5-1-1935

Obstetrical shock

Paul E. Anderson
University of Nebraska Medical Center

Follow this and additional works at: http://digitalcommons.unmc.edu/mdtheses

Recommended Citation
OBSTETRICAL SHOCK

Paul E. Anderson

Senior Thesis
1935

University of Nebraska
College of Medicine.
# Table of Contents

**Obstetrical Shock.**

1. History
2. Frequency
3. Phenomena of Obstetrical Shock
4. Etiology of Obstetrical Shock
   a. Predisposing factors
   b. Active factors
5. Physiology of Obstetrical Shock
6. Symptoms
7. Treatment
8. Summary
9. Conclusion
OBSTETRICAL SHOCK

Introduction.

In June 1934, while watching a patient whom I had assisted at delivery a few minutes before, I noticed that the patient seemed to grow suddenly pale and even cyanotic; she did not respond readily when spoken to, and her breathing became irregular, rapid, and shallow, her hands became cold and moist to the touch; and on feeling for her pulse, I found it to be very rapid, thready, and at times imperceptible. The delivery had not been long nor had it been difficult, and there had been no noticeable loss of blood. At present there was no evidence of hemorrhage. Frightened and at a loss to explain the condition, I called the obstetrician who made an immediate diagnosis of obstetrical shock and proceeded to treat the case as a typical case of shock.

The patient was left in a dorsal position, heat pads applied, head lowered, caffeine sodium benzoate administered hypodermically, and normal saline with glucose given while arrangements were made for a blood transfusion. The patient rallied somewhat but remained listless until some three hundred cubic centimeters of whole blood had been administered. Gradually respirations became deeper, the pulse more slow and of greater volume, and in the course of three hours the patient was considerably improved in appearance and fell into what seemed to be a normal sleep. The respirations and pulse became more normal, color returned to the patient's
face, and improvement continued. Convalescence was uneventful.

Because this phenomena was new to me, I made a search of the literature and textbooks available to further inform myself as to its frequency, cause, symptomatology, and treatment. To my surprise, I found only a very few references on the subject in the literature, and only scanty or no reference to the subject of obstetrical shock in the textbooks on Obstetrics. As a result of my desire to study this subject more closely, to determine its frequency, and its treatment, I have chosen this topic for my senior thesis and herein propose to review the subject of obstetrical shock in its entirety, although only briefly, covering in these pages to follow the history, frequency, phenomena of, causes, symptoms, and treatment as discussed in the available literature in the hope that this paper may be of some help to those meeting this problem or phenomena in the practice of obstetrics in their practice of medicine.

History.

The history of shock in regard to pregnancy and labor takes us way back to Old Testament times for we read in I Samuel, the fourth chapter and the 19th and 20th verses that when Eli's daughter-in-law, Phinehas' wife, who was with child near to be delivered, learned of her husband and father-in-law's deaths she bowed herself and travailed for her pains came upon her and she died, presumably from the shock that this news had upon her.

King, 1855, in discussing attentions required subsequent
to delivery during the puerperal state mentions the shock
to the nervous system from labor giving us the following
brief description. "The shock to the nervous system from
labor effects a derangement varying from mere restlessness
to absolute hysteria. In easy labors, the patient soon
recovers from it, requiring only a state of rest and sleep.
When severe it is characterized by symptoms of exhaustion,
with an alteration in the appearance of the eye; an anxious
countenance; derangement of the brain, the sensibility of
which is either diminished or increased; and a disturbance
of the circulatory and respiratory system as manifested by
the pulse, which is slow and labored or rapid and fluttering,
or alternating from slow to rapid, and which must not
be mistaken for the pulse of peritonitis and also by the
hurried, panting breathing. A quick, feeble, fluttering
pulse occurs in the collapse from the nervous shock."

Dorland, 1886, in discussing the management of the
puerperium mentions post-partum shock.

Davis, 1904, discusses the development of shock during
labor both as to causes and treatment. In 1905 this same
author mentions shock as a cause of sudden death during
pregnancy. He here review 25 cases from the literature of
sudden death after delivery of which many are unaccountable
for by autopsy findings. Thus here then we find the first
reports of anyone's attempting to find the true cause by
searching for pathology at autopsy; and here, too, we find
the first search through the literature for a review of

3.
the subject of obstetrical shock. J. Whitridge Williams, 1905, in discussing the above article of Dr. Davis, 1905, cites a case of his own where death was probably due to the shock from mental influences and another case where he suspected the shock due to mental influences and successfully treated by the use of morphine. In his textbook, 1927, Williams again refers to this second case of obstetrical shock due to mental depression.

In 1912 Miles Phillips reports three cases of shock in obstetrics, shock due to, or occurring with complete inversion of the uterus.

Harold Baily, 1917, in an article discussing shock in eclampsia speaks of shock following delivery.

In the more recent literature we find shock recognized as a definite entity and discussed in regard to its etiological factors, both remote and immediate, the physiological changes due to shock and accompanying shock, the symptoms, and definite treatment for shock.

Dr. Miller, 1922, discusses obstetrical shock as a definite entity, giving causes and treatment.

Akerman, 1928, discusses shock cases due to other factors besides hemorrhage, ruptured uterus, or excessive trauma and also reviews the literature on the subject.

Stone, 1928, reports three cases of obstetrical shock in the Yale Clinic in 9 months.

Randall, 1929, reviews the cases of obstetrical shock with their treatment at the Mayo Clinic.
McIlroy, 1930, reviews the cases under her observation from 1921 to 1929.

Phillips, 1931, summarizes the literature to date and discusses obstetrical shock.

Frequency.

Obstetrical shock appears to occur more frequently than is generally though by the average obstetrician. With the more recent literature discussing this subject as a definite entity, giving to it definite causes, symptoms, and methods of treatment more and more cases of the indefinitely understood labor complications are being recognized as shock; and we find reports of cases of obstetrical shock becoming more numerous. The milder cases of shock are less easily recognized as such and so are not reported so that the cases reported are those of a very severe form or even as a cause of death. Obstetrical shock has found a definite place in the maternal mortality records as a cause of death in obstetrics, Kerr, 1933.

Baily, 1917, sites four cases of shock following delivery.

Dame Louise McIlroy and Beatrice Turner, 1930, in a summary of 8,488 obstetrical cases from 1921 to 1929 report 26 deaths or 2.7 percent per 1000 of which six were due to obstetrical shock and four others to obstetrical shock combined with hemorrhage.

Phillips, 1912, reports three cases of shock with acute complete inversion of the uterus.
Dr. Partridge in discussing a paper by Dr. A. H. Miller, 1922, reports two cases of shock deaths but says that shock in obstetrics is a very rare thing. His two cases occurred one with ruptured uterus and the other with hemorrhage.

Audebert reports two cases, one of which complicated a frank attack of eclampsia.

Faucot, according to Stone, 1926, reports two cases in the presence of myocardial weakness and so-called infectious toxemia.

Stone, 1926 reports three cases in the Yale Clinic in six months.

Akerman in reviewing the literature on the frequency of shock reports,

1. Fournier reports one case associated with acute dilatation of the stomach.

2. Gautret reported a case in 1922 of emotional origin during the delivery of an unwanted child (stressing the psychic factor).

