per cent. in two hours. Showed no improvement under limited fluids, purgation, diuretics and digitalis. Remained in hospital for seven weeks and died shortly after returning home. Venous pressure from October 16 to November 23 varied from 20 to 41 cm. with an average pressure of 29 cm.

CASE 7.—33,922. P. W. R., aged 66, white, male. Admitted March 23, 1914. Clinical diagnosis: Myocardial insufficiency; auricular fibrillation. Dyspnea and palpitation on exertion for the past ten years. Symptoms became severe two years ago with swelling of the legs and abdomen. Was relieved by treatment but has had frequent recurrences. Admitted to the hospital with dyspnea, orthopnea, considerable edema and ascites and enlarged area of cardiac dullness, auricular fibrillation, and an enlarged tender liver. Under limited fluids, purgation and digitalis he improved rapidly and was discharged two weeks after admission. Venous pressure on admission was 26 cm., but fell in a week to 5 cm. The fall in venous pressure preceded a great increase in urine output which came eight days after admission.

CASE 8.—33,350. A. T., aged 44, colored, male. Admitted Oct. 4, 1914. Clinical diagnosis: Myocarditis; myocardial insufficiency. Cardiac symptoms began one month before admission with dyspnea, cough and edema. Examination showed an enlarged area of cardiac dullness with the signs of myocardial insufficiency. Was treated for four weeks and discharged considerably improved. Returned to work but in three weeks another break in compensation occurred. The patient was readmitted to the hospital with the signs of decompensation. Under digitalis, purgation, limited fluids and diuretics the patient's condition was improved and he was discharged after eight weeks in the hospital. The venous pressure on first admission varied from 10 to 24 cm. and during the second admission (November 22-January 19) varied from 18 to 5 cm., with an average pressure of 10.7 cm.

CASE 9.—33,166. C. A., aged 34, colored, male. Admitted Oct. 12, 1914. Clinical diagnosis: Aortic, mitral and myocardial insufficiency. History of rheumatism at 16 years. Cardiac symptoms began in August, 1914. Was improved by two weeks' treatment in the hospital. Returned to work and had his second break in compensation. Was readmitted to the hospital with dyspnea, orthopnea, tachycardia, cough, rusty sputum, râles at both lung bases, an increased area of cardiac dullness and the signs of mitral and aortic insufficiency. Improved somewhat by Karell diet, purgation, digitalis and diuretics and was discharged seventeen days after admission. The venous pressure varied from 28 to 12 cm. between October 15 and 29, with an average pressure of 17.4 cm.

CASE 10.—33,275. C. D., aged 49, white, female. Admitted Nov. 5, 1914. Clinical diagnosis: Arteriosclerosis, hypertension, myocardial insufficiency, chronic nephritis. Housewife with good general health until one year ago when she began having dyspnea, palpitation, orthopnea, edema of the legs and nocturnal polyuria, which became steadily worse. On admission, the patient showed albuminuria, a blood pressure of about 200/140, slight edema of the feet, a moderately dilated heart with signs of myocardial insufficiency, and occasional attacks of paroxysmal dyspnea. Under digitalis, purgation, diuretics and rest, the patient improved somewhat and was discharged seven weeks later. Venous pressure observed from November 12 to December 21 varied from 20 to 9 cm., with an average pressure of 14 cm.

CASE 11.—33,154. D. M., aged 46, white, male. Admitted Oct. 10, 1914. Clinical diagnosis: Mitral insufficiency; myocardial insufficiency; arteriosclerosis; tuberculosis (?). Alcoholic history. Thirteen months before admission began having epigastric pain, shortness of breath and palpitation. Admitted with a slightly enlarged liver, mitral disease and doubtful myocardial insufficiency. Wassermann positive. Suspicious signs of pulmonary tuberculosis. Digitalis and limited fluids. Discharged eight weeks after admission in improved condition. Venous pressure from October 14 to 22 varied from 12 to 8 cm., with an average pressure of 10.4 cm.

CASE 12.—33,531. J. F., aged 42, colored, male. Admitted Jan. 3, 1915. Clinical diagnosis: Syphilis of aorta; aortic insufficiency. Two weeks before admission the patient began having cough, shortness of breath and palpitation. On admission the patient was dyspneic and showed the signs of aortic insufficiency and moderate dilatation. Wassermann positive. Digitalis, purgation and limited fluids. Discharged in three weeks with the heart well compensating. Venous pressure from January 6 to 24 varied from 22 to 10 cm., with an average pressure of 15.2 cm.

CASE 13.—T. T., aged 42, colored, male. Admitted Jan. 19, 1915. Clinical diagnosis: Myocardial insufficiency and aortic insufficiency. Fourth admission to the hospital since March, 1913, for recurrent breaks in cardiac compensation. Admitted with dyspnea, orthopnea, edema, moderate ascites and an enlarged heart with the signs of aortic insufficiency. Under restricted fluids, purgation and digitalis his condition improved gradually. Venous pressure, observed from January 26 to February 16, varied from 20 to 10 cm., with an average pressure of 13.8 cm.

CASE 14.—33,420. W. H. G., aged 51, colored, male. Admitted Dec. 7, 1914. Clinical diagnosis: Myocardial insufficiency; syphilis of aorta. Patient had periodic attacks of dyspnea and palpitation for six years before admission but no edema. Last attack began five weeks before admission and was accompanied by edema of the ankles and swelling of the abdomen. Symptoms became steadily worse and on admission the patient was dyspneic, orthopneic, showed a dilated area of cardiac dullness, auricular fibrillation, a large tender liver, shifting dullness in the flanks, and edema of the ankles. Wassermann positive. Karez diet, limited fluids, purgation, digitalis and strophanthin. Discharged in five weeks with compensation well established. Venous pressure from December 11 to January 10 varied from 22 to 16 cm., with an average pressure of 19 cm.