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*Obstetric Hemorrhage

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OBSTETRIC hemorrhage is any bleeding from the vagina definitely associated with pregnancy, parturition or the puerperium. It constitutes one of the three major causes of maternal mortality, ranking only after sepsis and toxemia. Apart from such conditions as benign and malignant neoplasms, which may complicate a pregnancy and so cause bleeding, obstetrical hemorrhage may be divided into the following groups:

I. Early pregnancy:
   (a) Abortion.
   (b) Ectopic or Extra-uterine pregnancy.
   (c) Hydatidiform mole.

II. Complicating Late Pregnancy and Labor:
   (a) Placenta Previa.
   (b) Abruptio Placentae.
   (c) Rupture of the Uterus.
   (d) Rupture of Varices.
   (e) Uterine neoplasms, both benign and malignant.
   (f) Premature Labor.

III. Hemorrhage complicating the 3rd. stage due to:
   (a) Faulty mechanism or faulty handling of 3rd. stage.
   (b) Uterine atony.
   (c) Miscellaneous causes—retentio placentae, placenta adhesiva, placenta accreta.

IV. Post Partum Hemorrhage:
   (a) Early:
      (1) Uterine atony.
      (2) Inversion of uterus.
      (3) Deep laceration of cervix or upper vagina involving a branch of the uterine artery.
   (b) Late:
      A. (1) Failure of uterus to remain contracted;
      (2) Retained secundines or placental fragments.
      B. Chorionepithelioma.

Since it will be impossible to consider all of the various causes of hemorrhage in a single presentation, this discussion will be limited to consideration of Placenta Previa and Abruptio Placenta.

Morbidity and Mortality. It is universally recognized that the more serious types of hemorrhage are important factors in causing morbidity and mortality and may act either directly or indirectly. Directly, hemorrhage causes an acute, subacute or chronic anemia with resultant tissue changes

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leading to disability or death; indirectly, by lowering the resistance of the patient to an infection which may cause either a protracted or a fatal illness. Innumerable statistical studies show the importance of hemorrhage as a direct cause of maternal death. The maternal mortality study recently carried out in New York City revealed that 17.8 per cent of all maternal deaths were due to hemorrhage, including deaths associated with ectopic pregnancy. The figures from the Glasgow Maternity Hospital show a mortality of 21.8% from hemorrhage (see Lancet, Feb. 8th., 1931).

A recent study and publication by the Canadian Welfare Council shows 12% of maternal deaths due to hemorrhage, excluding abortions.

Prenatal Care, while not a direct means of preventing these hemorrhages, will have a definite influence if, in her antenatal instruction the pregnant woman has been warned that vaginal bleeding, no matter how slight, may be a sign of one of the most serious obstetrical complications and one usually demanding immediate and definite treatment. The significance of this symptom should be recognized by the physician also.

Frequently the patient is forwarned by slight hemorrhage which, if not ignored, leads to a proper diagnosis of the underlying causative factor. Thus more serious hemorrhage is avoided by an early diagnosis and mortality lessened by timely treatment. Delay in diagnosis or neglect in promptly placing such patients in proper surroundings for delivery jeopardizes their lives. The importance of adequate ante-natal supervision cannot be too highly stressed. Many of the women who die from hemorrhage have failed to receive any or adequate antepartum care or proper preparation for delivery.

PLACENTA PREVIA

Definition. By placenta previa is meant the development of part or all of the placenta in the zone of dilatation of the uterus, the lower uterine segment.

In considering placenta previa it is necessary to make a clear-cut distinction between this condition and abruptio placenta, since these are the usual causes of serious hemorrhage late in pregnancy. Not only is there a difference in the location of the placenta, it being normally placed in abruptio, but the mechanism of production of the hemorrhage is different. In placenta previa the detachment is entirely mechanical, and the bleeding results from the separation of the placenta incidental to changes which take place in the lower uterine segment preparatory to, or during labor. The premature detachment of the normally implanted placenta has a more obscure etiology and, while it may be traumatic in a few instances it is, for the most part, associated with vascular or tissue changes which are not infrequently associated with the so-called toxemias of pregnancy. The differentiation of these two serious pathological abnormalities is important because it vitally affects the prognosis and the course of treatment to be instituted.

Munro Kerr reports that 4.4% of all maternal deaths are the result of placenta previa. The Glasgow Maternity Hospital gives a maternal mortality of 10.7% for placenta previa.

Incidence. The incidence of this obstetrical complication is difficult to determine, inasmuch as some writers include in their statistics cases which others reject. The frequency of placenta previa is somewhat higher than that of abruptio placenta. Statistics taken from various parts of the world indicate

that in some places it occurs once in 130 cases and in other regions once in 1500 cases. In general, the stated incidence varies from one case in 100 to one in 250. There is likewise, considerable variation in the relative incidence of the various types of placenta previa, though it is generally agreed the partial form is the most frequent.

Etiology. Placenta previa has an obscure etiology. It seems to depend upon neither age nor parity, and is found in the healthy young primigravida as well as in the scarred uterus of the elderly multipara. Multiparity, however, seems to predispose to its occurrence, especially rapidly succeeding pregnancies. The ratio of its occurrence in primigravidae and multigravidae varies from about one in ten to one in sixteen cases. The fact that the condition is somewhat more frequent in multiparous women is probably due, not to the parity itself, but to some condition arising as a complication of child-bearing, and having an effect upon the endometrium which in some way favours a low site of implantation.

The essential fact in the formation of a placenta previa is that implantation must occur in or near the lower uterine segment. The growth of the placenta carries it down to the internal os or its vicinity. Such implantation could be regarded as due to (1) an accidental localization of the ovum at the particular site, (2) some condition of the ovum favoring its deposition at a lower level than usual, or, (3) an abnormality of the decidua in the upper uterine segment which would not favor implantation at this site.

It seems possible that the actual site of implantation may not always be predetermined and that in a certain small percentage of cases the ovum might sink to and lodge in the lower uterine segment.

Placenta previa is said to occur in patients with a previous history of endometritis, and it has been suggested that the increased size of the uterine cavity in this condition allows the ovum to drop into the lower segment of the uterus. In a study of one hundred unselected cases of placenta previa in our clinic, a history of menstrual irregularities, of benign uterine bleeding, post-abortal or post-partum infection, or of other signs and symptoms on which a diagnosis of endometritis could be made were lacking. This condition, therefore, though invariably quoted as an etiological factor, in our experience, bears little or no relationship to the causation of placenta previa.

Webster in 1903, suggested that the low implantation of the ovum may be due to its fertilization rather later than is usual, i.e. after it reaches the lower part of the uterus. Since, however, embryologists now agree that fertilization always takes place in the Fallopian tube, this theory no longer obtains.

The gravitation theory was generally accepted until 1888, when Hofmeier advanced the theory that the placenta is developed in whole or in part, in connection with the decidua capsularis. As the ovum develops sufficiently to fill the uterine cavity, decidua capsularis and decidua vera fuse, and vascular connections are established to the uterine wall over the lower segment. This theory was supported by Koltenbach in 1890, and Jolly in 1911 advanced excellent proof of its occurrence in many cases.

Strassmann has advanced the idea that, in consequence of defective vascularization of the decidua, resulting from inflammatory or atrophic changes, which in turn were caused by repeated and closely following pregnancies, a larger surface area of placental attachment would be favored in

order to obtain the necessary nutriment. A large and thin placenta may thus be formed, which approaches the os interum or may even cover it.

Since the foregoing theories do not explain all cases of placenta previa, and since the same etiological factor is probably not responsible for all such cases, it is suggested that placenta previa might be the result of one of the following circumstances:

(1) Failure of the factor which governs the cyclical and pregravid changes in the uterine mucosa to properly prepare the fundal endometrium for the reception of the ovum, with the result that it is deposited at a lower level:

(2) The development of the morula might be delayed so that it could not attach itself to the fundal endometrium: and

(3) It might be entirely accidental and perhaps the result of the assumption of the upright position by the primates.

Placenta previa may occur more than once in the same patient, but there is no marked tendency for it to do so.

Anatomy and Pathology. The typical placenta is one with a central attachment of the cord and in which growth has extended equally in all directions, thus forming a circular organ. It is difficult to state which factors determine the direction of growth of the placenta, but probably it is largely a matter of vascular supply and nutrition. In the case of the previa placenta, the placenta is usually larger and thinner than the normal. This is due to the fact that the uterine decidua gets thinner as it nears the internal os and, the fact that the glycogen content, essential for the growth of the fertilized ovum, is lower in the mucosal cells, the chorionic villi finding less tissue in which to find nutriment, the placenta spreads over a larger area to compensate for this.

It is obvious that the original site of implantation must be over the os interum, or near enough to it so that the growth of the chorionic villi will bring the placenta close to or over the internal os. It is possible that the site of implantation can be close enough to the internal os that, with the growth of the placenta, a previa must result.

Certain morphologic changes are known to occur in the lower uterine segment and cervix during the latter part of pregnancy and the first stage of labor. Before labor, the lower segment is hemispherical in shape, and during labor it is stretched and converted into a cylinder. (Each part, therefore, undergoes stretching, increasing in degree as it lies near the os internum.) When the placenta lies in the retracting and gradually effacing lower segment the more or less rigid placenta does not participate in these changes of the lower segment. Consequently some separation takes place at the lowermost portion of the placental site, decidual attachments are torn and blood sinuses are opened. The result is bleeding which varies in amount, depending upon the extent of the vascular injury. The blood comes from the uterine wall, i.e. from the placental sinuses and, to a very slight extent from the placental surface. In some cases it may be due to the laceration of the so-called circular sinus (Meckel) of the placenta. The blood escapes through the cervix and there is little or no tendency for the hemorrhage to be concealed.

The placenta may be abnormally large or irregular in shape and the part near the os is often the thinnest. Placental infarcts are rather common in the previa placenta. Microscopic examination reveals no characteristic

changes. Old blood clot is commonly found at the detached margin. The membranes are torn at or near the periphery of the placenta.

The frequency of abnormal presentations is greatly above the average. Fetal monsters are said to be not uncommonly associated with placenta previa. Prolapse of the cord is more frequent. The fetuses are apt to be premature and are frequently born dead as the result of suffocation or birth trauma incidental to methods of delivery used in the treatment of placenta previa, but there are no characteristic changes, except such as might occur from an acute anemia or suffocation.

The Types of Placenta Previa. There has been more or less confusion in distinguishing between various types of placenta previa. This confusion arises not only from difficulties in differentiating these conditions from one another but also from certain confusion in the terminology. In recognizing the different types of placenta previa one may consider two main groups:

(1) Placenta previa totalis or complete placenta previa. Here the os is completely covered at the time the case is diagnosed. The os may be completely covered when the cervix is only dilated 2 or 3 centimeters and incompletely covered when the dilatation becomes greater.

(2) Placenta previa partialis or incomplete placenta previa. In this group three main sub-divisions may be included:

(a) Placenta previa marginalis, when the placenta is felt at the edge of the cervix at the first vaginal examination.

(b) Placenta previa lateralis, when the lowest margin of the placenta is felt above the edge of the cervix at the first vaginal examination.

(c) Low implantation of the placenta may be diagnosed when the signs and symptoms strongly suggest placenta previa, and the edge of the placenta is palpated high in the lower uterine segment previous to parturition. Examination of the placenta and membranes subsequent to delivery will confirm indefinite cases.

Symptoms and Clinical Course. The clinical course of a case of placenta previa is usually uneventful until hemorrhage occurs. The trite statement that painless, causeless bleeding occurring in the last trimester of pregnancy is almost pathognomonic of placenta previa is a valid one. The more centrally located the placenta, the earlier, the more severe and serious, the hemorrhage. It is the liability of the patient to severe hemorrhage that makes placenta previa so dangerous.

Placenta previa usually affects the course of pregnancy, labor, and the puerperium. The period of gestation, at which the first hemorrhage occurs, varies considerably, though 30 to 36 weeks seems to be the most frequent. The occurrence of the characteristic type of bleeding in the latter half of pregnancy should excite suspicion, double precautions, and lead to taking steps for the establishment of the final diagnosis. This warning bleeding occurs in most cases, is usually not profuse, and rarely results fatally. It is due to the partial separation of the placenta, thereby laying open bleeding sinuses. Sometimes there seems to be an exciting cause, such as some effort, or shock, or local violence; but this is exceptional. It generally ceases spontaneously. The hemorrhage is, essentially, a hemorrhage of repetitions, occurring at irregular intervals, and becoming more copious as term approached. When

occurring for the first time during the first stage of labor, bleeding is generally excessive, because large vessels not previously thrombosed are laid open as the placenta becomes separated.

As a rule, there are no symptoms except those arising from blood loss. There is rarely any concealed blood loss except from blood clots retained in the vagina. The symptoms vary with the amount of blood lost and the rapidity with which the hemorrhage occurs. A severe anemia developing gradually is better tolerated than one which develops rapidly. The pulse rate is increased, the blood pressure falls, restlessness and air-hunger are noted, there is marked pallor as a rule, the skin is clammy and may even appear semi-transparent. The superficial veins are collapsed and bloodless. Usually there is no abdominal pain or tenderness and no uterine contractions unless labor is induced.

The general course of labor is unfavorably affected by placenta previa in several ways. Premature labor is the rule. Not more than a third of the cases approach term, and only slightly over one-half attain the period of fetal viability. Malpositions and malpresentations are more frequent because the capacity of the lower uterine segment is diminished. Transverse presentations are unusually common being about fifty per cent more frequent than in labors generally. As a rule, the pains are feeble and irregular. It is not uncommon, when the cervix is approaching complete dilatation for the pains to become strong and rapidly terminate the labor. On the other hand, often when labor has begun with fairly good pains, when the patient becomes exhausted from hemorrhage, they get weaker and cease. Since interference is frequently required to arrest the bleeding or to rapidly terminate labor as quickly as possible, there is increased risk of lacerations of the cervix and lower uterine segment. Air-embolism is mentioned as a danger, but it is extremely rare.

