Staff Meeting Bulletin
Hospitals of the » » »
University of Minnesota

Venous Pressure in Pregnancy

Volume XIV | Friday, April 30, 1943 | Number 23

STAFF MEETING BULLETIN
HOSPITALS OF THE . . .
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William A. O'Brien, M.D.

I. LAST WEEK

Date: April 16, 1943

Place: Recreation Room, Powell Hall

Time: 12:15 to 1:15 P.M.

Program: "Diagnostic Methods in Hepatic Disorders"

Frederick W. Hoffbauer

Discussion
C. J. Watson
G. Evans
George Fahr
I. McQuarrie

Attendance: 126

Gertrude Gunn,
Record Librarian

- - -

II. MEETINGS

1. ANATOMY SEMINAR

Saturday, May 1 at 11:30 a.m. in room 226, Institute of Anatomy.

"Conjoined fetuses (duplicitas superior)"
L. J. Wells

"Cerebellar Connections"
Charles Van Buskirk

- - -

III. STAFF FRY

You are invited to the first annual Staff Fry. Sponsored by the University of Minnesota Hospitals Internes and Residents. Saturday, May 8, 1943, at 8:00 p.m. at the St. Anthony Commercial Club. (Admission by ticket only.)

See your host and buy your ticket.

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IV. GOSSIP

Next Week -- Sorry!

V. WE WELCOME

THE NEW INTERNS - UNIVERSITY OF MINNESOTA HOSPITALS

Bruce Balken - Salt Lake City, Utah.
Washington University Medical School.
Ivan Baronofsky - Milwaukee, Wisconsin
Marquette University.
Dean Benton - Minneapolis, Minnesota
University of Minnesota.
Paul Blake - Hopkins, Minnesota
University of Minnesota.
Alice K. Brill - Westmoreland, Kansas
Syracuse University.
Albert Canfield - Minneapolis, Minnesota
University of Minnesota.
William Chalgren - Minneapolis, Minnesota
University of Minnesota.
Robert W. Emmons - Lapaz, Indiana
University of Indiana.
Helen Haberer - Minneapolis, Minnesota
University of Minnesota.
Richard Horns - Bemidji, Minnesota
University of Minnesota.
Carter Howell - Grinnell, Iowa
Duke Medical School.
Robert Huseby - Minneapolis, Minnesota
University of Minnesota.
Arnold H. Kadish - Detroit, Michigan.
Wayne University.
Julian Knutson - St. Cloud, Minnesota
University of Minnesota.
Richard Lewis -
University of Minnesota.
Allan Moe - Moorhead, Minnesota
University of Minnesota.
Jack Mosely - Monroe, La.
Louisiana State University.
Frank Messinger - LaCrosse, Wisconsin

José Novo - Mexico City, Mexico
National Medical School.
Roger M. Reinecke - Minneapolis, Minnesota
University of Minnesota
Samuel Ritvo - Hartford, Conn.
Yale University.
Donald Sjoding, Minneapolis, Minnesota
University of Minnesota.
Samuel Schwartz - Minneapolis, Minnesota
University of Minnesota.
A. Boyd Thomes - Minneapolis, Minnesota
University of Minnesota.
Charles F. Williams - Indianapolis, Ind.
University of Chicago.
James M. Wilson - South Bend, Indiana
University of Colorado.
Enrique Zuniga - Boston. Universidad Central, Escuela de Med.de Honduras.

VI. VENOUS PRESSURE IN PREGNANCY

Chas. E. McLennan

The writer's interest in venous pressure during pregnancy was aroused a few years ago by a series of reports in the literature (Burwell and others, 1938) indicating a comparatively enormous elevation of pressure in veins of the lower extremities. This was not a new discovery, since Runge had made similar observations in 1924, as had Villaret in 1925 and Rona in 1935, but for the most part these earlier reports had received scant notice. A review of all available literature on venous pressure in pregnancy revealed a number of disputed points which, it seemed, could be clarified by further investigation. There was a particular need for setting up standard values for venous pressure in both upper and lower extremities throughout pregnancy, and it was chiefly with this purpose in mind that the work to be reported here was undertaken. It was hoped, also, that confirmation of the high pressure in the lower extremity might offer an explanation for the edema commonly encountered at that site in pregnancy, and that perhaps the disturbance of fluid movement associated with high venous pressure might be given consideration as a factor in the development of pregnancy toxemias.