3. Four cases seen in consultation with other doctors in his private practice occurring without hemorrhage or excessive trauma.

Akerman in a careful analysis of 10,000 cases of labor with 39 maternal fatalities found six cases where shock was given as the cause of death. In three of these six cases hemorrhage was given as the underlying cause of shock, and in one of these three the hemorrhage was due to acrupto placentae, and the other three were associated with ruptured uterus.
Muret, 1923, and Gross, 1923, report cases during labor with and without hemorrhage. Miller saw it in prolonged labor. Schwarz, 1923, thought it more frequent in nephritic patients.

Shickele observed liver hemorrhage in a patient who died of obstetrical shock.

Driscoll, 1928, reports five deaths, three by shock in a series of 2,323 confinements in the maternity Center Association of New York City. On the Second (Cornell) division at Bellevue Hospital during 1924-1925 he (Driscoll) reports there were 2,310 deliveries with nine cases of shock, two of which ended fatally. He further states, "It is hardly beyond the realm of probability that many deaths attributed to hemorrhage and emboli are in reality due to shock."

Kerr, 1933, says "Obstetrical shock occupies a high place among the causes of death. Indeed, if the cases which develop slight shock are included, this alarming condition may said to be a relatively common occurrence."

While Katz, 1922, in reporting 35 cases of sudden death in maternity patients does not even mention shock.

From these reports then we find that obstetrical shock is apparently not a rare occurrence, although in some reports of maternal deaths and complications it is not even mentioned, which rather leads us to believe it has not been recognized and labeled as such, rather than being not present.
The Phenomena of Shock.

To report this condition we must first be aware of there being such a condition and familiar with the symptom complex that goes to make up this disease entity. What is Shock? What is this Phenomena, if we choose to call it such, that we call and speak about as Obstetrical Shock? To clarify our minds as to what it is let us take some of the descriptions given by the various writers on this subject and see what they imply by the term "Obstetrical Shock" as used by them.

Bernard Mann says "the term obstetrical shock implies a condition occurring in parturient women akin to the shock seen in surgical patients. It does not refer to the temporary collapse or fleeting exhaustion occurring in the women undergoing labor, but rather the classical picture of collapsed circulation and deranged respiratory apparatus which terminates in death if not relieved." Males Phillips says "Obstetrical shock is just the same thing as traumatic or surgical shock. It has the same causes, initiating and predisposing, and must be treated on the same principles. In marked cases the patient lies very still, as if felled, paying no attention to what is going on around her. Anxiety has changed to mental dullness. The skin and mucous membranes are cold, gray or cyanotic. The pulse is almost imperceptible, and as a rule very rapid. Respiration is shallow and quickened, the temperature supormal, and the arterial blood pressure is low, perhaps unrecordable. The condition must
be contrasted with that produced by hemorrhage alone. Here
the patient is mentally alert and acutely anxious, tossing
about or struggling to sit up as she gasps for air." Albert
Miller tells us that obstetrical shock may occur in strong,
healthy women, and in cases where the circumstances of the
delivery do not explain the occurrence of the resulting
depression. "The labor may have been protracted and has
been terminated rapidly, perhaps with the aid of instruments.
There has been no excessive loss of blood. Immediately
after delivery the patient is apparently in a perfectly
normal condition, she has been in the lithotomy position
during the delivery. An anesthesia has been used at intervals
during the labor and to produce unconsciousness and relaxation
during the delivery of the head. The degree of anesthesia
has not been deep and now the patient is rapidly regaining
consciousness. She is moved about in bed, placed in the
dorsal position with the head on pillows, and may be turned
from side to side while the soiled bed is changed. Now
develops one of the most alarming complications ever met with
in practice. There is extreme pallor of the skin and mucous
membranes. The eyes are closed, the conjunctiva is blanched,
the respiration is shallow and almost imperceptible. The
pulse is weak and may be unobtainable. The blood pressure
is low and may be impossible to determine. Consciousness
is depressed. There may be air hunger and some expression
of anxiety on the part of the patient. The skin is at first
9.
dry but later may be bathed in perspiration. The patient lies quietly on her back with entire absence of color in the face and lips, with no perceptible respiratory movement and with no radial pulse. She seems unconscious but rouses when spoken to. Muret describes this form of shock as a condition which resembles a large hemorrhage but occurring without excessive hemorrhage."

Williams, 1905, describes the following case "After the completion of labor I waited about the house for the best part of an hour, and hearing nothing from the nurse upstairs went to bid my patient goodbye. In spite of the fact that her pulse was good and the uterus well contracted, I did not like her appearance and decided to watch her a while longer. As I did so her face gradually assumed a collapsed appearance and became haggard, with the eyes far back in their sockets. The pulse slowly became more rapid and in a few hours her condition became alarming. Although there were no symptoms of hemorrhage, I introduced my hand into the uterus to make sure that it was uninjured, and found it tightly contracted, with no evidence of rupture. Six hours after delivery her condition was so alarming that I thought she was going to die, and accordingly asked permission to call a friend in consultation. As soon as the consultant saw the patient he said she was going to die, though he was unable to throw any light upon the cause of the condition. As the patient became worse and worse, but was quite conscious and quite prepared to die, I recalled the fact that she had
The Coxveld a fatal issue, and I wondered whether her condition might not be connected in some way with her forbodings. Acting on this supposition I gave her 1/2 grain of morphine by hypodermic which almost immediately put her to sleep. In a few minutes the pulse became slower and fuller and within an hour had become so satisfactory that I felt justified in leaving her. The next morning her condition was normal, and she made a very satisfactory recovery."

Baily says that shock may be described as a partial or complete loss of central vasomotor control due to profound injury to the tissue, loss of blood, or to severe heart depression. This definition would assume that collapse and shock are of the same nature differing only in degree.

Shickle, according to Stone, concluded that so-called obstetrical shock is a definite entity dependent upon anatomical changes but that, in the usual sense it bears only the most superficial analogy to surgical shock. Stone in the same article says that Riviere agrees with Shickle emphasizing, however, that labor is the active agent in exciting the predisposed tissues.

Driscoll in discussing symptoms says that "the pallor of the cool damp skin, the unquenchable thirst, the jerky respiratory effort, the restlessness, the weak fluttering pulse, and eventually unconsciousness are well known symptoms, but too often the diagnosis of shock is not made until the picture is complete."

Cook and Briggs and Crile believe that the earliest manifestation of shock and collapse is a fall in the blood pressure.
Schwarz in writing about blood pressure changes following pregnancy describes the following case of a nephritic toxemia patient following delivery. "About thirty minutes after delivery the patient developed a cyanosis, became faint, her respirations were shallow and increased, and her pulse was imperceptible." We can easily guess that this patient was in state of shock.

If from the writings of Jones, Phillips, Mann, and Baily we conclude that obstetrical shock and traumatic or surgical shock are the same or very similar, we can then easily turn to Cannon's work on traumatic shock and study his descriptions and clarification of the term "Shock" hoping that it will throw more light on our special phase of shock found in obstetrics.