The most common mode of death with placenta previa is from post partum hemorrhage. Nearly fifty per cent of the cases of placenta previa reported by the Children's Bureau had post partum hemorrhages. The poor retractile power of the lower uterine segment, the inefficient contraction and retraction of the uterus in an exhausted patient, and abnormal adhesions interfering with the process of separation of the placenta constitute the chief reasons for the post partum hemorrhage.

In the puerperium, puerperal sepsis is not an uncommon sequel. This is accounted for partly by the frequency of operative interference and serious cervical lacerations, and partly by lowering of the patient's resistance to infective processes, due to blood loss. The anemic condition of the patient after delivery also renders her more liable to phlegmnsia albadolens, pulmonary embolism and insanity.

The life of the fetus is also jeopardized by the premature separation of the placenta, which may cause fetal asphyxia and death by prolapse or compression of the cord, or by the interference required to stop hemorrhage or terminate the labor.

Diagnosis. A diagnosis of placenta previa may be surmised from a history of painless, prefuse hemorrhage occurring, without obvious cause, but the final determination rests upon the ability to feel the placenta. The type of placenta previa should be diagnosed promptly without aggravating the hemorrhage and should be made as soon as possible after the onset of the warning hemorrhage. It is vitally important that no pelvic examination be made

until proper measures have been taken to control the hemorrhage and treat the patient in case the examination should provoke further hemorrhage. A safe rule would be to examine only under anaesthesia after all preparations have been made to deal with the condition—and in hospital if at all possible.

The findings on examination apart from those made out per vaginam are not conclusive. There are no certain abdominal signs. Placenta previa should be suspected if the size of the uterus corresponds to the calculated gestation, if the fetus is easily palpable, if the presenting part is floating and displaced to one or other side of the false pelvis, if something is preventing its decent through the pelvic brim; if the uterine bruit is best heard over the lower uterine segment and if the fetal heart tones are distinct. *Rectal examinations should not be done as they rarely provide the necessary information, and may increase the hemorrhage, or renew bleeding which has temporarily stopped.*

The only conclusive method of diagnosing a placenta previa is to feel placental tissue through the cervical canal. The finger encounters a spongy, fibrous mass either completely covering the internal os or lying to one side of it. If such is felt the diagnosis of placenta previa is established, providing the very rare instances of prolapse of the normally implanted placenta is ruled out. If the cervical canal is closed, which is unusual, placenta previa is probable, if there is a boggy mass in one or both lateral fornices, separating the examining finger and the presenting part, making internal ballottement impossible. The cervix and lower uterine segment are softer than normal, and the uterine arteries, pulsating more strongly than usual, may be felt. Examination per speculum will detect blood coming from the cervical os, and exclude the possibility of polypi, neoplasms of the cervix, and bleeding from the bowel and urinary tract.

The differential diagnosis of the warning hemorrhage must be made from the following: cervical polypi, erosion of the cervix, neoplasms of the cervix, ruptured varicose veins of the vagina and vulva, and abruptio placenta. The chief condition requiring differentiation is abruptio placenta, and it may be difficult especially if there is partial separation of a low lying placenta. The hemorrhage from placenta previa is generally fresh, consisting of liquid and clotted blood, and it may come in gushes, especially during uterine contractions. In abruptio placenta the bleeding is apt to be continuous or more marked between uterine contractions, and consists of fresh blood, blood stained serum and old clots. Pain, tenderness of the uterus especially in the region of the placenta, and evidence of toxemia are frequently present in abruptio placenta, but, apart from toxemia, which may complicate the pregnancy, are absent in placenta previa. *On abdominal examination* the uterus is not contracted in placenta previa, whereas in abruptio it is apt to be tense and tender with the uterus frequently larger than the estimated period of gestation due to retained blood. In placenta previa the fetus is usually alive and distinctly palpable with a floating or malposed presenting part. In abruptio (placenta) the fetus is frequently dead and is palpable with difficulty. Vaginal examination reveals placental tissue intervening between the examining fingers and presenting part in placenta previa, which finding is absent in cases of abruptio, except in the very rare complication of abruptio where there is prolapse of the normally implanted and completely separated placenta.

Prognosis. The prognosis is always serious, but under ideal conditions the maternal mortality should be very low. A review of the more recent

statistics shows a maternal mortality ranging from five to nine per cent and a fetal mortality of thirty-five to sixty per cent for all cases. Caesarean section performed early and in good hands shows a much lower mortality for both mother and child.

The prognosis depends upon several factors, chief of which are,—early recognition, prompt hospitalization whenever possible, preparedness and careful management. It is more serious to both mother and fetus in the central than in any of the other varieties. The duration of pregnancy, the number of pregnancies, the presentation of the fetus, whether labor has started or not, the amount of blood lost and its effect, and whether or not it has been replaced, careful antisepsis, are all factors which influence the prognosis. The major dangers are from hemorrhage both ante-partum and post-partum; from trauma due to too rapid and energetic manipulations during delivery, resulting in injury to the fragile placental site, cervix and lower uterine segment; from infection due to lowered resistance; from the proximity of the placental site to septic areas, and from the neglect of proper technic in the hasty measures necessary in the treatment. Severe infections, if they do not result in death, may leave a trail of subsequent ill-health to the mother.

The prognosis for the fetus is very grave, especially where the mother dies. It depends on the fetal maturity, the amount of blood lost, the degree of asphyxiation, and the amount of birth trauma. The major factors affecting the outcome for the fetus are first, its degree of maturity, and second, the method of delivery. Caesarean section will save many babies doomed to certain death by delivery through the natural passages.

Treatment. Prenatal care should emphasize to the patient the importance of any vaginal bleeding following conception. A careful blood study should be made and if an anemia is found, proper treatment should be instituted.

Expectant treatment is rarely indicated and then only under hospital conditions where prompt emergency measures are always at hand. The patient should always be hospitalized without examination unless the initial bleeding is of such amount as to be alarming. It is rarely necessary to treat bleeding occurring late in pregnancy, in the home. When a patient enters our clinic a definite routine is followed by the resident staff. The patient is typed and a suitable doner found, usually a member of the family. If this is not the case, a compatible group four donor should be available. All preparations are then made for the treatment of the various complications which might arise on examination of the patient. These preparations should include facilities for immediate laparotomy, in the event that this is the treatment of choice. The patient is then examined vaginally. Rectal examinations should not be performed because they do not afford the necessary information, and may dislodge clots and environmental relationships in the uterus to such an extent that a fresh hemorrhage may be provoked. During the vaginal examination an attempt is made to diagnose the condition. If placental tissue is palpable in the lower uterine segment or covering part or all of the os, the extent of the placenta previa can be determined. The utmost aseptic precautions are necessary in the examination of the patients who are bleeding. They are more susceptible to infection than normal patients.

The treatment of placenta previa is individualized according to the conditions in the patient. If we find that the edge of the placenta is palpable high in the lower uterine segment we conclude that we are dealing with a low

insertion of the placenta. This type of placental insertion occasionally causes considerable bleeding, but its management is not difficult. Usually, simple rupture of the membranes is all that is necessary. After the escape of the amniotic fluid the presenting part may descend into the pelvis and compress the placenta satisfactorily, thereby controlling the bleeding. After this the patient usually goes into labor. The labor itself may be entirely uneventful.

Occasionally the edge of the placenta is palpable at the margin of the external os. This condition is diagnosed as a marginal placenta previa. Here again, simple rupture of the membranes may be all that is necessary to control the hemorrhage. At times this is not sufficient and in spite of the ruptured membranes the bleeding continues. The insertion of a colpeurynter or bag usually of the Voohees type, is indicated. The rubber bag should be large enough to control the hemorrhage, and on passing it there should be sufficient dilation of the cervix to allow for immediate delivery, if necessary. It has been our experience that a bag 10 or 11 cm. in diameter is desirable in the treatment of a placenta previa. A weight ½ to 1 pound can be applied to the bag with traction arranged over the end of the bed or with the aid of a pulley. This is usually sufficient to keep the bag closely applied to the lower uterine segment and the placenta.

If a part of the os is covered by placenta, then we are dealing with a partial placenta previa. These cases may bleed considerably, and the patient may be fairly exsanguinated on her entrance to the hospital. If the patient is in good condition, if she has lost little blood, if the cervix is soft and effaced and easily admits the examining finger, and if there is no contraindication to delivery through the natural passages, then again simple rupture of the membranes with the introduction of a bag will suffice to control the bleeding and to initiate labor. On the other hand, if a good portion of the os is covered by placenta, or the patient has lost considerable blood, or other borderline indications for abdominal delivery exist, a caesarean section may prove to be the treatment of choice.

If the entire os is covered by placenta, we are dealing with a total placenta previa. This is the most serious type. Immediate caesarean section offers the best prognosis for the mother and is still the simplest treatment for this serious condition. From the foregoing it may be seen that in the individual treatment of placenta previa followed in our clinic it is necessary to examine the patient carefully. Occasionally this vaginal examination provokes a sudden alarming hemorrhage and a temporary vaginal pack may have to be used to control it long enough to complete the caesarean section. The suddenness of the hemorrhage at times makes it necessary to have all preparations for laparotomy and the treatment of complications in readiness.

If it is desired to deliver the patient from below, she should be carefully observed during the entire labor. As soon as the bag has passed through the cervix it should be removed so that no blood is allowed to accumulate behind it. The delivery can be allowed to take place spontaneously if there is no alarming bleeding, or it can be completed rapidly by version and extraction or forceps, depending on the existing conditions. Suffice it to say that operative manipulations should be carried out with the utmost gentleness in order to minimize the trauma which may be extensive in cases of placenta previa.

The treatment of the third stage likewise demands considerable attention. Serious post partum hemorrhage can result. If the placenta does not separate quickly and completely, it should be removed manually. If there is bleeding

following the removal of the placenta, one should immediately inspect the lower uterine segment for lacerations, and if none is found the entire uterus and vagina should be carefully tamponaded. Pituitrin is useful in controlling the hemorrhage.

Blood transfusion probably has been the most important factor in the improvement of the treatment of obstetric hemorrhage. A liberal blood transfusion should precede any type of treatment in the patient who has lost considerable blood. Following delivery a patient can be transfused a second time if the delivery has resulted in considerable blood loss. Ringer's solution subcutaneously and 10 per cent glucose solution intravenously can be given where blood is not immediately available. These fluids are valuable but do not take the place of blood in the patient who has been exsanguinated as a result of her condition.

Braxton Hick's version should be used only in dead or non-viable babies where the other methods of treatment are not available. After rupture of the membranes a leg is brought through the cervix, thereby allowing the buttocks to act as a tampon against the placenta. Technically, it is a difficult procedure and should be reserved for emergencies. A simpler method for such a case in an emergency is to grasp the fetal scalp with a Willett forceps or a very heavy tenaculum forceps. Gentle traction can then be made so as to control the bleeding. The method should be feasible for treatment of a patient when hospital facilities are not available. Vaginal packs are of little use and materially increase the hazard of infection.

Nothing has been said about the baby in the management of placenta previa. It is true that many women begin to bleed before viability of the fetus. Treatment, however, should be instituted when the initial hemorrhage occurs, and thus many pre-viable babies must necessarily be sacrificed. It is rarely justifiable to delay the termination of pregnancy in a case of placenta previa in order to give the baby a better opportunity. If a patient is near the borderline of viability, if the baby is exceedingly valuable, as might be the case in an elderly primipara, and it is desirable to wait, then the patient should remain in the hospital under the most careful, continuous observation. One must always remember that the continuous presence of blood in the vagina alters the bacterial flora and may thus give rise to a pathogenic flora. Subsequent delivery may result in a puerperal infection of serious import. Caesarean section offers the best prognosis for premature babies.

Third Stage in Placenta Previa. The treatment of the third stage demands careful supervision, as it is often atypical and may be associated with hemorrhage. Additional hemorrhage at this time, even though small in amount, may prove fatal for the patient. If there is little or no bleeding, one may safely await spontaneous separation of the placenta before it is expressed. If there is active bleeding, Crede expression or manual removal should be promptly employed, with the strictest aseptic precautions.

Post-partum hemorrhage is of comparatively common occurrence in cases of placenta previa. It arises from laceration of the cervix, from the placental site, or from atony of the uterus. Pituitrin, 1 c.c. intramuscularly, and (or) 5 minims intravenously, may be life saving measures if given early. Ergotocin in 0.2 mgm. doses, intravenously or intramuscularly, gives more sustained action and severe reactions have not been experienced, which happens occasionally with pituitrin. If there is bleeding after the removal of the placenta, the genital canal should be carefully examined for injury, making

sure there is no rupture of the uterus. If no rupture is found, and pituitrin fails to arrest the hemorrhage, the uterus and vagina should be tightly packed using about twelve yards of gauze. Rupture of the uterus is best treated by immediate hysterectomy if the patient is in good condition and the rupture is in the upper portion of the uterus. But, where the patient is exsanguinated, or where the tear is confined to the lower uterine segment, and not through the peritoneum, uterine tamponade should be used.

Temporary clamping of the uterine arteries from below, by means of Henkel's clamps, or temporary ligation of the arteries by sutures, are methods which have been tried to control hemorrhage. It is reported to be relatively safe, but the danger of incorporating one or both ureters in the clamp or of making perforation through the vagina into the peritoneal cavity and incorporating bowel, are too real for this method of treatment to be commonly used. Compression of the aorta with one's hand, or an aortic compressor, will control any profuse hemorrhage and will give the physician time to collect his wits and make suitable preparations for the control of further hemorrhage.