Methods of Measurement

Venous pressure may be measured indirectly by some device which indicates the pressure required to collapse a vein. Many such instruments have been described, the one most commonly used being the pneumatic capsule of Hooker and Eyster, or some modification of this. These indirect instruments cannot be recommended since results obtained with them frequently are inaccurate.

All direct methods have been patterned after that of Moritz and von Tabora (1910) and differ only in the nature of the devices employed for registering the intraluminal pressure. In brief, venipuncture is performed with an 18 to 25 gauge needle and the latter is connected to a manometer through a column of saline solution containing an anticoagulant. Graphic records can be made with a float recorder in the manometer or, if desired, photographic records can be made with beams of light reflected from such devices as the Hamilton hypodermic manometer or the glass spoon manometer (Kubicek, Sedgwick and Visscher).

Our apparatus and method

In this study a simple water manometer was used. A reservoir of sterile citrate solution connected to the manometer made it possible to flush the needle and take repeated readings at the site of a single venipuncture. The manometer was mounted on an adjustable standard so that the zero point of the scale could readily be placed 10 cm. above the level of the patient's back, as recommended by Lyons, Kennedy and Burwell. The scale on the manometer could be read in millimeters. The glass and rubber portions of the instrument were kept sterile by filling with 70 per cent alcohol when not in use.

All determinations were made on recumbent subjects who had rested in that position for at least 10 minutes. Clothing was removed from the extremity to be used. In the antecubital vein the technique was essentially that advised by Lyons, Kennedy and Burwell, and in the femoral vein the procedure was similar to that described by Ferris and Wilkins. The femoral venipuncture site was anesthetized with novocaine, but anesthesia was not used in the arm.

An important factor in the technique of direct measurement is the selection of the reference point, or the level at which the zero point of the manometer is placed with respect to the heart. At least nine different reference points have been advocated from time to time by various investigators. The recent exhaustive study of this matter by Lyons, Kennedy and Burwell seems to indicate that the disturbing element of variable

thoracic diameter can be eliminated by placing the zero point in a constant relationship to the level of the table on which the subject lies. The least variable results in a large series of subjects were obtained with the zero point 10 cm. above the table.

There is some question as to the optimum degree of abduction of the arm during venous pressure measurements. In general, the lowest readings are obtained with the arm 45 degrees from the body. Abductions of lesser or greater degree occasionally produce marked elevations of pressure, presumably resulting from compression of the axillary vein.

Within the limits of gauge 14 to gauge 25 the size of the needle through which venous pressure is measured imposes no error on the final readings, but the gauge of the needle affects the time required to attain a constant level of the manometer column.

The role of exercise in raising venous pressure is well known, thus it is essential that the subject be in a resting state. From 4 to 8 minutes are required after cessation of activity before a constant venous pressure value is obtained. Tension of groups of muscles causes no appreciable change in venous pressure unless such tension directly affects the flow of blood between the needle and the thorax.

A number of workers have reported that venous pressure is likely to be above normal limits in obese persons, and we have made this observation on several occasions. It appears that, in certain persons, it is impossible to place the arm in a position which is free from venous compression by fat or muscle. It is not true, however, that there is any constant association between body weight and antecubital venous pressure.

It has been claimed that the use of a tourniquet as an aid in venipuncture gives falsely low values for venous pressure. This apparently is not true, providing sufficient time is allowed after its release for a return to normal circulatory conditions.

Normal values for venous pressure

While many authors have quoted "normal" values for venous pressure, most of these have been simple arithmetic means based on a few observations made in conjunction with studies of venous pressure in various pathologic states. With the exception of Krinsky and Gottlieb's 25 cases, no one has accumulated a large series of readings on normal persons and calculated for these the usual descriptive statistics. For the interested reader, tables in an article by Lyons, Kennedy and Burwell show most of the so-called normal values to be found in the literature. On the whole, there is a reasonable degree of uniformity in the values for venous pressure in arm veins. By indirect procedures, various investigators have obtained arm values ranging from 1 to 15 cm. of water, and by direct methods from 0 to 17 cm. of water. In the lower extremity, however, only a few observations have been reported and these are not in close agreement. The values range from 3 to 28 cm. of water.