Cannon quoting Fischer (Volkmann's Samml. Klin Vorträge, 1870 No. 10 page 70) gives the following case report. "The patient, a strong and perfectly healthy young man, was struck in the abdomen by the pole of a carriage drawn by runaway horses. We have not been able, after careful examination, to find any trace of injury to any of the internal organs. Nevertheless, the grave symptoms and the alarming look which he still presents made their appearance immediately after the accident. He lies perfectly quiet and pays no attention whatever to events about him. His skin and such parts of the mucous membranes as are visible are as pale as maroie, and his hands and his lips have a bluish tinge. Large drops of sweat hang on his forehead and eyebrows,
his whole body feels cold to the hand, and a thermometer indicates a degree and a half Centigrade in the axilla and a degree in the rectum, below normal. Sensibility is much blunted over the whole body, and only when a very painful impression is made on the patient does he fretfully pull a wry face and make a languid defensive movement. If the limbs are lifted and then let go, they immediately fall as if dead. The urine is scanty and dense, but free from any traces of sugar or albumin. The pulse is almost imperceptible and very rapid. The arteries are small and the tension is very low. The patient is conscious but replies slowly and only when repeatedly and importunately questioned. On being thus questioned, he complains of cold, faintness, and deadness of the extremities. His respiration is characterized by long, deep, sighing inspirations, alternating with very superficial ones, which are scarcely visible or audible. While being brought to the hospital he vomited several times, and nausea and hiccup still remain. His pallor, cold skin, and hoarse voice immediately recall the appearance of a cholera patient; characteristic dejections are alone wanting to make the resemblance complete."

Cannon also gives the following description by Warren, (Surgical Pathology and Therapeutics, Philadelphia, 1895, page 378) "A patient is brought into the hospital with a compound comminuted fracture where the bleeding has been slight. As the litter is gently deposited on the floor, he makes no effort to move or look about him. He lies staring
at the surgeon with an expression of complete indifference as to his condition. There is no movement of the muscles of his face; the eyes, which are deeply sunken in their sockets, have a weird uncanny look. The features are pinched and the face shrunken. A cold, clammy sweat exudes from the pores of the skin, which has an appearance of profound anemia. The lips are bloodless and the fingers and nails are blue. The pulse is almost imperceptible; a weak, thread-like stream, may, however, be detected in the radial artery. The thermometer placed in the rectum, registers 96 or 97 degrees Fahrenheit. The muscles are not paralyzed anywhere, but the patient seems disinclined to make any muscular effort. Even respiratory movements seem for the time to be reduced to a minimum. Occasionally the patient may feebly throw about one of his limbs and give vent to a hoarse, weak groan. There is no insensibility, but he is strongly apathetic, and seems to realize but imperfectly the full meaning of the questions put to him. It is of no use to attempt an operation until appropriate remedies have brought about a reaction. The pulse, however, does not respond; it grows feebler, and finally disappears, and 'this momentary pause in the act of death' is soon followed by the grim reality. A post-mortem examination reveals no visible changes in the internal organs."

Cannon makes the following resume as being typical of the complex called shock. "Shock is a general bodily state which occurs after severe injury and which is characterized
by a persistent reduced arterial pressure, by a rapid thready pulse, by a pallid or grayish or slightly cyanotic appearance of the skin which is cold and moist with sweat, by thirst, by superficial rapid respiration, and commonly by vomiting, and restlessness, by a lessened sensibility and often by a somewhat dulled mental state."

In studying the descriptions for shock both traumatic and obstetrical we see that the two types of shock are practically the same as to symptoms and the phenomena of shock can be assumed to arise due to the same fundamental changes in the functioning of the body. Therefore, in thinking and dealing with obstetrical shock, we must realize that it is similar in symptoms, and cause to traumatic shock and must therefore be recognized and treated similarly also.

From these above descriptions of shock we get a more or less definite group of symptoms or phenomena of shock that gives a characteristic picture of the complex called shock. However, we also must admit that the term "shock" as used by most of the above writers is loosely applied and covers a large number of symptoms, possible causes, or etiological factors, which are not in many phases very closely related to each other. We have need for a more rigid definition of shock in terms of both surgery and obstetrics. A closer study of the causes and physiology of shock should clarify our definition greatly, giving us a more definite understanding of this phenomena of shock whether traumatic, or as we are
particularly interested in this paper, obstetrical shock.

The symptoms of this phenomena of shock that seem to be mentioned by all the above writers as typical include the following. A fall in blood pressure, Cannon states that this lowered pressure is the central feature, or one of the most essential features, of shock. The lowered blood pressure gives us, due to the slowed circulation, the thirst, the rapid superficial respiration and the slightly subnormal temperature. Furthermore, the degree of reduction of pressure may be regarded as a fairly satisfactory index of the degree of shock which is prevailing. "There is no more important aspect of the complex of established shock than the state of the circulation as indicated by the sphygmomanometer." With the above symptoms we find a cold skin, moist, maybe cyanotic, a pulse rapid that may be or may not be imperceptible. The respirations, besides being shallow, are quickened and may be irregular. The patient is not unconscious yet is mentally dulled, the eyes are closed and seem to be sunken into their sockets and the features of the face are pinched and the face shrunken. These symptoms may be said to give the typical picture of shock, whether in trauma or obstetrics.

Etiology of Shock.

To cover all the possible factors or combination of factors that may play a part in the causation of shock or obstetrical shock in particular, giving the above complex of symptoms is not possible in this paper; therefore, we
shall limit this discussion to those phases that directly or closely indirectly play a part in shock.

That labor is the active agent in exciting the predisposed tissues to shock is mentioned by Riviere according to Stone. Orlic in his work on shock proposes a less concrete cause, namely, the combination of fear and anxiety with pain leading to a state of mental and physical exhaustion, and eventually to shock. Certainly all parturients suffer from fear, anxiety, and pain.

Dale showed experimentally that shock is more easily induced in etherized dogs. Suggesting that an anesthesia plays a part in the cause of shock, and Hofbauer has pointed out that there are more sources for an increased production of histamine in pregnancy and that ether greatly intensifies any existing toxemias. Miller, Phillips, Mann, and Driscoll all mention an anesthetic, especially ether and chloroform as being a contributory cause in the production of shock.

Phillips lists body fatigue from prolonged muscular exertion, cold from exposure, deprivation of food and water, sweating, anesthesia, toxemias of pregnancy, infections, and emotion as predisposing causes. McIlroy says, "Fear, anxiety, and intense suffering during labor are predisposing factors."

Williams says that prolonged and very difficult and very painful labors, the incident of loss of rest, imperfect rest, imperfect nutrition, and mental excitement may be looked upon as predisposing causes. Kerr also mentions emotion as a cause, and emphasizes acidosis as inducing
obstetrical shock when labor is protracted.

Toxemia, hemorrhage, a long arduous labor with anxiety and fear, vomiting, profuse perspiration, and excessive respiratory effort result in the loss of body fluids. Fatigue, and a lowered alkaline reserve are spoken of by Driscoll as having an indirect effect in producing shock.

From the above causes that may predispose or be the background on which there is developed a case of shock, we come now to consider those causes that will directly bring about a state of shock in the patient.

A sudden fall in blood pressure is the immediate cause of shock and gives us the resultant shock state with its other manifestations says Cannon. The reason for this fall in blood pressure will be discussed under the physiology of shock.