Early recognition of placenta previa, hospitalization, preparedness in controlling and treating hemorrhage, careful selection and deliberate carrying out of appropriate measures for delivery, guarding against and controlling third stage and post-partum bleeding, are the best methods of minimizing the mortality of these patients. The principal causes are hemorrhage, trauma, shock, and sepsis, and all of these are controllable if not entirely preventable.

ABRUPTO PLACENTAE

Abruptio placenta was suggested by DeLee to designate that serious obstetrical complication in which the normally situated placenta is partially or wholly separated from the uterine wall in the last weeks of pregnancy and in the course of labor. Ablatio placenta, accidental hemorrhage, concealed hemorrhage, partially concealed hemorrhage are also names which have been applied to this condition.

Most authors separate the cases of abruptio placenta into the mild and grave types depending on the patient's condition. Because it is sometimes dfficult and occasionally impossible to differentiate between partial and complete placental separation the patient's symptoms and findings must be made use of to help in the differentiation.

Mortality. Maternal mortality varies in different clinics. Statistics from the Maryland Medical School give a maternal mortality of ten per cent with a fetal mortality of 57 per cent. Broadhead gives a maternal mortality of a little over 26 per cent and a fetal mortality of over eighty-five per cent. This condition is thus more serious for both mother and fetus than placenta previa.

Incidence. Abruptio placenta is a common cause of ante-, intra- and post-partum bleeding. The frequency has been variously estimated at from one in 94 to one in 890 labors. Holmes claims that many cases are missed as the bleeding may be slight and the examination of the placenta casual or careless. He places the incidence in one in 200 labors. No doubt many mild or partial separations occur undiagnosed with little or no damage to mother or child, but also probably accounts for a fair proportion of intra-partum fetal deaths of undetermined cause. Partial separation occurs with greater fre-

quency than the total. The incidence of concealed and visable hemorrhage varies a great deal. In a series reported by Broadhead external hemorrhage was present in 67 per cent of the cases.

Predisposing factors. It is generally believed that multiparity is a predisposing cause. In a series from Chicago-Lying-In Hospital, 35.8 per cent of all the cases occurred in primipara which agrees with Williams, Burgess and others. Thus we must conclude that parity plays little part in the causation of this condition.

Etiology. The etiology of abruptio placenta is unknown. Various theories have been suggested but aside from those cases in which mechanical factors play a role and the groups associated with the so called toxemias of pregnancy, there is very little definitely known about the factors producing the hemorrhage associated with placental detachment. Various figures are quoted which range from an incidence of 33 per cent to one of 82 per cent for association with toxic conditions. It seems to be less frequently associated with eclampsia than it is with the vascular and nephritic conditions. In our series the association with eclampsia was less than one per cent. In the same series some evidence of a toxic state was found in over 56 per cent of these cases during their prenatal period. On the basis of this and other findings two types of abruptio may be recognized—the toxic associated with hypertension and/or albuminuria, odema, etc., and the non-toxic. There seems to be no essential difference in the type of hemorrhage in the two conditions.

Trauma appears to be a factor in about 2 per cent of the cases. This factor consists of abnormal uterine contractions, a relatively short cord, and direct trauma from external version or other force applied to the abdomen. The improper use of pituitrin for induction of labor or during labor has been mentioned as a very pertinent factor.

Endometrial disease is probably a more common cause of abortion and placenta previa than it is of premature separation, though there can be no doubt that it figures in the etiology of abruptio. Diseases of the myometrium, inflammation of the placenta, infarction, no doubt also play a role in the causation of this condition.

Pathology. The pathological process begins with a hemorrhage into the decidua basialis. Whether this initial bleeding comes from the placenta itself or from the small blood vessels as a result of some changes in their walls is a debatable question. The bleeding causes a decidual splitting and the development of a retroplacental hematoma or likewise a hematoma in the placenta itself in some cases. The extent of the process may become limited and we have a partial separation or it may continue to extend until the entire placenta has been separated off the uterine wall. The retroplacental bleeding may be limited to the uterine cavity by the placenta, the membranes, or tightly engaged presenting part and will be entirely concealed. It may rupture through the membranes and into the amniotic cavity and still be concealed, or it may extend to the margin of the placenta, dissect the membranes away and appear externally. On delivery of the child, there may be a gush of dark and clotted blood. The placenta will show some area of depression on its maternal surface and to it there may be attached some disorganized blood clots. The extent of these findings will indicate the extent of the separation.

The uterus in some cases shows very few changes, but in the type known as utero-placental apoplexy, first described by Couvelarie, definite changes

occur in the uterine wall and the structures immediately surrounding it. Grossly the uterus, tubes, and ovaries, and often the broad ligaments present a bluish mottled appearance. The uterine wall is the site of a hemorrhagic extravasation of varying extent in severity, which however, tends to be worse in the neighborhood of the placental site and in the superficial rather than in the deeper layer. Sometimes dark free blood is found in the peritoneal cavity having oozed through the fimbriated ends of the tubes or through tears in the serosal surfaces of the uterus and adenexa. The organ is flabby and stretched, having lost the power of contractility. The cut surface is wet and presents a bluish mottled appearance most marked in the outer and inner layers of the uterine wall.

Microscopic examination chiefly reveals hemorrhage between the fibers and muscle bundles. These hemorrhagic areas seem to be more numerous towards the serosal and mucosal surfaces. The cytoplasm of the muscle fibers shows odema and evidence of degeneration. The endothelial cells of the vessels are swollen and appear to be undergoing degeneration.

Clinical Course. The clinical course varies greatly in different cases and depends particularly upon the amount of placental separation and whether the bleeding is mainly concealed or visable. The mild type or the partial separation of the placenta usually occurs near the end of labor. The patient may have had a normal course, when suddenly the pains become much harder, last longer, and the interval between pains becomes much shorter. There may be some pain or sensitiveness over a localized area of the uterus and a small showing of blood from the vagina. On auscultation of the fetal heart one finds evidences of fetal asphyxia demonstrated by an irregularity in rhythm or a decrease in rate. The fetal heart tones may entirely disappear if the infant cannot be rapidly rescued. A sudden change in the character of the labor, hemorrhage, evidences of fetal distress, and the finding of old blood clots in the uterus and partially attached to the placenta make the diagnosis of a partial abruptio placenta.

Complete abruptio placenta, or the grave case presents an entirely different clinical picture. It most often occurs before the onset of labor, in fact, any time in the last trimester of pregnancy.

The onset of the abdominal pain may be dramatic, with sudden excruciating pain in the lower abdomen. It is usually continuous and the uterus often assumes a board-like hardness and becomes very tender and painful. A careful outlining of the uterus may reveal a gradual increase in size due to an accumulation of blood within it. There may be some visible hemorrhage or the escape of bloody serum, which is usually indicative of clots retained in the uterus. Completely concealed hemorrhage is rare, but in many cases the visible blood loss is much less in amount than the concealed. The amount of external bleeding is no index of the amount of blood the circulation has lost. In addition to pain and distress and visible bleeding there are symptoms of an acute anemia and of shock. A point in the history which is of considerable value is that the fetus may have been excessively active for a brief time, after which it became quiet and no further movements were felt. The patient may grow rapidly worse and die within a relatively short time unless prompt treatment is instituted.

Diagnosis. The diagnosis of partial abruptio placenta can be made by the sudden change in the character of the labor, the external hemorrhage,

and the signs of fetal asphyxia as is indicated by the slowing of the fetal heart rate. Uterine tenderness is often present and may be due to the underlying pathology. Uterine contractions may be present and persist, but the uterine wall does not relax well between pains. After the delivery of the baby there may be a passage of dark liquid and clotted blood. The placenta may have some dark antepartum clots attached to one portion of it, and there may be a depression of the maternal surface of the placenta where they have been attached.

The complete separation can be diagnosed by the occurrence of external hemorrhage in the last trimester of pregnancy associated with pain of moderate or extreme severity. There may be present a mild or severe toxemia. Abdominal examination will reveal a sensitive, ligneous uterus with no relaxation of note. The progressive picture of hemorrhage rapidly develops. The blood pressure is a better index of the true condition than is the degree of anemia apparent superficially or on blood count and a rapidly falling blood pressure is a serious sign. Shock may often be out of proportion to the hemorrhage. The gradual increase in the size of the uterus and a change in its contour may mean concealed hemorrhage. The fetal parts are palpable with difficulty and the fetal heart tones are absent.

On vaginal examination there are no characteristic findings, except the appearance of blood from the cervical canal. The presence of blood-stained serum instead of whole blood, is quite characteristic. The placenta cannot be palpated in the lower uterine segment. The presenting part is usually palpable with ease, and displacing it may allow the escape of blood and clots.

The differential diagnosis of bleeding occurring in the later months of pregnancy must be differentiated from, (1) placenta previa, (2) uterine rupture, (3) premature labor, (4) acute polyhydramnion. In placenta previa the onset is usually painless and causeless, and on pelvic examination the classical signs of previa are found. Rupture of the uterus usually occurs later in labor, and there are other signs of protracted labor and disproportion, or there may be a history of previous caesarean section, or myomectomy. Premature labor may be attended with slight vaginal bleeding but has none of the diagnostic signs of abruptio and other serious lesions. In some instances an acute polyhydramnion has simulated abruptio placenta due to the distention and tenderness of the uterus. External bleeding is usually not present in this condition. Rupture of the membranes reveals the excess liquor amnii. The baby is usually alive. Normal uterine motility rapidly terminates the labor.

Prognosis. The outcome for the mother depends largely on the promptness with which the condition is recognized and treatment instituted. According to available statistics the maternal fatalities range from 2.6 to 66%. The fetal mortality varies from 60 to 95%. In our series the maternal mortality was 7.3% and the fetal mortality was 60%. Thus, this condition can be said to be one of the most serious complications of pregnancy.

Treatment. As in the case of placenta previa the treatment should be individualized and will depend upon the conditions met with in the patient. The general indications are to empty the uterus with the least danger to the mother, the control of hemorrhage, combating shock, and restriction of the blood loss.

The partial separations, or the mild cases, most often occur in labor. These cases can be treated conservatively and labor allowed to terminate as nearly as spontaneously as possible. The patient's condition must be carefully watched noting the amount of bleeding, the possibility of concealed hemorrhage, the character of the labor pains, the blood pressure, and the state of the fetus. If the membranes are intact they should be ruptured to make internal bleeding less likely, to facilitate the labor, and to permit a closer adaptation of the uterus about the baby, thereby decreasing the possibility of hemorrhage. As soon as the labor can be terminated with safety to the mother and to the baby this should be done by the most conservative means. Immediately on the birth of the baby, one cubic centimeter of pituitrin should be given. The third stage should be terminated promptly by manual removal of the placenta if hemorrhage continues following the second stage. The danger of continued post-partum hemorrhage in these cases is very real, and the patient should be carefully protected from it during the third and immediate post-partum stage.

In the complete separations of abruptio, if the patient is in labor, and making normal progress, conservative measures may suffice, but in the grave cases more energetic methods are necessary. In many instances it will be possible to terminate the labor from below. The baby is most often dead and needs no consideration. As soon as there is sufficient dilatation the labor may be expeditiously terminated by forceps, or a craniotomy, depending on the existing conditions. Small doses of pituitrin intramuscularly may hasten the labor and lessen the bleeding.

If utero-placental apoplexy is suspected in a toxemic patient, caesarean section is frequently indicated irrespective of the fetal condition, because these cases frequently present a hemorrhagic tendency which is difficult to control without performing a hysterectomy, because the uterine musculature may have lost its ability to contract. Whenever there is any doubt as to this ability on the part of the uterus to take hold of the situation it had better be sacrificed.

Timely treatment is most important in this grave condition. Patients in shock must be rapidly prepared before radical surgery is attempted. The use of liberal blood transfusions, 500 to 1000 c.c. or more before, and after, operation if necessary, is the most important single measure in the treatment of serious cases. In the absence of available donors, acacia solutions may be substituted, glucose and saline intravenously and subcutaneously, and morphine will help to bring the patient out of shock and improve her operative risk.

The third stage is the most serious in the entire labor. Even a slight post-partum hemorrhage may be sufficient to cause a fatal termination. Everything should be in readiness for manual removal of the placenta and uterine tamponade. If there is an unusual amount of bleeding following the delivery of the baby and Crede's expression unsuccessful, it should be removed manually. The uterine cavity should be thoroughly explored and all clots removed. If the uterus has a tendency to remain flabby, or if the bleeding continues after massage and pituitrin intramuscularly, uterine tamponade should be immediately instituted. The proper packing of the uterine cavity is very important, because it is extremely dangerous when the packing acts as a plug allowing the bleeding to go on behind it. Six to twelve yards of gauze are used to firmly tampon the uterine cavity, the cervix and

the vagina. If firm packing does not control the bleeding, it may mean an error in diagnosis has been made and that the uterine musculature is probably disintegrated beyond recovery. Under these conditions a large blood transfusion, followed by a rapid hysterectomy may still save the patient.

In conclusion—general treatment to combat the anemia and shock should be instituted early in every case. Blood combats the anemia and shock at the same time. Every pregnant woman who enters the hospital bleeding should be typed and matched and a proper donor or donors kept available until the patient is out of danger. Quiet, unalarming, and methodical procedures, and a kindly, sympathetic attitude towards the patient herself will do much to secure the much needed cooperation in these cases.

*Psychiatric Clinic

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I MUST start off by telling you somewhat apologetically that I am giving you a rather poor example of what one should do when confronted with the type of patients I am going to present this morning. Ordinarily, under usual circumstances, I should have spent some time in preparation to be capable of giving as clearly as possible the facts of the situation and to have at least a few basic historical and biographical data and a precise knowledge of the development and onset of the patients' difficulties. However, in the short time at my disposal, it was quite impossible to do just that: first, because of the condition in which the patient themselves were at the time of the brief interview; secondly, because family members were not available to supplement histories and keep us from getting a one-sided picture from the patients, one of whom, as you will see, is not in a position to give adequate information because of her state of confusion.