A comparatively small number of reports on venous pressure in pregnancy have appeared since 1921. Eight authors found arm venous pressures in excess of 14 cm. of water, the value frequently noted as the extreme upper limit of normal, while 8 others found all values in the normal range. Of 5 reports including determinations in women with toxemias of pregnancy, all indicate that arm venous pressure values may lie above normal limits, although this specific conclusion was reached by only 2 authors. There are 7 reports on venous pressure in the lower extremity during pregnancy and all indicate that the values are elevated above normal. Only 2 investigators have attempted to demonstrate a gradient of pressure in the leg as pregnancy advanced, and the numbers of such observations are too few to permit definite conclusions. There have been no previous observations on venous pressure in the lower extremity during pregnancy toxemia.

Material

The results being reported here are based on 827 determinations of venous pressure in 270 different subjects. 390 determinations were made in the antecubital vein and 437 in the femoral vein. Thirty of the subjects were normal, non-pregnant young women used as controls, 228 were pregnant patients seen repeatedly throughout their pregnancies, and 12 were gynecologic patients with large pelvic tumors. 55 of the pregnant women exhibited signs of some form of hypertensive toxemia (pre-eclampsia, eclampsia, arteriolosclerotic toxemia).

Results of study

In young normal adult females, under precise laboratory conditions, the mean value for antecubital venous pressure in the supine posture was 7.88± 0.31 cm. of water and for femoral venous pressure was 11.43± 0.64 cm. of water. The difference between these means is statistically significant. (Proof of this and of similar statements appearing here may be found in McLennan, Am.Jour.Obst.& Gynec., 45:568, April, 1943).

In normal pregnancy the mean antecubital venous pressure was 7.72± 0.23 cm. of water and immediately after delivery it was 8.03± 0.29 cm., a difference of no significance statistically. And these values are the same as for the non-pregnant group.

Because of a rising femoral venous pressure throughout gestation, it was necessary in the interests of comparison to divide the arm and femoral pressure determinations into a number of small groups (intervals of 4 or 2 weeks). While there were minor fluctuations in the mean values for antecubital venous pressure determined at various times antepartum and postpartum, the range being 6.97± 0.39 to 10.75± 1.66 cm. of water. None of the group means was significantly different frm the controa value for non-pregnant individuals.

The mean values for femoral venous pressure in the various gestational groups showed a more or less constant upward trend throughout pregnancy, the normal control level being exceeded first at about the 13th week of gestation. In the last 2 weeks of pregnancy the mean femoral venous pressure was 24.37± 0.65 cm. of water or 17.9± 0.47 mm. of Hg. It fell abruptly following delivery so that the mean value during the first 48 hours postpartum was 10.08± 0.36 cm. of water, i.e., essentially that of the non-pregnant controls. During the next few postpartum days femoral pressure tended to be below the control level but it rose to normal again as soon as the patients were up and about.

Since toxemia of pregnancy does not commonly appear in the first half of gestation, it was difficult to collect sufficient numbers of pressure determinations in the early months to allow statistical analysis of the data. Prior to the 25th week of pregnancy only simple arithmetic means were calculated. The mean values for antecubital venous pressure in the toxemic patients fell within the range of normally pregnant mean ± three standard errors of the mean. Similarly, the values for femoral pressure in the toxemic patients fell within the normally pregnant range.

In an attempt to determine, if possible, the major cause of the elevated femoral venous pressure, continuous determinations were made in a number of patients during delivery by cesarean section. Representative curves from these experiments will be demonstrated. Femoral venous pressure is exceedingly labile and fluctuates within a wide range during the course of an abdominal operation, particularly in association with such factors as muscular rigidity, breath-holding, straining, talking and crying (under local anesthesia). Certain manipulations by the operator also produced transient rises in venous pressure, for example, palpating the exposed gravid uterus and attempting to rotate it within the abdominal cavity, retracting the abdominal walls, and inadvertently exerting pressure at the site of the venipuncture. In none of the operations was there any appreciable change in venous pressure at the time the peritoneal cavity