Miller states that in obstetrical shock we are dealing with a mechanical problem of two factors, relaxation and posture. The abdominal muscles have been relaxed with an anesthesia, the uterus has been emptied rather suddenly, contributing to a further drop in the intra-abdominal pressure. The patient has been for some time in the lithotomy position. Then she has been lowered to a dorsal position, moved about in bed and her head raised on pillows. As a result the entire mass of blood is steadily accumulating in the dilated capillaries and in the abdominal veins. Phillips agrees with Miller in that the sudden lowering of intra-abdominal blood pressure which follows the emptying of the uterus is
one of the chief factors, and cites the experiments on rabbits described by McCleod as the Lenard Hill experiments on gravity shock. "Gravity shock is caused by stagnation of the blood in the splanchnic vessels and consequent inadequate filling of the heart in diastole. It occurs when the erect position is assumed. In animals in which the mechanism which ordinarily compensates for the tendency of gravity to make the blood flow to the dependent parts is inadequate. Thus, when a domesticated rabbit with a large pendulous abdomen is held in the vertical position for any length of time the animal gradually passed into a shocked condition and may die in a short time (from twenty to thirty minutes.) Observation of the blood vessels of the ear or a record of arterial blood pressure will show that the cause of shock in this case has been a great curtailment of the blood supply to the upper part of the body and therefore to the nerve centers. The shock is entirely dependent on the laxity of the abdominal muscles for if a binder is applied to the abdomen or if the experiment is on a rabbit whose abdominal musculature is in a good condition, gravity shock does not occur. In men whose compensation for the erect posture is highly developed, shock from gravity occurs only when there has been some other considerable upset in the circulatory mechanism." Schwarz in discussing blood pressure drops following delivery also cites these experiments as a proof of sudden relaxed abdominal pressure causing a drop
in blood pressure. In normal pregnancy the vasomotor mechanism is sufficient to take care of the sudden emptying of the uterus, and relaxation of the abdominal musculature. In cases of toxemia, or other predisposing factors, however, due to the already strained vasomotor mechanism, perhaps chiefly influenced by the presence of toxic substances, compensation does not take place and there results a great splanchnic dilatation with the resultant insufficient amount of filling of the heart during diastole.

Cannon suggests that traumatic shock is caused by some poisonous substance which is liberated from the damaged tissues and causes widespread relaxation of the capillaries. Its effect is to add to the stagnation of blood in the already dilated capillaries of the muscles.

Phillips further suggests that the unexpected secondary phase of obstetrical shock may be due to extensive lacerations of the large muscles of the pelvic floor, the levatores ani especially, when such injuries are not accompanied by deep tears of the perineal body and vagina sufficient to expose the lacerated muscles. Hemorrhage is often a considerable factor, yet of itself it does not give rise to a prolonged permanent fall of blood pressure unless more than twenty-five percent of the body blood is lost. Combined with trauma it naturally aggravates shock.

Baily also cites the fall in blood pressure after delivery due to relief of pain and excitement, and possibly to some dilatation of the splanchnic area following the diminution of the size of the uterus as being a direct cause of shock.
Gasser and Erlanger have amply demonstrated in all forms of experimental shock and in all grades of injury that one of the most outstanding features of shock, as a rule, is collapse due to the lack of fluid in actual circulation. Tweedy and Wrench state that great shock is the chief symptom of acute inversion of the uterus and remains as long as the uterus is inverted. Phillips, 1912, also says that acute complete inversion of the uterus always causes shock.

McCleod sponsors, as the cause of shock, the bruising of tissue and the subsequent absorption of highly toxic proteins, and the sudden release of intra-abdominal pressure, both of these factors being present at childbirth.

Mann and Isreal suggest that "The excessive perspiration and deep respirations during labor effect a great loss of body fluids, which contributes readily to an already induced shock. A sudden loss of body fluid is enough in itself to cause shock for the circulation may not be maintained. It is very apparent that many factors, crushing of tissue, sudden decrease in abdominal tension, acidosis (caused in normal labors by excessive respiratory effects, starvation, and the absorption of waste products or excess energy), loss of fluids, pain and anesthesia are present in the course of a spontaneous labor and delivery which may precipitate a state of shock. All of these are obviously aggravated by such complications as hemorrhage, rupture of the uterus, toxemia, or ectopic pregnancy."

One of the two cases of shock reported by Audebert complicated a frank attack of eclampsia. Paucot (Stone)
reported two cases that involved the sudden decompression of an over-distended uterus in the presence of myocardial weakness and so-called infectious toxemia. Akerman reports a case of Fournier's where shock was associated with acute dilatation of the stomach in a labor terminated by high forceps extraction, and a case by Gautert of an emotional origin. Akerman gives hemorrhage and excessive trauma as direct causes.

Kerr says that shock is a result of a combination of relatively minor unfavorable conditions, none of which individually is responsible for the disaster.

Cannon lays great stress on the sustaining factors which result from the shock itself. These are (1) the lowered metabolism resulting from the fall of blood pressure, (2) the diminished heat production, (3) the defective oxygen supply to all the tissues and to the nervous tissue in particular, and (4) the damage to the walls of the capillaries. "These injuries to the elements which are essential to the maintenance of an efficient circulation continue the state of shock which has been originated by other factors and may lower still further the already low arterial pressure. A series of vicious circles are set up which lead to a depression of the circulation so profound that it cannot be restored and the patient dies."

Physiology of Shock.

What are the physiological processes taking place in the body that give us a state of shock? How is shock produced? Cook and Briggs state that the factors efficient in its production act by way of the peripheral afferent nerves on the
central nervous axis. The resultant underlying condition, whatever it may be, which is responsible for the clinical or experimental phenomena of shock, affects chiefly the medullary vasomotor center, the periphery contributing only secondarily to the elaboration of the picture. As a consequence to this altered condition of the center, there is produced an abnormally low blood pressure within the whole arterial system, and to this marked hypotension are due all the features presented by the patient in shock.

A. L. Pimenta Buena says that there is a compression and decompression interdependence between the blood pressure in the abdomen and in the skull. In the abdomen the elastic walls allow great fluctuation in the pressure without harm, but the unyielding skull may entail grave accidents when the pressure fluctuates here. Congestion is the splanchnic area from the paralysis of the sympathetic nervous system is accompanied by corresponding anemia and decompression in the skull, and this is the main mechanism of traumatic, operative, anaphylactic, and other shocks, as he sets forth in detail.

Again referring to Cannon, he gives the following modern conception of shock. He says that histamine or some closely allied substance and resembling it in action is present in all tissues at all times and is readily liberated as a result of even trivial injuries. This poisonous substance liberated from damaged tissues causes widespread relaxation of the capillaries and also increases the permeability of the capillary walls and so produces a reduction of blood volume by escape...
of plasma into the tissues. The amount of blood plasma circulating to the other parts of the body, particularly to the vital centers of the brain, is greatly diminished.

Phillips tells us that shock is due to afferent impulses causing reflex vaso-dilatation and a consequent marked fall in blood pressure but does not mention the reason for these impulses. For long it was thought that the blood vessels of the abdominal viscera were chiefly involved, but it is now recognized that it is in the reflexly dilated capillaries of the skeletal muscles that the blood is dammed up, and for the time being, lost to the circulation. The consequent diminished blood supply to the brain will result in faintness and even is loss of consciousness. In obstetrics the above is true with the added factor that we have here also the more or less suddenly lowering of the intra-abdominal blood pressure which follows the emptying of the uterus. The Lenard Hill experiments previously referred to bear out this point.