Our first case is that of a woman about whom Dr. Atlee will be kind enough to say a few words of introduction.

Dr. Atlee: "This patient came in some time ago with a vaginal discharge and a good deal of pain in her left side. The pain kept on, and disappeared, and she came back again, and I opened her abdomen and I found the left ovary tied by adhesions and cysts, and I removed it. Following that she has had very definite smothering attacks; she thought on two occasions she was going to die, she had to be moved to the window before she felt at ease. In addition to that she is the type of a woman who says she is not feeling good at all, and who always has some new symptom pointing somewhere else."

The patient was brought in and was questioned by Dr. Kanner as follows:

"Good morning, Mrs. W. how are you this morning?"

"I feel a little better this morning."

"Have you had any more difficulties since I saw you yesterday? Here are a few doctors who want to know something about you, about the kind of attacks you used to have."

"I used to be very short of breath; it seemed as though I couldn't catch my breath."

"How did it feel?"

"Felt awful distressed like and like something was smothering me."

"What did you think would happen before the attack was over? When did those attacks start?"

"I thought I was going to die. They started right after the operation, lasted two or three days."

"When you had the operation, you had it under an anaesthetic; were you afraid to go under?"

"No."

*Conducted during the Dalhousie Refresher Course at the Victoria General Hospital, Halifax, N. S. September 3rd, 1937.

"Mrs. W. had her first operation because of pain in her side which began three years ago. At that time she had a great deal of difficulty at home, her husband was out of work, there was economic stress and she worried about the health of her older child. Then more drastic things began to happen in her family. Mrs. W. lost both her father and mother two years ago, and that affected her quite a bit and gave her the idea of dying, and of course the pain in her side added to that. Then about a year ago she lost her brother through drowning. Now she realizes, and said so herself yesterday, that her shortness of breath is not due to any physical origin, but feels it was 'nervousness'. She feels more reassured now and that things are getting better. Thank you for coming in here."

Patient removed.

This phenomenon, in a case which I admit has not been worked up by me so thoroughly as I should have liked, is very interesting and not so unusual. These so-called anxiety attacks, with shortness of breath, palpitations, all sorts of emotional disturbances and utter distress, especially fear of dying, may occur at any age. I have seen them in children as young as five years of age and I have seen them in old people. They have a background of serious domestic difficulties, problems connected with unemployment or vocational misplacement and especially, as in this case, a great deal of preoccupation with health, death (you find that several deaths have occurred in quick succession in Mrs. W. family), and worry about having inherited some disease. Anxiety attacks are especially apt to be precipitated after an operation under an anaesthetic. The patients often go under the anaesthetic after thinking for several days: "Perhaps I shall never get out from under and I might die on the operating table". After the operation is over, the anxiety attacks may come on as late as several weeks afterwards.

In Mrs. W. case there is a history of difficulties. About three years ago there was financial strain at home, then followed an operation which in turn gave rise to a need for a second surgical procedure, then illness of a child, then three deaths in the family and worry about her oldest boy, twenty-one years old, who is said to have something wrong with his back. After her first operation she had anxiety attacks for three days only. In the present situation the patient requires a great deal of reassurance. She needs an explanation of what the operation really was about and competent assertion that the results warrant a good prognostic outlook and that therefore the health prospects are good. At the same time it would be a good thing to see that her boy is given a thorough examination and if there is anything wrong he can be looked after so that she can be reassured on that score. She has another child who has been diagnosed as having bad tonsils; arrangements for their removal should be made if this is necessary. With all that, with improved economic situation in the family (her husband now works regularly), with the assurance that she has had quite a bit of sympathy from her relatives and a feeling that she will continue to get it, I think she will do very nicely, especially if the nature of the attacks is explained to her clearly and convincingly. It is very difficult for a patient to accept things unless you help her to work them out for herself. It is best to have the patient tell you things. You ask and direct your questions in such a manner that the patient comes to see things in their proper relations. You help the patient to work out her own problems, to see them in different connections than she had thought of them before. This has been done yesterday in the case of Mrs. W.

It is not only a physical condition, "nervousness", that caused her heart to palpitate, but the attacks were a reaction to a situation, and she has learned that. It seems to me she has worked that out herself with our questions as a sort of guide.

The next patient is a woman of forty, who came here primarily with the complaint of a vaginal discharge.

(The history was given by the interne, Mr. Stewart.)

Before presenting this patient I might say a word or two that has no particular bearing on this case, something that might sound to you revolutionary or radical. You noticed that in presenting the patient you just saw I did not have her in the room when her history was read or when the diagnosis, differential diagnosis and prognosis were discussed. In our teaching hospitals it is very difficult to consider the patient when you have a group of students around a ward bed. Students ask questions and under those circumstances the patients, who are certainly not inanimate, begin to worry and have difficulty comprehending what it is all about. All sorts of Latin terms are used, and there are many tragicomic instances when the patients carry some of these remarks home with them and quote or misquote them to their friends. I have seen a great many psychiatric difficulties either develop or become aggravated through case presentations when the patients are present and dire prognostic possibilities are mentioned. The patient hears names of various diseases and leaves with considerable preoccupation and apprehension. Demonstration before a group is a major event in the patient's life, to the teacher and students it is a matter of routine; it means something to be brought in before so many doctors. Before the patient goes out it is always best to make a few reassuring remarks.

Here the patient, Mrs. S., was brought in.

"Will you have a chair? Here is a group of doctors who are interested in just what is bothering you as they try to learn as much as they can about these things. They see quite a number of patients that come to them with the same difficulty and they want to learn about your complaints. Would you tell them just what is the main thing that bothers you?"

"I have a pressure over the left side in front."

"It goes back to the back of her head, it is a feeling which she has described as being a queer feeling, but one which Mrs. S. who is an intelligent person is not capable of defining."

"I do everything but I have a cut-off feeling."

"Her reason tells her that things are the same and she understands that, but things seem changed as though she were looking through glasses, she just can't get at them, she can't enjoy them. What are your general spirits, happy, unhappy, sad?"

"Mostly sad."

"But that is not her usual disposition. She told me yesterday that she is in a different condition; she usually is a jolly, sociable person who likes to be with other people, just the right kind of a good sport. She lives in Sydney where her husband is making a satisfactory living, and always has; no outstanding financial difficulty; no children, and she and her husband get along well. Eight years ago she had a similar condition which then was even more definitely associated with sadness, and at that time she cried more than at present and she had more pain than now and a feeling as though she couldn't go on. Now she does not have that feeling so much. It is just a milder degree

of sadness because the other time she had it she screamed 'in pure agony'. Now, Mrs. S. has said something that is very wise. She said that after all the much severer condition eight years ago took its course; after two years it was over and she returned to her usual mode of living. This time where the condition is not so severe she hopes that things will get better more quickly, especially as she has come to the hospital and her vaginal discharge will be taken care of with medical care. It is possible, but she is not quite sure of it. Would you be a little more certain if you knew that people here and myself have definitely the knowledge that you are going to get out of this worry just as soon and better than you got out of the other?"

"Of course, people are not going to tell you the contrary".

"You see I have a reputation to take care of, and if I came all the way from Baltimore to tell you a falsehood in the presence of all those doctors I think it would be bad for my reputation, but you see I am quite sure of that, Mrs. S. Thank you very much. Good-bye, Mrs. S."

You see there is quite a difference between Mrs. S. and Mrs. W. Mrs. W. also suffered in her anxiety attacks and there also were worry, fear and preoccupation with her health, but you have the impression that you deal with two altogether different reactions. In the first case you have a woman who tells you of the spells that she has had and feels she is getting better and has no reason to see why she should not. In this case you see that my appeals and reassurance did not carry so much weight and she showed some scepticism. Mrs. S. is an intelligent person and knows that this is not her usual condition. She is downcast; she is worried; she is depressed; her own statements are very telling. She is sad; has been that way since May. She told me yesterday that she herself had looked for possible reasons for that condition, but she cannot say it is worry. There is a history of jealousy on the part of her husband, and that has played a part, but in her normal state she has been able to manage that very satisfactorily. When normal, she is jolly, sociable, and goes about her domestic affairs efficiently and interestedly. Then came her depression. These conditions are described as manic-depressive psychosis. The patient is sad, suffers agonies, often has a feeling of unworthiness, of sinfulness, very often with suicidal tendencies. It is very interesting that both at this time and during her first episode Mrs. S. had what has been termed as unreality feelings; she felt as if things were far away, as if they were there but somehow you have no contact with them; things look different and changed.

Depressions are of various origin and may be associated with cerebral conditions or schizophrenic reactions. Here you have a woman who has preserved her intelligence; she can converse intelligently about current topics but all the while everything is coloured by her sad mood and a fear of losing her mind. Of course, she is in a condition now where she is mentally affected, but this she does not quite recognize. She fears she might lose her mind. There is headache, with that dull queer feeling that she has. She is in a depression and is affected mentally. The way she is at present, only mildly affected, you would not think perhaps of sending her to a mental hospital, though I have an idea that her illness is going to last a few more months. There is something that must always be looked for even during the period of convalescence. You are never certain that there are not going to be strong suicidal urges, and that is something to caution the nurses about in order to have intelligent watching of the patient. The persistent, intelligent watching of the patient in more severe cases becomes quite a problem in hospital

wards. There are all sorts of medication suggested for depressed persons that have not had the effect that was hoped for, but great help can be expected from the administration of sedatives in patients who are agitated and suffer a great deal from sleeplessness. The next thing to do, especially in a mild case of this sort, is to try slowly to reassure the patient again and again, but not through argument. Dose your reassurance as you dose medication; give a little at a time without making the patient feel that you are just doing it half-heartedly and that you do not quite believe yourself in what you say. Occupational therapy, properly dosed, is another great help in the treatment of depressed individuals.

Mrs. S's depression was not brought about by any particular worries as Mrs. W's attacks were. There is a strong constitutional element in the etiology. Unfortunately, we have not enough data about her family history; there is no known history of "insanity". "Insanity" has come to be a legal term much more than a medical term. We know that there are people whose personalities are more sweepingly affected than others. It would be a good thing if we could dispose of the word insanity and the arguments of physicians in court as to whether a person is or is not "insane". This woman, if it were not for her vaginal discharge and severe headache, might have gone through life with mild depressions and mood changes without the need of hospitalization.

Dr. K. A. MacKenzie: "A woman with a mild psychosis, should we commit her?"

Dr. Kanner: "That all depends. If we were to commit all the mild psychoses, all people with psychotic manifestations, all moderately retarded individuals, then we should immediately fill every vacancy in our institutions, even if we had twice or three times as many vacancies. This is a practical question. It is a question first of the facilities of our communities, which are not equipped even to take adequate care of all those psychotic people who are definitely and desperately in need of hospitalization. Ideally, I should say that those patients who do not need commitment should have a period of treatment and observation in a general or private psychiatric hospital. If Mrs. S's depression becomes more outspoken, if the suicidal risk becomes greater, she should be sent to an institution. If you send her to a hospital she will get there the best care and treatment. Crowded conditions in our psychiatric institutions have offered quite a problem everywhere. There is fortunately a growing demand for adequate psychiatric care.

Most communities realize now that being in a mental hospital is not a disgrace, but that these places offer possibilities for the same kind of work and research that is done in other medical centres. However, we have gone through a period of economic depression in which facilities were not increased and were often decreased, and now the demand has grown for mental hospitals, for training schools for the retarded and also reform schools and other institutions. In practically every State and Province there are numerous people on the waiting lists for admission to institutions and reform schools. Many needy children and adults are kept out of hospitals because of lack of space. This is a really important and, I think, a tragic state of affairs. So, under ideal circumstances, Dr. MacKenzie, the best thing for this patient would be placement and treatment in a hospital until we are reasonably sure she has recovered from her present condition. If the patient is mildly depressed

and her husband is appraised of suicidal dangers, if she can get help from her family physician and perhaps a private nurse when she returns home, it might be possible to steer her through the depression as you will be called upon to steer many patients through mild depressions, who are not sufficiently ill to be sent to hospitals.

Dr. MacKenzie: "The difficulty I have in mind is quite a number of suicides in the last few years who were not sent to hospitals, and I wondered if they should not have been."

Dr. Kanner: "In every case the suicidal risk is the first thing to be thought of."

I now have a case of incipient paresis, a thirty-seven year old woman. Dr. Morton called my attention to this case yesterday. The mother, who is getting old and somewhat senile, brought Jennie to the chest clinic because of mild respiratory difficulties. The mother says her mind is completely gone; she won't keep still, always doing something, cutting her stockings. Jennie has always been very seriously retarded. The mother says she has "got her wore out" all the time: "To-day might be months back, that is the way she talks". She reached the fourth grade in school at the age of thirteen, then she went to work. Twelve years ago she gave birth to a child, about whose father very little is known. Jennie told me that she believes he was drowned. The boy is now twelve years old and is in the fifth grade at school. The patient is the oldest of nine children, of whom two are dead: The mother flattered me by thinking I was a native and therefore she could not quite understand how it came that I did not know anything about her son, who is apparently a well-known prize-fighter here. The general intelligence level of the family is not high, but Jennie seems especially affected. Her pupils react normally, are round and equal; knee jerks are present; Romberg is strongly positive; test phrases cannot be pronounced at all. She is disoriented as to time: she thought it might be July or December. Blood Wassermann is positive.

The patient was brought in.

Dr. K.: "Have you ever seen so many doctors in one room? Would you tell these doctors what seems to be the trouble?"

Patient: "A nervous breakdown."

Dr. K.: "How long have you had that?"

Patient: "Since before Christmas."