was opened. Changes in femoral pressure associated with incising uterine wall, rupturing fetal membranes, delivering the infant, and extracting the placenta were somewhat variable. The only uniform observation was a lack of appreciable diminution in pressure at the moment of delivery of the placenta. It is difficult to say, however, that no major shift in venous pressure occurred at the time of placental separation from the uterine wall since the moment of actual separation could not be determined accurately. Furthermore, the time between removal of the infant and delivery of the placenta was so short that pressure changes apparently attributable to extraction of the fetus undoubtedly masked any additional changes resulting from obliteration of the placental circulation.

A few observations were made on patients during normal labors. In general, the process of labor is associated with wide fluctuations in both antecubital and femoral venous pressure. The tendency for femoral venous pressure to rise during each uterine contraction is much more of a uniform phenomenon than is elevation of the antecubital pressure with each pain. The former seems to be, at least in part, a direct consequence of the contraction of the uterus, while the latter depends chiefly upon the patient's general reaction to uterine pain. For example, breath-holding, bearing down efforts, crying, increased muscular tension, et cetera, all tend to produce elevations in the antecubital venous pressure. Overventilation, on the other hand, may keep the pressure from rising or may actually lower it. In the resting state between uterine contractions the arm venous pressure falls within the normal range. Likewise, femoral venous pressure between contractions corresponds to the expected value for the period of gestation. But even the mildest uterine contraction will produce a noticeable elevation in the femoral reading and this usually may be detected 10 to 15 seconds before the patient is aware of the oncoming contraction. With bearing down efforts in the second stage of labor the femoral venous pressure has been observed to rise more than 30 cm. of water above the resting level (i.e., as high as 60 cm.).

Since elevation of venous pressure in the lower extremities late in pregnancy seemed to be chiefly the result of mechanical pressure by the gravid uterus, the writer thought there might be some relationship between height of venous pressure and size of fetus. To determine the degree of correlation, the weights of 100 newborn infants were compared with femoral venous pressure determinations on their respective mothers made within one week of delivery. The coefficient of correlation was $+ 0.374 \pm 0.1005$, which proved to be significant when subjected to the usual test. However, the result does not indicate a really important relationship between femoral venous pressure and infant weight since the coefficient (r) must be at least +.86 to account for even 50 per cent of the total variation (Treloar).

For comparison with the gravid patients, 12 women with large pelvic tumors were studied in regard to venous pressure preoperatively and postoperatively. Before surgery the femoral pressure always exceeded the antecubital, and the femoral readings were elevated on the basis of the fact that they fell above the range of control mean $\pm$ 3 S.E. In general, large gynecologic tumors are associated with elevations of femoral venous pressure entirely comparable in magnitude to those seen in the latter months of pregnancy. An appreciable lowering of femoral venous pressure occurred in all subjects after removal of the tumors.

Finally, to test further the suggestion (Burwell) that the elevated femoral venous pressure in pregnancy might be due in large part to the arteriovenous-shunt-like vascular arrangement at the placental site, determinations were made on a few patients after intrauterine death of the fetus. Unfortunately only 8 such subjects could be found during the course of the study. In most instances it was impossible to determine with any degree of accuracy the exact date of fetal death, so that difficulties arose in attempting to define the duration of gestation in the various cases. Only two

of the femoral venous pressure values fell below the range of normally pregnant mean pressure ± 2 standard deviations (accounting for more than 95 per cent of the variability). Thus it would appear that death of the fetus in utero usually is not accompanied by a marked fall in femoral venous pressure. But one cannot be certain, of course, that death of the fetus necessarily implies occlusion of the intervillous circulation in the placenta.

Comment

There would seem to be no doubt that venous pressure in the lower extremities begins to rise above its usual levels by the beginning of the second trimester of pregnancy and continues to increase throughout the remainder of pregnancy, the rate of increase being most marked between the 20th and 30th weeks of gestation. It should be noted, however, that while the mean femoral venous pressure in the last 2 weeks of pregnancy was more than double that of the non-pregnant control subjects, it was less than 3 cm. of water above the limit of control mean plus 3 standard deviations. In other words, although individual rises in femoral venous pressure are striking and unmistakable, and although the mean values early and late in pregnancy are clearly far apart, still the average value at term cannot be looked upon as enormously elevated by comparison with the possible range of normal variation in this measurement.