Baily and Driscoll say that shock may occur in an apparently normal labor because of the effect of pain on the respiratory mechanism bringing about an excessive elimination of carbon dioxide through over breathing and with food abstinence giving shock from acidosis and exhaustion. The cardiac depression which may result from an anoxemia of cardiac muscle, occurs during an apneic period.

Gasser and Erlanger in clinical medicine estimations of blood volume in patients with shock have repeatedly shown the blood volume to be diminished. Baily says that shock
is a complete loss of central vasomotor control. This is in harmony with Cannon's statement, that it is due to profound injury to the tissue, loss of blood, or to severe heart depression. Cook and Briggs explain the fall in blood pressure as being due to relief of pain and excitement, and possibly to some dilatation of the splanchnic area following the diminution of the size of the uterus.

Schwarz, citing Hill's experiments, says the drop of blood pressure may be due to the dilatation of the splanchnic area and sudden change in abdominal pressure, and the marked laxity of the abdominal wall. Adding, that in shock the normal vasomotor mechanism has failed to take care of the sudden emptying of the uterus and relaxed abdominal musculature. Compensation does not take place, and we get a great splanchnic dilatation, with insufficient flow of blood into the heart during diastole.

Miller mentions the accumulation of blood in the dilated capillaries and the abdominal veins.

McCleod tells us that the fall in blood pressure, with the arterioles maintained in a contracted state, is due to an inadequate output of blood from the heart.

Dale and Richards have shown that the fundamental cause of low blood pressure is inadequate blood flow to the heart. They have also shown that histamine, while constricting the arterioles, dilate the capillaries and at the same time increases the permeability of the walls and so permits the plasma to leak through. Also they observed that with ether
anesthesia the animal is ten times more susceptible to the action of histamine, indicating that whereas the normal animal is able to combat the toxic action of histamine, ether greatly depresses this power. Histamine does not readily produce shock in nitrous oxide anesthesia. They tell us that the poisoning effect of ether persists for some time after the anesthesia is removed and is no doubt dependent upon a toxic action on the endothelium of the capillaries for it is particularly seen in such animals that concentration of the blood is evident after histamine. Hemorrhage predisposes to histamine shock. Bacterial toxins have effects like those of histamine. The facts warrant the suggestion that shock may be due to liberation from the damaged tissues, particularly muscles, and the viscera of a toxic substance acting like histamine. Cannon (McCleod p. 520) has demonstrated that this is true.

This diminished heart output of blood must be due to the interference with the heart action itself independently of the blood carried to this organ or a deficiency in the filling of the ventricles during diastole. Experiments (McCleod) have shown that the heart action itself has not been interfered with; therefore, the deficient discharge of blood must be dependent upon improper diastolic filling. After this condition of oligemia has set in, it becomes progressively worse because of the weakening of the heart muscle, consequent upon the failing blood supply through the coronary vessels,
and this again upon a curtailment of the amount of blood in actual circulation. The cause of this oligemia may be due to mechanical interference with the blood return in the inferior vena cava caused by (1) excessive movements of the thorax, (2) excessive ventilation, stagnation of blood in the splanchnic areas—the patient bleeds into his own splanchnic area blood vessels (capillaries and venules) because these have lost their tone, and (3) further curtailment of blood in that much is lost in the capillaries of the tissues outside of the abdomen. Cannon has shown that the blood is concentrated in these capillaries making the blood much more concentrated indicating that plasma must have left the blood in the capillaries.

Since the fall of blood pressure is the primary sign and the fundamental change on which shock is produced we then can account for the other changes as being resultant from this primary change. The disturbances in sensation and motion, and fall in body temperature thus follow the fall in blood pressure. The raised threshold of sensory stimulation is no doubt an effect of the low blood pressure, as is the muscular weakness. The fall of body temperature is dependent upon the muscular inefficiency.

The Symptoms of Shock.

The patient in shock displays his symptoms dramatically and is self diagnosed. The symptoms of shock form a definite recognizable clinical picture.

Baily, Crowell, Randall, Miller, Phillips, Mann, and Driscoll mention the following symptoms in writing about
obstetrical shock which we also find Crile and Cannon listing as the symptoms of surgical shock.

1. Lowered arterial blood pressure.
2. Increased pulse rate--may be thready or even imperceptible.
3. Increased respiratory rate--increased ventilation of the lung.
4. Cold moist skin.
5. Pallor--sometimes a yellowish tint, even cyanotic.
6. Cyanotic nails.
7. Reduced metabolism.
8. Loss of body heat.
9. Loss of muscular power.
10. Loss of mental power.
11. Shrunken facies.
12. Dull, listless, expressionless eyes with heavy lids.
13. Increased hydrogen ion concentration of the blood--diminished reserve alkalie.
14. Concentration of the blood--relatively high red blood count.

Treatment.

The treatments of shock, both surgical and obstetrical, have been varied and many, meeting with all gradations of success in the past, and changing as we have come to know more about the phenomena and the causes of the condition. At present, the treatment has become fairly well established along definite lines with certain universal methods adopted because of the physiological proof by experience and experi-
mentation that certain solutions work physiologically in reestablishing the normal body functions that are out of order or only partially functioning in the phenomena of shock.

We must first recognize that prevention wherever and whenever possible is the primary step in treatment. To forestall a possible shock condition developing in an obstetrical patient we can and should carry out the following routine as far as feasible.

1. Prevent fatigue by sufficient rest. Mann suggests small doses of morphine judiciously used in slightly prolonged labor or threatened collapse to quiet and rest the patient. Phillips suggests the use of scopalamine grains 1/100 every hour for three doses and then every two hours for pain and rest if necessary. Dr. Pollard, however, is opposed to the use of scopalamine at all and advocates rather the use of sodium amytal if a sedative is thought necessary.

2. Plenty of food and fluid should be given to prevent dehydration and to keep up the energy supply.

3. Prevent or allay nervous strain as far as possible. McIlroy lays emphasis on the prevention of emotional shock by pre-natal schooling to dispell apprehension. Possibly, the best way to do this is to go over the procedure of the pains, labor, and delivery with the patient (depending on the patient), telling her what to expect, not to be afraid, dispelling fear, and reassuring her that all is apparently in fine shape for a normal delivery. Here again sedatives should be used if necessary during the labor to quiet the
patient's fears and rest her.

4. Care should be taken to prevent cold from exposure both during and preceding the delivery as well as post partum.

5. Care should be taken to prevent excessive trauma, hemorrhage, and unduly long labors.

6. Repeated blood pressure readings should be taken to assure the doctor that circulation is good and also to warn him of any sudden changes.

7. A more careful use of anesthetics, especially chloroform and ether should be practiced.

8. In cases of toxemia the patient should be induced early and delivered in the least traumatic way. Here chloroform and ether should never be used. Rather use gas and oxygen. Phillips suggests giving glucose to these patients by mouth before and during the labor if possible.

9. In suspected infected cases the wounds should be left open applying sterile saline or glycerine dressings till the acute stage of the infection is over.