Dr. K.: "How does it show?"

Patient: "On the nerves, I cannot stop my hands and my throat does the very same thing."

Dr. K.: "How old are you?"

Patient: "Thirty-seven years."

Dr. K.: "Single or married?"

Patient: "Married".

Dr. K.: "What church do you belong to?"

Patient: "St. Paul's".

Then the patient was asked to pronounce "Methodist Episcopal", which she could not do.

Dr. K.: "You came here with your mother to-day. Is your mother treating you all right? Is she sometimes a little harsh with you?"

Patient: "Well, sometimes."

Dr. K.: "What happened a little while ago?"

Patient: "I washed my feet in mustard for the bottoms of my feet. I cannot think."

Dr. K.: "Do you know where you are now?"

Patient: "In a big place."

Dr. K.: "What kind of place is this?"

Patient: "Doctors come here."

Dr. K.: "Where do doctors work, and in what kind of a building?"

Patient: "In the V.G." (Victoria General Hospital).

Dr. K.: "Were you ever here before?"

Patient: "I was working here."

Dr. K.: "You were working in other places also."

Patient: "I worked at the Green Lantern for eight years and the V.G. for six years."

Dr. K.: "What kind of work did you do?"

Patient: "I was in the diet kitchen, in the elevator and in the operating room."

Dr. K.: "What did you do in the diet kitchen?"

Patient: "I waited on the customers."

Dr. K.: "What did you do in the operating room? You didn't operate, did you?"

Patient: "No, I cleaned it."

Dr. K.: "Would you like to go to a hospital and be treated for your nervous breakdown?"

Patient: "Yes."

Exit patient.

This patient does not need very much discussion. You have a case of general paresis, and the obvious thing to do is to have her sent to a hospital where in this early stage she could be treated with malaria or other fever therapy.

(Jennie was committed a few hours later to the Nova Scotia Provincial Hospital.)

*Nephritis and Kidney Function

C. B. WELD, M.A., M.D.

The Normal Kidney—The purpose of the kidney in so far as the body is concerned is to excrete waste and foreign materials, to excrete excess water and inorganic salts and to maintain the normal acid-base balance of the body. These three activities are of course closely related. Professor Bean has already described to us the embryological development and the structure of the kidney. The kidney may be said to be made up of a number of functioning units, each being termed a "Nephron". The nephron consists of a glomerulus and its associated tubules. Let us consider the nephron and briefly indicate its mode of action.

The Glomerulus, with its capillary tuft forms an admirable filter and serves to act as such. Some of the blood plasma is filtered through the glomerular tuft: the filtrate is a protein free plasma. The force which effects the filtration is the blood pressure within the glomerular capillaries, and it is of interest to note that the pressure in these capillaries is exceptionally high. It is said to be about 75-80 mm. Hg. and probably ranges between 30 and 90 mm. Hg. or thereabout according to the degree of vasoconstriction and dilatation of the afferent and efferent vessels.

The tubules, considering the proximal and distal convoluted and Henle's loop as a single whole, transform the simple plasma filtrate which has been formed by the glomerulus, into urine. This is done by absorbing back into the blood all of the glucose, most of the water and inorganic salts and practically none of the waste products such as urea and creatinine. The resultant urine is a relatively concentrated solution and it is the concentrating of the urine that entails the work or the energy production on the part of the kidney. A diminution in the functioning power of the kidney is for this reason apt to be first shown in its impaired ability to concentrate urine. It is important to realize that the blood supply to the tubules is from the glomeruli; the blood which has passed through the glomerular filter and has lost some of its fluid is passed into a second capillary system where most of the fluid is restored. This second capillary system surrounds the tubules. Furthermore, obstruction to the blood flow in the glomeruli will deprive the tubules of their blood supply. The question as to whether the tubules, in addition to selectively reabsorbing certain constituents of the glomerular filtrate back into the blood, can also excrete certain materials into the urine, has been subject to much investigation and controversy. It would seem that some latent power of secretion may have to be granted but that it is of no significance in the normal kidney. Except for certain foreign substances such as some dyes and perhaps for traces of creatinine the function of the renal tubules is purely one of reabsorption.

As we excrete about 1½ lr. urine per day (twenty-four hours) and as it is calculated that in order to obtain this urine some 90 lr. filtrate must be formed by the glomeruli, can we reasonably expect the kidneys to accomplish this? There are at least two million nephrons, and if each glomerulus were to filter

*Given as a part of a Symposium on Nephritis during the Dalhousie Refresher Course, Halifax, N. S., August 30th, 1937.

just one drop of fluid in the day this would give us about 100 lr. filtrate. The total area of the glomerular capillaries is estimated at about 1½ sq. M. and the total length of the tubules about 100 miles. The blood flow through the kidneys totals some 1500 lr. in the day. On the basis of these figures the load on the kidneys doesn't seem so unreasonable.

The pressure relationships of the kidney are important, because it must be realized that normal renal functioning depends greatly on factors external to the kidney. For example, should the *arterial pressure* be low, as in cardiac failure or shock, there will be little filtration and hence oliguria with a concentrated urine results. Similarly if there is back pressure in the renal tubules as is found in ureteral or urethral obstruction, the high *tubule pressure* impedes filtration and oliguria again results. A high *venous pressure*, as in venous congestion due to cardiac failure partially occludes the tubules and so raises the tubule pressure.

So far we have been speaking of the perfect kidney. We are however, dealing with living membranes, and relatively minor changes in nutrition, oxygenation or hormone control may appreciably alter their degree of permeability. A slight increase in the permeability of the glomerular capillaries, will allow a trace of plasma protein to leak through. *Albuminuria* is thus simply produced, and its presence need not be a sign of renal disease, particularly after exercise. Albuminuria in a bed patient or a resting individual is to be regarded with more concern than in the active subject. The term proteinuria is in reality more correct than albuminura (and it is being used with increasing frequency in the literature) because both albumin and globulin are present: albumin is the more abundant. Some of the protein may become inspissated in the tubules and give rise to *casts*, which become granular when mixed with cellular debris and cellular when plated with blood or epithelial cells. The presence of the casts indicates proteinuria and their significance depends therefore on their number and on the type of cell associated with them: an occasional cast may be found in normal urine. The presence of *blood* in the urine, if it is derived from the glomeruli, denotes a much greater degree of glomerular capillary permeability than albumin and may be generally considered to denote some disease process. However, an occasional erythrocyte may be found in normal urine. Even when albuminuria, casts and blood are marks of renal disease, they convey no information as to the amount of kidney tissue involved.

The tubles too may show some deviation in their behavior from that considered perfectly typical. They may leak and allow a certain amount of waste products such as urea to leak back into the blood. Probably this is of slight concern to the normal, but it may perhaps explain the unexpectedly great retention of nitrogenous substances and the high blood N.P.N. in cases involving obstruction of ureter or urethra, such as prostatic obstruction.

Nephritis—By this term we are thinking of true nephritis, diffuse glomerulo-nephritis, an inflammatory process involving the kidneys. Professor Smith will discuss the nomenclature of the various renal diseases and describe the kidneys of the glomerulo-nephritic type.

In the early or *acute stage* of the disease there is damage to the capillaries of the body, resulting in some edema. The glomerular capillaries are particularly affected, and renal inflammation results. If the damage is mild there is a changed permeability while if severe there may be disruption of a part or the whole of the glomerulus. Circulation through the whole glomerulus may be

occluded, in which case the corresponding tubules degenerate and ultimately disappear. Due to the inflammatory reaction the renal tissue becomes swollen and distends the capsule. The rise in intrarenal pressure interferes with tubule drainage and impedes the renal circulation, thus still further embarrassing the kidney. Decompression operations (decapsulation etc.) have been done but are quite impractical. In this stage the kidney is in an unstable, rapidly changing condition. The urine findings are oliguria with blood, protein and casts. The treatment is designed to rest the kidneys.

In the later, more *Chronic Phases*, the inflammation is receding, those nephrons which were only partly damaged are recovering function, the patient is better, and the condition is more stable. Blood, protein, and casts in the urine are diminishing and indeed may be absent in the chronic stage though they are not likely to entirely disappear as long as there is any inflammation still present. The question as to whether or not there is much residual damage to the kidney now becomes important, and in view of the minimal urinary findings, not readily answered. There is no single quantitative test of renal function which is wholly satisfactory, but there are several simple means of investigation which indicate impairment of renal reserve. They are at least more precise than clinical observation and give objective information, forming valuable records of the case and acting as useful guides in its management. They will be discussed under the heading of "Renal Function Tests". It is to be remembered that the polyuria, perhaps manifested only by nocturia, of many of these patients is a sign of diminished renal function. As the kidneys cannot concentrate the urine efficiently, the excretion of waste products is limited by the volume of the urine. The excretion of some of the waste products which would normally take place during the day, is delayed and occurs during the night. This enhances the volume of the night urine.

The treatment of these cases will be discussed in detail by Dr. Carney, but the object is to rest the kidney. In this regard it must be remembered that the condition is a protracted one and that if less than a good maintenance diet is given the body will consume its own substance to make up the deficiency. Thus the poor diet defeats its own end for not only will the waste materials have to be excreted anyway but the dangers of malnutrition such as increased liability to infection will also be incurred. Edema when met with is due to a low level of plasma protein consequent to loss of protein in the urine. A high protein diet is the logical treatment. The diuretics (including digitalis) and the use of a low salt diet which may be so useful in edema of cardiac origin are of little value in renal edema in the chronic nephritic.

Renal Function Tests—No attempt is to be made here to describe all the tests which have been proposed, but a few of the more useful are mentioned and described in a somewhat simplified form. Further details if desired are readily obtainable from any standard text.

1. *The Concentration Test* (Addis). No fluids are given between 8 p.m. and 9 a.m. The patient voids at 8 a.m. and 9 a.m. The specific gravity of the last specimen should be more than 1.026. It will usually be about 1.030.

2. *The Dilution Test* (Volhard). After emptying the bladder 1.5 lr. water is taken (within 20 minutes) on a fasting stomach. Urine is obtained every half hour for 4 hours. The total volume excreted in the 4 hours should be 1.5

lrs. or more and the specific gravity of some of the specimens should drop to 1.002 or lower.

3. *Two Hour Test* (Mosenthal). During a day in which three normal meals are eaten, urine is collected at 2 hour intervals from 8 a.m. to 8 p.m. and the subsequent 12 hour night specimen is obtained. One specimen at least should have a specific gravity of 1.020 or more, there should be a range in gravities of at least 0.010, and the volume of the night specimen should be less than 725 c.c.

4. *The Urea Concentration Test* (MacLean). Fifteen grams of urea (in 5 ounces water) are given by mouth, after emptying the bladder. Urine is obtained in one and two hours. The urea concentration of the two specimens should be greater than 1.5% and 2% respectively.

5. *The Dye Test.* The dye (phenolsulphonephthalein) administered should appear in the urine within 10 minutes, be 50% excreted in 1 hour and 70% in 2 hours.

6. Determination of *Blood N.P.N.* (non-protein nitrogen). The normal range is 25-40 mg.%.

7. *Urea Clearance Test* (Van Slyke). During the morning two consecutive one hour specimens of urine are obtained and a sample of blood taken. The urea content of the three samples is determined and the volume of blood which has been cleared of urea is calculated. The standard figure is 54 c.c. per minute and normals will give a value at least 70% of this.

Of these tests, from the point of view of the practitioner, the first (concentration test) is the best and it is the easiest to apply. The dry regimen results in a concentration of the urine, the degree of which is reflected by the specific gravity. The measurement of the specific gravity in these tests must be done with care, and the degree of albuminuria should at the same time be noted. The presence of 1% of protein in the urine will raise the specific gravity by 0.003. Hence if much protein is present a correction can be made on this basis while if only a small amount is present the effect of the protein is negligible. A patient recovering from an acute nephritis is not well as long as the concentrating power of the kidney is poor, and when the test has become normal one may be assured that no excessive damage has been done. The second and third tests convey the same general type of information but are somewhat less sensitive. Furthermore the second is useless if edema is present and the third requires a more prolonged cooperation on the part of the patient. The fourth (Urea Concentration) stands with the first in value but is not as simple to carry out. Furthermore changes in the rate of absorption from the intestinal tract would affect the result though it seldom does interfere. It is perhaps less affected by edema than the first. The dye test is not useful in nehpritis but is of value in some of the surgical affections of the urinary tract.

In chronic nephritis the blood "Non Protein Nitrogen" (sixth test) may be normal even with badly damaged kidneys: a rise is a late, almost terminal, event. However in pregnancy the test is a more valuable sign as N.P.N. retention may here occur before great permanent renal damage is done. Also in surgical cases such as prostatic obstruction the N.P.N. is higher than the renal damage warrants, and the test is a useful guide to treatment.

The urea clearance test is the most sensitive and quantitative test for renal function that has yet been developed. However it demands careful

laboratory control and is therefore a hospital procedure. Furthermore it does not in the ordinary case show diminished renal function any more clearly than the concentration test. It frequently does however show a recovery of function weeks or even months before the concentration test shows improvement and is therefore often a valuable favorable prognostic sign. If the clearance test has not become normal within four months of the acute phase of the nephritis, the prognosis is poor.

Summary—Some of the features and physiological properties of the normal kidney have been briefly reviewed and some of the effects of nephritis upon renal activity have been indicated.

The more useful renal function tests have been outlined and the application of the simple concentration tests for assessing renal damage has been recommended for more general use.

*Venereal Diseases from a Public Health and Social Standpoint

DR. J. K. MCLEOD

TUBERCULOSIS and cancer are generally looked upon as two of the most serious health problems the profession has to deal with. I think, probably venereal disease exceeds even these in importance for it is so widespread, so difficult to control and difficult to cure.