Is the rise in femoral venous pressure of sufficient magnitude to change capillary pressure and influence the movement of fluid through the capillary walls in a manner conforming to the Starling hypothesis? In the light of the observations of Krogh, Landis and Turner, as well as those of Landis and Gibbon, one must conclude that a venous pressure of 24 cm. of water (mean femoral pressure at term) is sufficient to filter fluid into the tissues. However, at venous pressures in the neighborhood of 20 to 25 cm. of water the actual quantity of fluid filtered per minute is not particularly great, and it seems unlikely that this increase in filtration would overburden the mechanisms for the removal of tissue fluid. Furthermore, in the presence of continued venous congestion, some force, presumably tissue pressure, diminishes the power of a given venous pressure to filter fluid from the blood into the tissue spaces. The accumulation of 1.5 cc. of fluid per 100 cc. of tissue results in the rapid removal of fluid even in the presence of a venous pressure of 20 cm. of water (Landis and Gibbon). Additional evidence that venous pressures of 20 to 25 cm. of water are not particularly effective filtering pressures is brought out when filtration is measured indirectly by determining cell volume, hemoglobin and erythrocyte counts (Landis, Jonas, et al). While loss of fluid from blood is conspicuous at venous pressures of 40 to 80 mm. Hg it can barely be detected at a venous pressure of 20 mm. Hg (27 cm. of water).

The experiments of Drury and Jones demonstrated that palpable edema in the leg did not occur until the amount of collected tissue fluid had increased the limb volume by 8 per cent. If this be true, then, in the light of what has just been said about filtration rates at low venous pressures, it seems quite unlikely that the venous pressure existing in the lower extremity in the latter part of pregnancy could lead routinely to the production of clinical, pitting edema. And this is borne out by the fact that while all pregnant women at term exhibit elevated femoral venous pressure, not all have pitting edema of the lower extremities. On the other hand, it must be concluded that the increased venous pressure does tend to promote the accumulation of tissue fluid in the lower extremities, and thus set up subclinical degrees of edema which are easily thrown over into obvious edematous states by some further derangement of the normal mechanism for fluid exchange.

Conclusions

1. Antecubital venous pressure in normal pregnancy is not significantly

different from that in the non-pregnant state and shows no particular trends during the course of pregnancy.

2. Femoral venous pressure in normal pregnancy begins to rise in the early part of the second trimester, rises rather rapidly between the 20th and 30th weeks of gestation, then somewhat more slowly, to reach an average peak value at term of approximately 24 cm. of water. It falls quickly after delivery to non-pregnant levels and apparently lies somewhat below the average control level during the puerperal period of bed rest.

3. Patients with toxemia of pregnancy have antecubital and femoral venous pressures within the range of those for normally pregnant persons.

4. During the course of abdominal cesarean section, femoral venous pressure usually shows a marked decline after removal of the fetus from the uterine cavity, and is not appreciably affected by opening the peritoneal cavity, incising the uterine wall, or delivery of the placenta.

5. Femoral venous pressure rises markedly with each uterine contraction during normal labor; antecubital pressure may exhibit similar but less marked rises in the non-relaxed subject.

6. Fetal weight cannot be predicted accurately from measurements of maternal femoral venous pressure, although there is a mild degree of positive correlation between these two factors.

7. Patients with large pelvic tumors show elevations of femoral venous pressure comparable with those seen late in pregnancy.

8. Pregnant women with dead fetuses in utero appear, for the most part, to have elevations of femoral venous pressure of a magnitude similar to those observed in normal pregnancies with living fetuses, at the same stages of gestation.

9. The evidence obtained from this study does not support the concept that the elevation of femoral venous pressure in pregnancy may be largely the result of an arteriovenous-shunt-like mechanism at the placental site.

10. Reasons are presented for concluding that the level of venous pressure observed in the lower extremities, while admittedly abnormal, is not sufficiently high to produce clinical edema in all pregnant women, although it probably promotes the formation of subclinical edema almost routinely.

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