10. Miller suggests that if we leave the patient in the normal position after the delivery and raise the foot of the bed eighteen inches we do much to prevent shock.

If in spite of our endeavors above to prevent shock, shock does develop it then becomes our active duty to recognize it early and treat it actively. Our first point in the treat-
ment after occurrence is the recognition of the condition. Since the first and primary symptom of shock is a fall in blood pressure, its treatment becomes of primary importance in the treatment of shock.

Cook and Briggs tells us that in shock "hypotension is not merely a symptom of value in diagnosis but it is in itself the chief source of danger to life, and in too many cases the actual immediate cause of death. The lasting relief of hypotension by therapeutic measures means the radical cure of shock. And it is the first duty of the attendant on a case of shock to raise as quickly as possible the dangerously low blood pressure and to maintain it at a level compatible with life." Keith has shown (Randall) that Nature's first reaction to shock is to supply fluid and not erythrocytes to the depleted circulation. Cannon tells us that the stagnant blood is not in the veins or arteries but in the capillaries as shown by chart I which gives a comparison of the red counts, capillary and venous, in cases of severe and moderate shock and in patients without shock.

Cook and Briggs cite a series of ten cases in shock where central stimulants were used to raise the systolic blood pressure. In all cases, strychnine was given, in seven digitalis was also, and in one case (with positive effect) cocaine was employed. 'All the drugs were administered hypodermically. The smallest doses given were strychnine grains 1/60, and for digitalin grains 1/40, the largest doses were grains 1/10 of each. In most of the cases the unit dose was a combination of 1/20 grains of each, repeated as 31.
Comparison of Red blood counts, capillary and venous, in cases of severe and moderate shock, and in patients without shock.
demanded by the course of the blood pressure. Of the ten cases five showed permanent improvement with the blood pressure returning more nearly to the normal with each successive stimulant hypodermic injection until the physiological level was reached and maintained without further treatment. Three other cases improved temporarily (10-20 mm Hg rise in blood pressure) lasting from one-half to one and a half hours, and two cases with no rise in blood pressure but without any further fall in blood pressure by the hypodermics given. In these cases where the central stimulants were used effect- ively in securing a rise in blood pressure to a safe level visible signs of improvement in the general condition were observed. One of the cases is illustrated by chart II. Here strychnine was effective in raising and maintaining the blood pressure.

Vasoconstriction of the arterioles by adrenalin intrave- nously gives a temporary rise.

Pituitrin acts on the smooth muscle of the arterioles constricting the arterioles and gives a more lasting effect than adrenalin. This constriction in the tips of the arterial tree causes more accumulation of blood in the arteries and leads to a temporary better flow through heart muscle and cerebral vessels; but it is only temporary because the body is suffering from diminished quantity of blood and the volume flow in the capillaries is not improved. What is needed is an increased nutritive flow through the capillaries all over the body, obtained by an increase of volume flow. This, says Cannon cannot be accomplished by medication. He further states that the use of stimulant drugs such as strychnine,
Combined shock and hemorrhage in twin labor, after prolonged forcible attempts at instrumental delivery. Strychnine in large amount producing immediate and permanent rise in blood pressure.
and vasoconstrictor drugs like pituitrin and adrenalin have practically disappeared for treatment of shock since the World War.

What then shall we use to increase the volume flow and by so doing raise the blood pressure to that compatible with life?

Blood transfusion if a donor is quickly available is the universal method of choice. Yet in the absence of the possibilities of a transfusion or while arrangements are being made for the transfusion other methods must be resorted to.

Gessell has pointed out by experimentation that a relatively small loss of blood greatly reduces the volume flow through the peripheral organs and conversely when the circulation is failing from a low content of the vessels a slight increase of blood volume will greatly increase the peripheral flow. The nutrient flow may be increased several percent by injection of an inert solution, an increase out of all proportion to the dilution of the blood produced by the added fluid.

Rous and Wilson state that up to 75 percent of the total hemoglobin may be safely removed if the bulk of the circulation fluid is maintained. Robertson and Bock have proved that in cases of reduced blood volume fluid administered in large amounts by way of the alimentary tract is to a high degree retained in the circulation. Crile advocates large quantities of fluids by mouth, by rectum, and subcutaneously. However,
Cannon states that the injection of normal saline intravenously only temporarily effects the blood pressure and in a short time it is as low or lower than before injection. Chart III illustrates this.

Cook and Briggs tell us that normal saline given subcutaneously does not produce a rise in blood pressure but tends to cause a fall in blood pressure as soon as local tension is produced.

Bayliss has shown experimentally that a gum salt solution will restore permanently a low blood pressure produced by the removal of 40 percent of the estimated blood volume; while Meek and Gasser and Drummond and Taylor have declared gum salt solution harmless for man. Cannon in Chart IV shows the effect of an injection of gum-salt solution in building up a satisfactory blood pressure.

De Kruif has subjected gum-salt solution to very thorough tests as to its toxicity, with negative results.

Cannon states that all experimental testimony indicates that the properly prepared solution is innocuous to man.

Erlanger and Gasser by experiments on animals with 25 percent gum in 18 percent glucose believe this hypertonic solution to:

a. Draw fluid from the tissues into the blood stream and thus increase the blood volume.

b. Maintain the increased volume through some property of the gum acacia.

c. Dilate the arterioles through a specific action.
Two pints of normal salt solution administered intravenously on admission; immediate rise of systolic pressure to 75 mm. but within an hour a fall to a lower level than before. (From Fraser and Cowell).
Intravenous injection of gum salt solution during operation. Blood pressure 80 mm. Hg. and fair pulse. Next day blood pressure, 120 mm. and later 140 mm. where it continued (from Fraser and Cowell).
of the hypertonic glucose.

d. Increases the energy and food supply of the heart.

e. Be an augmentation to metabolism.

And on testing this method on man they found the solution was not only innocuous, but produced results strongly suggestive of beneficial action.

Taussig reports a case so treated by Dr. Erlanger successfully.

Mann tells us that the best solution for raising blood volume and combating shock is 6 percent gum acacia solution.

Farrar has more recently advocated this solution for obstetrical usage. In explaining the success of this solution she gives the following report on the physiological effects produced by this solution; "During any surgical operation, shock, or hemorrhage, the acid by-products of the body are greatly increased. If the bicarbonates of the blood which carry the acid carbon dioxide gas to the alveoli of the lungs are present in large amounts the combining power of the blood, that is, the ability of the bicarbonates to unite with the carbon dioxide, is high and lung ventilation is maintained. If the bicarbonates are low or the acid by-products greatly increased, a condition of intracellular acidosis results which may endanger the life of the patient if the condition be prolonged. Carbohydrates are necessary for the complete oxidation of acetic acid. Glucose is normally present in the blood and is assimilated in this form without further
metabolism. An individual will absorb .8 grams of glucose for each kilogram of body weight without the production of a glycosuria and this rate can be maintained for several hours if desired. Gum acacia is a colloid of the same viscosity as that of the blood, and when added in a 6 percent solution to a 20 percent glucose solution will keep in the blood stream the water which the glucose attracts from the tissues. A satisfactory blood pressure is thus maintained, and the loss of bicarbonates into the circulation prevented. If the blood pressure is maintained and the acid by-products completely oxidized, acidosis during operation will be prevented. Salt solution does not maintain blood pressure because the salt is taken up by tissues as the latter attracts the water. A small volume of a higher viscosity such as glucose and gum acacia is therefore preferable because the blood is not diluted, and the burden on the heart is consequently lessened." In summarizing the indications for the solution Farrar says that it is of great value in combating acidosis, preventing post-operative vomiting, in cases of shock where it is necessary or desirable to maintain a normal blood pressure, in hemorrhage, and to promote diuresis.