Owing to the nature of the disease and the manner in which patients are infected the disease is hidden from the family and from the public. I think it is generally conceded that venereal disease with its complications and serious sequellae is the greatest killing disease of which we have any knowledge. No organ of the body is exempt from the infection and the brain and spinal cord in so many instances become infected that patients end either in the asylum or as permanent paralytics. In years gone by houses of ill fame and what are commonly known as street-walkers were counted largely as the cause of these diseases. But now the disease spreads largely through those who are morally and physically impure and who infect innocent victims. Maids in our families, entrusted with the care of children, are often the cause of spreading the disease to innocent children.

In the City of Sydney the Government, as elsewhere, established a clinic about ten years ago and there has passed through it in the neighborhood of a thousand cases, the large majority of whom are not of the low type we sometimes expect these cases to be, several being under ten years of age. These young cases of course, are congenital and were not known to be infected until blood tests were made. The cases attending the clinic are cases unable to pay for treatment. Besides these, doctors treat many cases of which we never hear. It is said that 10 percent of the population in large cities is infected. This will give some idea of the prevalence of the disease and the seriousness of it from a public health and social standpoint.

Under the circumstances what should be done in this connection? The governments of almost every civilized country have established venereal, free clinics which have done a great deal of good, not only in the treatment of the disease, but also as educational centres. Many cases come for the treatment now which formerly received none and it is rare indeed, at the present day, to see the open cases of syphilis so frequent in general hospitals, forty or fifty years ago. But further steps should be taken towards the control of this disease by a follow-up service, similar to that in tuberculosis, where every case is known and every case under treatment. In addition, those who deliberately infect others should receive such severe punishment as such conduct deserves. Education in homes, schools, church and through welfare associations and the press should be carried on until the public are thoroughly informed of the dangers of these vile diseases and the method by which they are spread.

*Read before Annual Meeting Provincial Association of Medical Health Officers, July 6, 1937.

What is Acidosis?

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THERE is a lack of clarity in our conception of the state of acidosis. As generally used the term covers several different physiological relationships which are encountered frequently in clinical medicine. The etiology of these various conditions is different although the symptoms may be the same. Acidosis is usually associated with the hyperpnoea known as Kussmaul breathing with increased rate and depth of respiration and absence of cyanosis. In other words it is the picture associated with diabetic coma. A wide-spread misconception exists however in the minds of the public and of some members of the medical profession that diet contributes acid to the human body such that "acid" occurs in the blood manifesting itself mainly in the form of cutaneous lesions. This conception is in part due to advertisements appearing in the lay press and on the medical side to the existence of many allergic reactions due to food which at present are little understood.

The purpose of the present article is to present as clearly as possible the various conditions which give rise to acidosis and the different types that are known.

The term, *acidosis*, is not sufficiently exact in its meaning of the condition of metabolism in which abnormally large quantities of acid are present in the body. The reason for this will be apparent later. In the first place it must be realized that it is an important normal function of all tissues to produce acid in the form of carbonic acid in the daily processes of oxidation of the food. Such processes account for the energy of muscular action and for the heat of the body. A simple calculation based on the consumption of the average diet consisting of 100 grams of protein, 100 grams of fat and 400 grams of carbohydrate gives the daily amount of 1116 grams of carbon dioxide. This is equivalent to 2.5 pounds of CO_2 and the amazing figure of 567 litres in 24 hours (or approximately 400 c.c. per minute at N.T.P.). These figures are based on the assumption of the complete digestion and utilization of the diet. This is however a large amount of acid for the blood to transport and for the lungs to excrete. Every cell of the body is adding its quota of carbon dioxide from the oxidation of the foodstuffs. Of these the carbohydrate is giving rise to most because of its preponderance in our diet but the fats are also normally completely broken down to carbon dioxide and water. The proteins constitute the most complex materials of the diet and in oxidation give rise to many products. Urea, carbon dioxide and water are quantitatively the most important. Proteins do however give rise to other acids of much greater strength than carbonic acid for there are also formed small amounts of the mineral acids, sulphuric and phosphoric, from the sulphur and phosphorus of the native proteins. Whereas the carbon dioxide is excreted by way of the lungs, these acids, in the form of their salts, are excreted by the kidneys. They comprise only about one three hundredth the amount of the carbonic acid formed.

It must be apparent from these facts that our bodies are well fitted to control large amounts of acids. The production of acid is a normal function of all tissues and it is obvious that the ingestion of a few grams of an "acid" fruit or vegetable presents no tax or strain on the general mechanism unless there be a pathological condition of the respiratory, circulatory or excretory systems. An inefficient heart, an oedematous lung or a nephritic kidney immediately presents a physiological problem in the disposal of the large daily quota of acids.

Many years ago the great French physiologist, Claude Bernard, stressed the importance of what he called the internal environment. Here surely is a good example of the importance of internal environment in the control of the acidity of the tissues and especially of the medium of transport, the blood. It is one of the surprising facts in physiology that the reaction of the blood is so constant despite the transportation of so much acid. Normal life cannot be sustained unless the reaction of the tissue fluids is kept practically constant and such a condition must be slightly basic. This is commonly spoken of as controlled by the acid-base equilibrium and any deviation from this equilibrium constitutes the condition of acidosis on the one hand and alkalosis on the other. The reaction of the blood is maintained within the remarkably narrow limits of 7.30 and 7.50 measured in terms of pH. As a matter of experience the variation is much less than this and can be measured conveniently in blood before and after passing through the lungs with consequent loss of carbon dioxide. Human arterial blood is usually 7.35 and venous blood 7.33. The extreme limits of pathological variation on record are 6.95 on the acid side and 7.80 on the basic side accepting 7.0 as neutrality. *It is thus safe to state that the blood practically never becomes acid in normal or pathological conditions.* What then is acidosis?

To make an answer to this question possible it is essential to understand the mechanism of the transport by the blood of the large quantities of CO_2 formed daily. This is related to the acid-base equilibrium of blood and tissues which constitutes the "alkali reserve" of the body. Several substances are present in blood and tissues which resist the action of acids or bases to change them in respect to the reaction of their solutions and which are called buffers in consequence. These are the bicarbonates, phosphates and the alkali salts of the proteins, including haemoglobin and oxyhaemoglobin. Each exists as a team of two members, one reacting to acids and the other to bases. Thus

$$(1)\ \frac{Na_2HPO_4}{NaH_2PO} \qquad (2)\ \frac{\text{Na-Protein}}{\text{H-Protein}} \qquad (3)\ \frac{NaHCO_3}{H_2CO_3}$$

in these pairs as acid accumulates there is a shift of the compound in the numerator to the compound of the denominator with a very small shift in the pH of the blood. In the first two there will also be an accumulation of bicarbonate as more carbon dioxide is forced into the circulation. This then is what is meant by the carbon dioxide combining power of the blood and which has also been expressed as the "alkali reserve". It is base held in union with weakly acidic compounds in the blood which *can* be released when the accumulating acid demands it. Van Slyke has calculated that if all the CO of the blood were present as carbonic acid (H_2CO_3) the blood would be about a thousand times more acid than it is. If on the other hand the CO_2 were all present as bicarbonate, the blood would be several hundred times more alkaline. It is evident then that we have two forms of CO_2, the free and the

combined. The former will be present in solution in the blood as such or as carbonic acid. The latter will be present as bicarbonate and the ratio of H_2CO_3/ $NaHCO_3$ will indicate the acid-base equilibrium of the individual. This ratio is normally 1 to 20. If there is an abnormal production of acid or a loss of some of the base by excretion the ratio will increase and 1/16 or greater represents a condition of acidosis. Conversely if the production of acid decreases and base accumulates the ratio will decrease and one of 1/23 or less is considered an alkalosis. We thus see that the condition of acidosis or alkalosis is a relative one dependent upon the amounts of carbonic acid and bicarbonate. At this point we may try to define more exactly the condition of acidosis. *The term acidosis is generally used to designate an abnormal condition caused by the accumulation in the body of acids or the loss from the body of base.* In subacute conditions when the deficit of base is small and the hydrogen ion concentration (pH) of the blood is not altered sufficiently to be measured, the condition is called *compensated acidosis.* If the condition is sufficiently acute to induce a measurable change in the pH of the blood towards the acid side, the term *uncompensated acidosis* is applied. The latter condition is sometimes referred to by English writers as *acidemia.*

Van Slyke (1921) has defined acidosis as a condition in which the base in the blood available for combination with bicarbonate is diminished. Graham and Morris (1933) state that anything which increases the ratio of free to combined carbon dioxide will result in an acidosis, and conversely anything which diminishes the ratio of free to combined carbon dioxide will result in an alkalosis. The difficulty of defining exactly what is meant by the term acidosis will now be apparent.

We have three factors susceptible of determination on a small sample of blood in obtaining information as to the state of an individual's acid-base equilibrium, (1) the pH, (2) the free CO_2, (3) the combined CO_2. The relationship of these three has been expressed mathematically in the form of the Henderson and Hasselbach equations where

$$cH = k \frac{(H_2CO_3)}{(NaHCO_3)} \quad \text{or} \quad pH = \text{r}K + \log \frac{(NaHCO_3)}{(H_2CO_3)}$$

From a practical angle the determination of the pH of blood is difficult in order to achieve the accuracy that is necessary. Generally it requires the use of a potentiometer and an electrical assembly which requires constant and expert attention. There is a method of determining pH by means of the indicator, phenol red, which can be applied to routine procedure. It is however easier and more accurate to determine the total carbon dioxide content of the blood. This can be done on about 5 c.c. of oxalated venous blood in a small glass apparatus devised by Van Slyke and Cullen (1917) in which the free and combined carbon dioxide can be estimated by extracting the CO_2 by means of a vacuum and measuring its volume directly. Strictly speaking the alkali reserve is the total CO_2 content of the blood minus the free CO_2. Expressed as cubic centimeters of gas in 100 c.c. of blood the normal would be

$$\frac{3}{60} = \frac{\text{Free}}{\text{Combined}}, \quad \frac{2.5}{40} = \text{acidosis}, \quad \frac{3.5}{80} = \text{alkalosis.}$$

This is the more extended expression of the common statement that the total carbon dioxide content of the blood plasma is normally 53-74, whereas in

acidosis it is less than 50 and in alkalosis more than 75. This is the method of choice for routine clinical work in that it can be done rapidly on very small amounts of plasma. There is still another method in which the CO_2 of alveolar air is determined and expressed as millimeters of mercury. This method requires special apparatus and careful technique in gas analysis. Normal alveolar air contains 4.7 to 6.8 per cent CO_2 or a partial pressure of 35-53 mm. of mercury. In acidosis this figure diminishes because of increased respiration while in alkalosis it rises with diminished respiration. A value of 2 per cent CO_2 means that coma may supervene within 24 hours. A value of 3 to 4 per cent is less dangerous; at the worst coma will not appear for at least two or three days.

We have now defined the conception of acidosis in relation to the fundamental acid-base balance in the body and outlined the methods of measurement. We can next direct our attention to the conditions which alter the normal equilibrium and thus tend to give rise to acidosis.

(1) *Acidosis from overproduction of acid.* This is the most common cause of acidosis. With the large production of carbon dioxide from the daily intake of food it will be quite evident that any abnormality in this mechanism giving rise to some other product will soon flood the system with that product. If this substance happens to be an acid not excretable by the lungs the body must adjust itself. This is what happens in diabetes. Fats are not completely oxidised, and, instead of giving rise to nothing but water and carbon dioxide, intermediate products, the acetone bodies, appear in the system. Two of the acetone bodies are acids known respectively as acetoacetic acid and beta-hydroxybutric acid. The third is a neutral compound, acetone. The latter is highly volatile and escapes by the lungs but the acids are not and are immediately neutralized by the bicarbonate. They are then excreted by the kidneys. The acidity of the urine rises. This condition of the appearance of the acetone bodies in the system is defined as *ketosis* and their presence in the urine as *ketonuria*. The neutralization of these acids will however tend to diminish the alkali reserve and will entail a loss of base when excreted in the urine. To prevent this the kidney possesses a further reserve in its ability to produce ammonia and to substitute this in place of the bases of the blood. This mechanism is only moderately efficient. The ability then of an individual to resist this attack will depend on his alkali reserve. A *ketonuria* is not sufficient evidence on which to diagnose the condition of *acidosis* but must be taken as a danger-signal since continued production of acetone bodies will eventually lead to acidosis. The condition passes from compensated to uncompensated with respect to the $H_2CO_3/NaHCO_3$ ratio through loss of bicarbonate. In well compensated cases coma may quickly supervene. This is the condition in which the rising hydrogen ion concentration of the blood stimulates the respiratory centre and typical "air hunger" or "acyanotic dyspnoea" results. The customary treatment with insulin will remove the cause of the acid production but will not replace the base lost through the kidney. This will be remedied either through administration of some alkali *per os* or in due time through the foodstuffs eaten and the concontinued production of ammonia by the kidneys.

It must be realized that excess production of acids of the same or like nature arises in other pathological conditions. An interesting example of this is found in the use of ketogenic diets in the treatment of pyelitis and epilepsy. It may result from starvation which may in turn be caused by so

many diverse conditions. Prolonged nausea due to pregnancy or malignancy in the alimentary tract, fever, post-operative inanition, undernutrition, are all to be considered in this category.