Driscoll says that the crystalloid action of the glucose promotes a reflux of the escaped fluid from the surrounding tissues into the blood vessels. The gum acacia by its colloid action raises and maintains the blood pressure, slows the pulse due possibly to direct action on the heart; and respiration, likewise, improves. Blood pressure will be maintained
approximately two hours, then more solution should be given. He illustrates the effect of the solution in the following two cases so treated.

Case 1. (Chart V)

M. D. admitted to Bellevue Hospital, April 10, 1927. Age, 25 years. Para ii. Diagnosis of placenta previa, hemorrhage and shock. The patient had been under the care of a midwife for the past three weeks. There had been a small amount of bleeding daily during that time. Upon admission from the ambulance, the patient was complaining of faintness, nausea, and dizziness. The skin and mucous membranes were pallid, perspiration was free, the radial pulse was imperceptible, and vaginal bleeding quite profuse. She was 37 weeks pregnant. The fetal heart could not be heard. The blood pressure was 40/16. The patient was immediately placed in the Trendelenberg position and external heat applied. Morphine grains 1/4 was given. After 1000 cubic centimeters of saline by hypodermoclysis at 1:40 A. M. the blood pressure was 64/40. 250 cubic centimeters of gum glucose was given intravenously beginning at 2:20 A. M. Thereafter the blood pressure was 90/64. The bleeding had ceased. At 3:50 A. M. 500 cubic centimeters of blood were transfused by the direct method while a DeRibes bag was inserted in the cervix. The blood pressure was 92/70. The bag was expelled spontaneously at 5:15 A. M., and under ether anesthesia a version and breech extraction was done. The blood pressure fell to 84/68, but forty-five minutes later it was 100/78. At 1 P. M. the blood pressure was 80/60. It continued to rise after this time and the patient recovered uneventfully.
The effective use of gum glucose in raising blood pressure in central placenta praevia with hemorrhage and shock.
Case 2 (chart VI).

A. C. admitted to Bellevue Hospital March 29, 1937. Age, 31. Para i. After a labor of 29 hours, the patient was delivered by a breech extraction, following episiotomy, and low forceps to the after-coming head. The extraction was exceedingly difficult. The baby weighed 4115 grams. The placenta was retained 1 1/4 hours and during that time 1000 cubic centimeters of blood was lost. The blood pressure which had been 144/80 before delivery dropped to 74/40. To control the hemorrhage the placenta was removed manually. The blood pressure dropped to 50/20. 1000 cubic centimeters of saline was injected under the breasts and 25 minutes later the blood pressure was 82/50. An intravenous injection of 500 cubic centimeters of gum glucose was given and the blood pressure rose to 132/72. A blood transfusion of 500 cubic centimeters was given 48 hours later. Recovery was uneventful.

Farrar, Cannon, and Driscoll all emphasize the need of careful preparation of gum solutions. For the method used by Farrar the reader is referred to her article "Acidosis in Operative Surgery."

The injection is made with the salvarsan apparatus using small needles. The temperature of the solution on entering the vein should be 104-105 degrees Fahrenheit. The rate of injection should never be over 3 to 4 cubic centimeters per minute. Doses range from 200 to 400 cubic centimeters, depending on the size of the patient and should be repeated as often as deemed necessary.
The use of gum glucose in hemorrhage and shock following breech extraction.
Keith states that the injections must be early in shock for in late shock stages the capillaries may become so damaged that they are no longer capable of retaining fluids even though it be in a colloidal solution.

Randall reports 14 cases of women with shock and hemorrhage during and after delivery at the Mayo Clinic treated with an infusion of 5 percent gum acacia solution in .9 percent saline, illustrating the effect in the two following case reports.

Case 1. A pregnant woman, aged 38, who had born one viable child, was admitted to St. Mary's Hospital, June 1, 1928, at 11:30 A. M. Her last menstruation had occurred November 1, 1927. She had not had prenatal care. The previous evening, at 6 o'clock, she had been seized with severe abdominal pain, followed by moderate vaginal bleeding. Morphine was administered with some relief. At 3 A. M. June 1, there had been a sudden gush of blood from the vagina, succeeded by oozing. The patient had become weak and had fainted several times. Examination on admission revealed extreme pallor, and a pulse that beat at a rate of 120 each minute and was weak and thready. The skin was moist and cold. She was restless and irritable. There was very little vaginal bleeding. The abdomen was tender, and the uterus ligeuous. Fetal heart tones could not be heard. There were no definite uterine contractions. The cervix was well effaced and dilated to 4 centimeters in diameter. The blood pressure in millimeters of mercury was
30 systolic and 68 diastolic. The hemoglobin was estimated at 36 percent and the erythrocytes numbered 2,200,000 in each cubic millimeter. A diagnosis of premature separation of a normally implanted placenta was made. Seven hundred cubic centimeters of solution of acacia was given while the patient was being prepared for delivery. This caused an increase in blood pressure to 100 systolic and 70 diastolic, and an obvious improvement in the general condition. The pulse was still rapid but full and regular. The cervix was easily dilatable. The dilation was finished, and version and extraction were done. During the course of the operation, which was not hurried, blood became available for transfusion and 500 cubic centimeters of citrated blood was given. The placenta was removed and the uterus tightly packed with iodoform gauze. In this case, hemorrhage had to be stopped and shock combated. An attempt to deliver before increasing the volume of the blood and treating the shock might have been fatal to the patient. It is important to guard against further hemorrhage, so the uterus was packed. The next day the blood pressure remained above 100 systolic, and, aside from weakness, the patient felt well. The hemoglobin fell to 28 percent and the erythrocytes to 2,000,000. For the anemia, another transfusion of citrated blood was given. The subsequent convalescence was uneventful.

Case 2. A pregnant woman, aged 37, who had borne two viable children, entered the hospital with the head of the child resting on the perineum. Delivery was precipitate and took
place before sterile preparation could be made. Considerable
hemorrhage from the uterus followed delivery. The mother's
pulse rate rose to 130; she was pale, and the respirations
were shallow and rapid. There was slight cyanosis. The
blood pressure was 78 systolic and 16 diastolic. Immediately,
450 cubic centimeters of acacia was injected intravenously.
Following this the blood pressure rose to 108 systolic and
68 diastolic with slowing of the pulse rate and disappear-
of the evidences of air hunger. Examination showed the
placenta in the cervix, from which it was removed. The
uterus was tightly packed with iodoform gauze, and solutions
of pituitary and ergot were administered intramuscularly.
When the patient was returned to her room, one hour later,
the blood pressure was 138 systolic and 46 diastolic. The
pulse rate was 100 and the quality full. A hemoglobin
determination shortly after the patient was returned to
her room was 65 percent. The next day it was 62 percent,
and the erythrocytes numbered 3,290,000. This is an instance
of shock, probably due to sudden hemorrhage which was treated
with solution of acacia and no other intravenous injection
in which recovery was satisfactory. The usual postural
treatment was given with the application of external heat.
He also emphasizes the need of very careful preparation of
the solution and suggests, "as preventive of shock I feel
that it is often well to give an injection of a solution
of acacia in cases in which the patient is fatigued or
debilitated and in which obstetrical operations are to be
In addition to treating the blood pressure we should give morphine for acute pain and restlessness, provided there is no cyanosis present. The foot of the bed should be elevated. No operative procedures or repairs should be attempted as long as the blood pressure is below 100, and ether or chloroform should never be used. The body heat should be preserved with blankets, hot pads, etc., care being taken not to overheat. Circulatory stimulants as epinephrine, caffeine sodiobenzoate or strychnine are always to be considered and are often indicated. Other treatments may be used as the individual case may indicate.