(2) *Acidosis due to primary alkali deficit.* As pointed out above the kidneys play an important part in the excretion of acids, either as free acids, or combined with basic radicals, such as sodium, potassium and ammonia. Should the kidneys fail to perform this function there will be a retention of acid in the blood and consequently a neutralization of available base and a lowering of the alkali reserve. The system will shift from compensated to uncompensated acidosis. Such a condition may arise in acute or subacute nephritis and in the terminal stages of chronic nephritis. L. J. Henderson has remarked, "nothing is more wonderful or instinctive than the condition of an individual who has long been suffering from a disease like chronic nephritis, who is approaching his end but who remains adapted in every part and in every activity to the changed and almost impossible conditions of life." The retention of acid phosphate and sulphate from the oxidation of protein constitutes the danger and is due of course to the inefficiency of the kidney. This type of acidosis may be caused by prolonged, severe, muscular exercise due to the accumulation of lactic acid in the system. It may be brought about by the ingestion of large amounts of ammonium chloride. It may develop from many types of poisoning such as mercuric chloride and from the loss of fluid and base in cases of severe or prolonged diarrhoea. The determination of the total carbon dioxide content of the blood is a useful index of the condition. A value of less than 30 would be considered very severe. There is also a hyperphosphatemia. The symptoms of this type are essentially the same as in the first type.

(3) *Acidosis from increased free carbon dioxide.* This condition may arise from respiratory obstruction or insufficiency. Here the carbonic acid of the blood rises although the bicarbonate content remains within normal limits. Compensation is essayed by increased breathing and an effort on the part of the kidney to retain fixed base. The urine becomes more acid. Such a condition arises clinically in pulmonary impairment as in emphysema, and any condition tending toward anoxemia. Depression of the sensitivity of the respiratory centre may do the same thing as in morphine poisoning but in this case the subject breathes more slowly.

Sufficient has been written of the general principles involved to indicate when an acidosis may be expected and its probable nature. Those who have attempted to outline a ketogenic diet will realize the difficulty in producing a ketonuria in the essentially normal individual. Such statements that a diet may be too acid and will give rise to "too much acid in the blood" have no foundation in fact unless there be definite pathology present.

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THE AMERICAN COLLEGE OF SURGEONS IN CONGRESS

THE American College of Surgeons was at home to her Fellows and their guests, the last week of October. Surgeons from the four corners of the continent and this little appendage of Nova Scotia, as well, thronged the city in numbers that must have approached the three thousand mark. Chicago must always be the happiest meeting place of the American College; what hostess is not most gracious in her own drawing room? The beautiful buildings at No. 40 East Erie Street made an impressive background for the Convocation exercises. The John B. Murphy Memorial Hall, its chaste, classic beauty offset by the colorful blue and scarlet gowns of the initiates, filled us with an awe that we thought had gone with other illusions of our undergraduate days.

Situated in the heart of the continent, though a bit more to the right of the midline than the left, Chicago is an ideal convention city. Hospitals and medical schools are in abundance and there was a touch of homeliness in the Chicago Tribune's lusty attack on the disgraceful conditions said to exist at the Cook County Hospital. The world is, indeed, our neighbor. Despite Fellows from California, British Columbia and Texas, Nova Scotians were looked upon as akin to the Martinians, visitors from a land of dreamstuff, rather than granite and earthiness. And, of course, we met the inevitable chap—he attends all such gatherings and this time hailed from Pittsburg—who, on hearing of our nativity, inquired solicitously about the Grenfell hospital at Labrador. On plumbing the depths of our ignorance on this score he happily remembered that Halifax had had an explosion recently. Our memory centre for figures, usually most recalcitrant, was stimulated by the midwestern air and we fortunately were able to entertain him with satisfactory statistics of carnage and gore.

Particularly noticeable in the Convocation proceedings was the increasing severity of Fellowship standards. The College's earliest efforts in hospital standardization, cancer control and even her more recent advances in the field of medical services in industry, being on a firm footing, more rigid standards are being demanded of fellowship applicants. The prize winning case reports of this year, in a bound volume of 2000 pages, culled from the major surgery of two years past, were masterpieces of detailed composition, illustrated by watercolors and microphotographs. Graduates of 1938 and thereafter, will

be required to submit certificates of three years of interneship in approved hospitals, in addition to their case records.

Perhaps the most interesting phase of the clinical program, to us, lay in the opportunity of hearing men whose names are famous in their surgical fields. Kanaval, who in his *Infections of the Hand*, presents what is probably the most complete and scientific monograph in surgical literature, carries out in his teaching and practice, the same principles of which he writes. He considers that three or four hours spent in the repair of a deep wrist wound is not slow, but sound and thorough surgery. All those who have been face to face with this practical problem in surgical anatomy will be in hearty accord with him. Cheatle, of London, made the sound, meticuluous presentation that we expected of the author of the masterpiece on the surgical pathology of the breast.

There were no subjects introduced of a startlingly new nature. The most talked of, recent development was the internal fixation of fractures of the femoral neck. Gurd, of Montreal, presented scientific work to show that bone atrophy following fractures, is due, not to poor circulation and blood stasis but to its converse, a relative hyperaemia. Sherman, of Philadelphia, in the fracture of oration, made a brilliant plea for the use of bone plates where good closed reduction cannot be obtained.

We were disappointed in the almost universal use of the formal, prepared address as a method of presentation in the symposia at the general conferences. Granted that the medical man, the world over, is a poor speaker, this form, which has been consistently discouraged at Dalhousie Refresher Courses for years past, because of its undoubtedly poor teaching qualities and its lack of appeal to the listener, was used by experienced teachers who had a sufficiently thorough grasp of their subject to talk for hours unprompted.

The ubiquitous movie, which is supplanting, more and more, the legitimate stage in Chicago, made its appearance with greater vigor than ever before at the congress. The potentialities of the moving picture as a teacher of surgery are almost without bounds. In what operating theatre can you, as a guest, put your nose in the incision and keep it there with the same all-seeing consistency as can the movie camera? It is true, the camera closes its lense to the little artery that pumps away in the pelvis and resists hot pads and imprecations. It does not see the mesenteric glands left behind in the resection of a carcinomatous colon. Hands move with the facility that only a speeding film can create and surgery becomes a science of quick and effortless simplicity. But it teaches through the eye which absorbs faster than the ear. Without the qualifying judgement of an experienced man, it would probably be an unwise dish to place before the ambitious medical student, but to those who can appreciate the unseen difficulties, it is of great value. These films, approved by the American College, are available for medical societies.

It is difficult to assay the benefits, to the individual, of a gathering such as this. In the notebook of his memory he can enter a few cold facts that he has acquired. These seem as slight reward for the effort expended. But beyond all this is an intangible something of more real value; perhaps it is made up of the little things, gathered on the way, without conscious effort, and silently assimilated into what we call experience; and above all, the realization that, alone with his home problems though he may be, he is, in communion with his fellows all over the continent, working for the greater glory and advancement of his profession and the happiness of mankind.

A. L. M.

YOUR attention is directed to a letter from Dr. Burris to be found under correspondence, on page 639, in which he comments on a recent editorial appearing in these columns.

The matter under discussion was written to further the ends of preventive medicine, and to emphasize the fact that venereal diseases are not a peculiar and special visitation for sin, but everyday communicable diseases. I attempted to examine in daylight that which I believed to be the basic reason for our inertia and our everlastingly dodging the issue by covering our heads with a blanket of "hush, hush". Unfortunately, such an examination as this cannot be successfully carried out without touching the subject of morals; in fact, if it were not for their entanglement with venereal disease there would be no reason for even an indirect reference to them. For example, pneumonia or brain abscess can be discussed without reference to morals: at the present moment this does not apply to venereal disease and that is, I believe, the explanation of the many papers which start to discuss this subject but never arrive, why legislation is enacted and straightway forgotten or worded so as to be useless.

You will believe me when I say that I deeply regret giving offence to anyone's religious sensibilities, and I can assure you it distressed me, more than it did any of my readers, on learning of this unhappy result. My intention was to stimulate discussion as to the best way of applying preventive principles to the end that our people be freed from these scourges. I concluded my remarks with a series of constructive suggestions. The first on the list involved definite mental effort which, if made, would result in a fundamental change of outlook.

My critic is definite in his accusations, but fails to present a single quotation as evidence in their support. Perhaps it is just as well that this method of registering protest is resorted to, as it rules out any possibility of a defensive answer and thus brings to an abrupt close any discussion that might become tinged with an element of religious controversy. My remarks may have been expressed imperfectly, but I would ask you to be so gracious as to overlook such de fects andgive attention to their purpose.

The article was prepared as a serious contribution to a serious problem and I would earnestly ask that you reread it slowly, calmly, with a mind free from prejudice and foregone conclusions and then, after due reflection, decide whether you are for or against the gonococcus as a participant in the marriage contract. If against, are you willing to jettison ideas that may stand in the way of many fruitful and happy marriages?

H. W. S.

CASE REPORTS

Manic Depressive Psychosis.

G. M., male, aged sixty, clerk, consulted me in December, 1933. His complaints were weakness, worry, constipation and sleeplessness. On questioning him he expressed the opinion that something very serious was wrong with his bowels and that he believed the drinking water in his residence had been deliberately poisoned.

Family History. One sister had an attack of melancholia ten years before his illness which required institutional care for over a year, but which eventually showed enough recovery to admit of her return to near normal life. His father showed fits of depression of varying intensity and length for many years before his death, but never apparently reaching a definite psychosis.

Personal History. There is nothing of particular interest to note in the personal history. The patient led a normal life, with family life also normal. He is married and has a normal son and daughter, now in early adult life. He was a moderate but enthusiastic user of alcohol.

Examination. The appearance of depression and woe immediately strikes one. The colour is good, but the face is wrinkled and anxious. On examination the presence of many delusions were brought out. "Don't bother listening to my lungs they were destroyed by the poison they gave me", etc. The heart and lungs were normal. The B.P. was 144/86. Nervous system negative. The arteries, in spite of the near normal blood pressure reading, were extremely hard and could be rolled under the fingers when one palpated the wrist and arm. The skin was wrinkled and dry and the weight low, with history of rapid loss of weight in the past two months. Examination was undertaken in the Halifax Infirmary a few days later with the following results: urine, Kahn and blood chemistry negative, X-ray of chest, G. I. series and rectal examination negative. A diagnosis of manic-depressive psychosis was made and the patient was returned to his home.

Differential Diagnosis. The only other condition that merited consideration was physcosis with cerebral arterio-sclerosis, but this was pretty well ruled out, the hard arteries notwithstanding, by the absence of memory defect and progressive dementia which are so characteristic of this condition.

Treatment and Progress of Case. The treatment was symptomatic only and brought neither comfort to patient and relatives, nor satisfaction to the medical attendant.

For three years he remained in bed alternating between short attacks of acute mania and long attacks of profound depression. He frequently required food, or sedatives, or both, by tube feeding. The only outstanding feature during this period was the devotion of his life partner who refused to commit him to an institution, and who nursed him with a devotion rarely seen in this material age.

In January of this year he started to improve, both mentally and in appearance and weight. This improvement continued and he started going

out for short walks about three months ago. One month ago he himself brought up the question of the most consistent and outstanding of his delusions, namely, that he was being poisoned via the water entering his home and the time seemed opportune to try the effect of contradictory proof. This was furnished, in the form of a satisfactory report on the water supply, from a competent pathologist and was accepted by the patient. With it his last delusion vanished.

Comment. This case illustrates the fact that manic-depressive insanity even when long continued very frequently ends in recovery. This recovery may or may not be permanent. It also illustrates the fact that while contradictory proof is of no value against a fixed delusion it may be of value once improvement starts and the patient begins to waver in his delusional trend.

J. W. MACINTOSH.

Secondary Carcinoma—Primary Focus Undiscovered.

J. M., male, aged sixty-seven, merchant tailor, asked my advice in June, 1936, because of three painful lumps on his back. He complained also of poor appetite, weakness, shortness of breath and headache.

Family and Personal History. Unimportant. There was no family history of carcinoma or tuberculosis and the patient himself had led a well ordered, uneventful life.

Present Illness. For about three months the general health had not been good. In that period the loss of weight was at least twenty-five pounds. General malaise, weakness, palpitation, shortness of breath and poor appetite all made their appearance in this period.

Examination. All systems were negative except the observations that he was quite anaemic and underweight and that his few remaining teeth were in a very foul condition. Three lumps were present over the right scapular area. They were about the size of marbles—very hard, adherent to the skin but not to underlying tissue and showing some evidence of redness or inflammation at the skin margins. He was given a tonic—Eastons syrup in tabloid form, told to consult his dentist and return in one week. On his return many more similar but smaller nodules were present on his back, and the anaemia and general condition were obviously worse and he was referred to the Halifax Infirmary for investigation. A frantic search in the medical literature at this period was not very helpful though the nodules appeared to resemble the textbook story of molluscum fibroma.

A complete physical examination (with rectal examination) was undertaken in hospital and was essentially negative. The special investigation was as follows—

(1) Urine and Kahn examinations negative.

(2) Blood picture—"Erythrocytes.....2,400,000
Leucocytes..... 10,200
Haemoglobin..... 30%
Lymphocytes 9: Monocytes 7: Eorinophiles 1: Basophiles 0: Juveniles 0: Stab cells 2: Segmented 81: Total Neutrophiles 83."

There is moderate anisocytosis—the microcytes are numerous. Polychromasia is marked. There is very slight poikilocytosis and very little stippling. No neucleated reds. The leucocytes are of normal type. (A fairly severe anaemia of secondary type with definite bone marrow activity.)

(3) X-ray examination: (report issued after the pathological report).

"Examination of the G. I. Tract. Oesophagus—normal. Stomach—the stomach is honeycombed by numerous small oval tumors. No sign of malignancy—primary. Deudenum is normal. Function of the stomach is good. Small intestine—negative. Colon—no pathological change noted. Chest—old tuberculosis noted in the upper lobes but healed. Heart is normal in all respects. No metatases noted anywhere. Comment: The appearance of the stomach is part of the general picture of (as we know now) metastatic carcinoma."

(4) Biopsy on tissue from one of the larger nodules on back.

"The histological appearances reveal a secondary malignant adeno-carcinomatous deposit."

Diagnosis. Secondary adeno-carcinomatous deposits in subcutaneous tissue. Primary focus undiscovered.