Summary.

Having discussed the subject of obstetrical shock at some length, we may now come to a brief resume of that discussion summarizing it into the following points.

1. Obstetrical shock is a definite recognized entity in the practice of obstetrics and is very similar to traumatic or surgical shock in all its aspects.

2. The history of shock connected with pregnancy labor and delivery although only infrequently mentioned in the literature dates back to Biblical times.

3. Obstetrical shock occurs more frequently than is generally supposed, many cases being unrecognized.

4. Obstetrical shock falls into four general groups or types for classification:
   a. Shock due to excessive hemorrhage.
   b. Shock due to excessive trauma.
   c. Shock due to emotional and nervous strain.
and stress without hemorrhage.

d. Shock due to the combination of any of the above types.

5. The phenomena of shock includes a definite recognizable group of symptoms. These are:

a. A fall in blood pressure with a rapid thready or even imperceptible pulse.

b. Cold, moist, maybe cyanotic skin.

c. Respirations are shallow, quickened and may be irregular.

d. The patient is not unconscious yet is mentally dulled.

e. The eyes are closed and seem to be sunken into their sockets.

f. The features of the face are pinched and the face shrunken.

g. The causes, although at times but indefinitely understood, resolve themselves into a predisposing group which include the following:

a. Labor itself.

b. Fatigue, both mental and physical.

c. Fear, anxiety, and pain.

d. Deprivation of food and water.

e. Cold from exposure.

f. Long, difficult labors.

g. Infections and toxemias.

h. Emotions.

i. Sweating.
Anesthetic used, especially chloroform or ether.

k. Lowered alkaline reserve due to vomiting, excessive respiration, loss of body fluids.

And a group of active factors that precipitate the shock condition. When superimposed on the predisposing causes these include:

a. A sudden fall in blood pressure due to the sudden emptying of the uterus.

b. Sudden drop in intra-abdominal pressure.

c. Excessive hemorrhage.

d. Excessive trauma.

f. Long, difficult, or protracted labors.

g. Ruptured uterus, toxemia, eclampsia.

h. Collapse due to lack of fluid in actual circulation.

The treatment has been greatly experimented with and now has resolved itself into a definite group of procedures which are more or less universally followed at present. These may be summarized as follows (remembering that we are always dealing with individual cases, and the treatment should be so administered as best for the individual case being treated.)

a. Prevention, if possible by careful elimination of the predisposing causes.

b. Recognition if shock does occur.

c. Raise the blood pressure, by blood transfusion if immediately available; if not, then by intravenous injection of six percent gum acacia in twenty percent glucose
or in normal saline.

d. Morphine for pain and restlessness if no cyanosis is present.

e. Heat by blankets, pads, etc.

f. Dorsal position, no movements, surgery or repairs while the state of shock exists.

g. Force fluids, by mouth, rectum, and under the skin.

h. Stimulants and other treatment deemed advisable in the individual case.

In conclusion, we may say that obstetrical shock does exist and is similar to other types of shock, both in symptoms, causes, and treatment. It is important to understand the phenomena of shock, to be alert to its possibilities of occurring and to be able to recognize when it does occur, thus taking measure to prevent its occurrence. When it does occur be prepared to actively and energetically treat the patient in the proven procedures. The prevention, recognition, and early treatment will, in many cases, lead to a successful termination of shock and recovery of the patient that may otherwise have terminated by a fatal outcome. It is hoped that this paper will make it more easily possible to recognize and treat and thus prevent the fatal ending or some of these cases.
BIBLIOGRAPHY.


Audebert J. L. J.A.M.A. V. 84, Jan. 31, 1925, p. 404


Bayliss Intravenous Injection in Wound Shock London 1913 p. 24

Longmans, Green & Co. Reference from Cannon

Bible T. Sam. Ch. 4, verse 19, 20.

Cannon W. F. Traumatic Shock Appleton & Co. 1923


Crile G. W. Surgical Shock W. B. Saunders 1920 p. 17-18, p. 113, p. 134

Crowell Lancet V. 2, 1913, p. 138
Dale H. H. J. of Physiol V. 52, 1918 p. 110

Davis, Edward P. Obstetrics Lea Bro. & Co. 1904 p. 405


De Kruif Annals of Surgery V. 49, 1919, p. 311


Driscol W. P. Anesth. & Anesth. V. 7, March April 1929 p. 115


Erlanger, J. & Gasser H. S. Annals of Surgery V. 37, 1919 p. 395

Erlanger J. & Gasser H. S. Annals of Surgery V. 37, 1919 p. 420

Farrar, L. K. P. Am. J. Obst. & Gyn. V. 1, 1920, 92


Gessel A. J. Physiol. V. 47, 1919, p. 503
Grosse, A. J.A.M.A. Obst. V. 81, 1933, p. 511


Hofbour, J. Am. J. Obst. & Cyn. V. 12, Aug., 1930, p. 2


Katz, H. J.A.M.A. V. 78, 1932, p. 69

Mar. 1919 No. 27, p. 13 Reference from Cannon.

Kerr Maternal Mortality & Morbidity Woods Co. 1933 p. 52

King Am. Obst. Moore Wilstock Keys & Co. 1855 p. 270

Mann, Bernard & Israel. S. L. Med. J. & Record V. 138
July 17, 1933 p. 42

McCleod Physiology & Biochem. in Modern Med. C. V. Mosby Co.
5th Ed. 1926, p. 514, 519, 520.

McIlroy, Dame Louise & Beatrice Turner Lancet Jan. 11, 1930 p.97

Mack & Gasser Am. J. Physiol V. 45, 1917-18, p.549


Muret J.A.M.A. Obst. V. 80, 1923, p. 881


Phillips, Miles Brit. J. Obst. & Gyn. V. 21, 1912, p. 159


Pollard, C. W. Personal Class Notes


Rous & Wilson J.A.M.A. V. 70, 1918, p. 219

Schwarz, H. O. Am. J. Ob. & Gyn. V.6, 1923, p. 656

Stone, Emerson L. Am. J. Obst. & Gyn. V.II, 1926, p. 656-7

Taussig, F. I. Am. J. Obst. & Gyn. V.I, 1920, p. 95

Tweedy & Wrench Practical Obst.

Warren Surgical Pathology & Therapeutics Phila. 1895. p. 278


Williams, J. Whitridge Text Obst. 1st Ed. 1927, p. 973.