Progress of Case. The patient returned home, where he died six weeks later. The nodules by this time had appeared in hundreds in the subcutaneous tissue all over the body, including the face and the hands and feet. No severe pain was present in the abdomen at any time, most of the pain and discomfort being from the subcutaneous lesions. Occult blood was demonstrated in the stools but no bright haemorrhage. Sugar did not appear in the urine. While wasting was extreme and the abdominal wall thin and relaxed it was not possible at any time to palpate any pathological mass in the abdomen. No post mortem could be obtained.

Comment. The wide distribution of the secondary lesions and the method of dissemination were the unusual features here. The position of the primary lesion remains undiscovered.

J. W. MACINTOSH.

Abstracts from Current Journals

MEDICINE

Hypertension with Experimental serum Nephritis.—Melville, et al; Clinical Reflections and Recollections (xii) **Abdominal Surgery in Infants and Childhood.—Herzfeld.**

Studies in Method and Standardisation of Blood Serum (V) **The Blood Platelet Count.—Harvey; Erythrogenic Toxins of Haemolytic Streptococci.—Thomson; Tuberculin Survey of Children in the West of Scotland.—Blacklock,** etc. *Edinburg Medical Journal—April,* 1937—*New Series (iv) xliv.*

From the excellent articles in this copy of the Journal some highlights are selected from the second Article—Abdominal Surgery in Infancy and Childhood—Herzfeld. It is a pity to attempt to condense this article, it should be read in its entirety:

The vitality of the new born infant is amazing, for operations a few hours after birth and very severe are often the safest in surgery, e.g. exomphalos in which 2/3 of the abdominal contents lie outside the abdominal cavity.

The Author stresses the difficulties of the "acute abdomen". The operator must note (1) the extraordinary progress of the inflammatory lesion in the peritonial cavity, e.g. in appendicitis. (2) The rapid deterioration in acute obstructive lesions with the result that early operation is more urgent than in the adult.

Diagnosis must be made early. The history is obtained from a relative, not the patient, and is often inaccurate. The examination of a child may be very difficult and one may have to use an anaesthetic and that has obvious disadvantages. Examine the child when asleep if possible.

The general appearance and posture of the child gives valuable information, e.g. the "abdominal facies" or, the drawn-up legs in appendicitis. Inspect the patient before palpating for the later may cause pain. Make an examination of the rectum with the little finger. Examine the urine. Mark the rate of the pulse and the respiration. If the temperature is over 102, one is rarely dealing with an acute abdomen.

Dr. Herzfeld then gives a classification of nineteen conditions causing "acute abdomen" and deals carefully with the following: intussusception, appendicitis, congenital lesions and lesions due to foreign objects in the abdominal tract.

Some conclusions are drawn as follows:

(1) Early and accurate diagnosis.
(2) Careful preoperative treatment, i.e. make good the loss of fluid by intravenous injection.
(3) Gentleness combined with speed in operating.
(4) Limit operative treatment to absolute essentials. There is special emphasis laid on the dangers due to purgation.

L. R. M.

Sciatica and Lumbago, Wilcox; Observations on Agranulocytosts, Wilkins and **Israels; Cardiac Emergencies, Hoskins; Recent Advances in Anaesthesia, Dawkins; Diagnosis of Prostatic Enlargement, Power; Without Comment, Hunter.** (This article is a comment on Obstetrical cases.)—*The Clinical Journal*—1937.

The first Article is chosen for review:

SCIATICA AND LUMBAGO:

Sciatica is defined as an "Intestinal Fibrosis of the sciatic nerve."

Lumbago is defined as a form of fibrosis of the muscles of the loin and their aponeurosis, tendinous attachments and fibrous tissue. Aetiology-lumbago and sciatic are both forms of fibrosis, and both are varieties of rheumatism. Consider the causes of these diseases—(a) Is there an infective cause? Is there a focus of infection?

Specific infective causes are: Gonorrhoea, syphilis, tuberculosis, brucella fever, bacillary dysentery, typhoid fever, etc.

Non-specific causes are: Teeth, tonsils, bowels, bladder, urogenital tract or pelvis.

(b) Physical causes are: Injury, strain, twists, cold, living in damp houses, insufficient clothing, posture, fatigue, mental strain, and overwork.

(c) Metabolic causes are: Glycosuria, renal conditions and gout.

(d) Dietetic causes are Alcoholic Drinks.

(e) Circulatory conditions especially in the lymphatic circulation.

(In such conditions the Oestopath shines over his regular practitioner.)

(f) Action of the skin.

(g) Endocrine factors.

(h) Physical factors.

Symptoms of Sciatica are—pain from the buttock to the heel and spasm of the muscles of the loin.

It is essential to map out accurately the anatomical site of the nerve and ascertain if it be tender along its course.

Be sure to eliminate hip, spine and pelvic troubles.

Symptoms of Lumbago may come on gradually or be extremely acute. There is great pain and tenderness on one or both sides of the loin. Carefully examine spine, nervous system, sacro-iliac and hip joints, as well as for renal calculus. An X-ray is essential.

The Author advises the following: Blood count, haemoglobin test, sedimentary rate test (to demonstrate an acute toxic condition and for probable general infection) bacteriological and biochemical tests, e.g. blood-uric, blood-urea, blood-calcium (parathyroid) and urine.

The Author then demonstrates four cases with regular rambling remarks, only two of which are sciatica and none of lumbago.

Treatment of Sciatica—keep patient absolutely in bed and apply heat, e.g. infra-red irradiation, antiphlogistine or electric pad. Give phenacetine, aspirin and codeia separately or in combination. He says, never give morphia.

In extraordinary cases use X-ray in slight doses to reach affected parts, or, inject the nerve first with 2-3% novocain followed by 5 occ of sterile normal saline.

In case of infection use autogenous vaccine.

L. R. M.

Classification of the Glycosurias—(*B. M. Journal—January* 16, 1937) T. H. OLIVER, Manchester Infirmary.

AN attempt is made to classify the glycosurias in above article—Mortality statistics of diabetes mellitus show the expectation of life has been increased 3½ years since the introduction of insulin. This gain is entirely in young patients, after 55 mortality has continued to increase at the same rate as prior to 1922 when insulin came into use. The glycosuria of a man of 80 and acute diabetes in a child of 8 may give the same fasting blood sugar figures and both are labelled diabetes but the treatment is different in the two cases. In other words we must differentiate the types or we may use the remedy (insulin) in cases when the primary fault is not a deficiency of the pancreas.

Many text-books state definitely that diabetes mellitus is due to a deficiency of islet tissue although physiology has shown that glycosurias may be due to a larger variety of causes. Statistics show that the young patient dies of coma while in the older potient no deaths are due to this cause. In the younger, he dies of a disturbance of his metabolism but in the older patient the complications are the dominant factor.

The classificiation of glycosurias depends on the complicated subject of carbohydrate metabolism,—(1) Three groups of organs appear to be involved, the nervous system, the pituitary, the thyroid, possibly the adrasuals. (2) We have the islet tissue at fault, an impaired production of insulin which appears to be concerned with the storage of glucose as glycogen. (3) The liver and muscles are the great store-houses of carbohydrates and the principal users of sugar.

Therefore it would appear that the four possible causes of hyperglycaemia are as follows—

1. Increased katabolism leading to an increased mobilization of carbohydrates and fat from the store-house in which the nervous system, the pituitary and the thyroid are involved.
2. Dimished capacity to store owing to lack of insulin which is pancreatic disease.
3. A defect in the store-houses themselves, the liver and muscles, as seen in liver disturbances, gout and so forth.
4. An inability of the tissues to use as in pernicious anaemia and arterio sclerosis.

After giving case histories illustrating above conditions in which liver therapy in pernicious anaemia and thyroidectory had improved the glycosuria without insulin, his conclusions are:—

1. Glycosuria is a sympton and not an entity and each particular case must be studied to find the primary disturbance.
2. The optimum blood sugar level may not coincide with the so-called normal blood sugar level.
3. Successful treatment is not the achievement of a laboratory ideal, the rationale of which we do not fully understand, but whether the patient does or does not show clinical improvement.

L. R. M.

Immunologic Allication of Placental Extract—(*New England Journal of Medicine—March* 18, 1937)

The development and use of Placental Extract for immunity is the outgrowth of the search for a better method of controlling measles. The use of convalescent serum of children exposed to measles dates from the observation of Nicolle and Conseil in 1918. Certain hospitals have for a number of years used convalescent serum effectively in the control of measles. But the use of this serum has been limited by the lack of adequate supply; and in the control of measles by this means it has proved impractical. The results of the use of adult serum has led to the general acceptance of the observation that the blood of most urban adults contains the protective antibodies. Furthermore if the mothers are immune infants during the early months of life are also immune to measles, apparently by reason of the passage of antibodies from the mother to the foetus through the placenta. In addition the new-born infant is usually immune to poliomyelitis scarlet fever, diphtheria, and certain other diseases. The actual presence of immune bodies in the unbilical cord blood have been amply demonstrated. These observations have formed the back ground of investigations into the use of the human placenta and the contained blood as a source of antibodies against measles.

Placenta Extract can be prepared under large scale furnishing a sourc of measles serum in the form of human immune globulins and at the moment placenta extract the best material for use in the control of outbreaks of measles in institutions.

Immunity following injection of placental extract is passive in type and of short duration. Two weeks is the period of immunity and seems tobe the limit of protection against re-exposure. The administration of placental extract is intra-muscularly and orally. In some instances a local or general reaction, but generally mild, is seen in about six per cent of patients following intra-muscular injection.

L. R. M.

SURGERY

Inguinal Hernia, Repair By Intraperitoneal Approach.—Sutton, *Annals of Surgery*, December, 1936.

As one reads this article one reflects on the many and varied techniques that have been suggested for the radical cure of inguinal hernia. Many of these have passed to the limbo of forgotten things and the conservative operations of Bassini, MacEwen, Halstead and others have well stood the test of time. The method of approach suggested is not an entirely new one having been put forward long before this article was written. It possibly has some advantages in a difficult case with internal ring adhesions but as a routine type of operation it would appear that the difficulties of technique are not found offset by the increase of statistical cures.

One does not anticipate any great swing to the newly suggested approach but the article is well written and worthy of careful study.

Spinal Anaesthesia.—Saklad, *American Journal of Surgery*, January, 1937.

A most interesting and instructive article is contributed by the author to the January issue of the American Journal of Surgery. The present status

of spinal anaesthesia shows an increasing agreement among anaesthetists of formerly disputed phases. Methods are more standardized and there is greater care in the details of administrative technique. This type of anaesthesia has of recent years been extensively used and in this article one may find a very excellent presentation of all the factors entering into spinal anaesthesia. The outstanding difficulties are failure of respiration and fall in blood pressure. Suitable treatments for these difficulties are suggested as well also as a table showing the relative toxicity of the different drugs employed in producing the anaesthesia. One is rather struck by the absence of novocain from the list of drugs employed.

The article is well written, instructive and a worthy contribution to the "symposium on anaesthesia" issued by this journal in its recent issue.

D. A. MacL.

CORRESPONDENCE

Dartmouth, N. S.,
November 8th, 1937.

The Editor,
THE NOVA SCOTIA MEDICAL BULLETIN,
Halifax.

Sir:

I should like to draw attention to the tone and the content of the Editorial which appeared in your October number, but insofar only as that article dealt with morals and religion. The article was entitled "Fornication and Disease". May I make a few comments.—

First. The title itself might have been improved upon.

Second. The parts of the article which introduced and dwelt upon the moral and religious aspects of the question were very unfortunately worded. I am convinced that the writer gave far too little thought to the exalted and sacred nature of some of the problems which he discussed and that, in his manner of presenting them he failed to do himself justice. Nevertheless as the words of the Editorial appear they will offend the sensibilities and come into conflict with the moral convictions of the vast majority of those who may read them. If they are to be taken at their face value one must conclude that they arise from a system of thought which is in serious error concerning some of the greatest tenets of Christian belief and teaching. These parts of the Editorial may be described very concisely I think, by that phrase "Old heresies in the shape of new doctrine".

Third. I doubt very much the wisdom of introducing moral and religions matters in any subject of this type, published in The BULLETIN but, if they must be introduced let every care be taken to deal with them in a manner which is appropriate to their high and difficult nature, as well as to their very great importance.

The BULLETIN will be read by many people not of our profession and I believe that, if this Editorial should go unchallenged, impressions will gain headway concerning the profession at large which will be unmerited by them. This is one reason which has impelled me to write as I do. But, even if it should go no further than our office desks, the objectionable features are too apparent. There are many members of the profession, subscribers to The BULLETIN who regret the moral and religious conclusions as they are stated or implied in the Editorial, and as one of them, I protest.

M. G. BURRIS.

Department of the Public Health

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Those physicians wishing to make use of the free diagnostic services offered by the Public Health Laboratory, will please address material to Dr. D. J. MacKenzie, Public Health Laboratory, Pathological Institute, Morris Street, Halifax. This free service has reference to the examination of such specimens as will assist in the diagnosis and control of communicable diseases; including Kahn test, Widal test, blood culture, cerebro spinal fluid, gonococci and sputa smears, bacteriological examination of pleural fluid, urine and faeces for tubercle or typhoid, water and milk analysis.

In connection with Cancer Control, tumor tissues are examined free. These should be addressed to Dr. R. P. Smith, Pathological Institute, Morris Street, Halifax.

All orders for Vaccines and sera are to be sent to the Department of the Public Health, Metropole Building, Halifax.

Report on Tissues sectioned and examined at the Provincial Pathological Laboratory from October 1st, to November 1st, 1937.

During the month, 231 tissues were sectioned and examined, which, with 24 tissues from 7 autopsies, makes a total of 255 tissues.

Tumours, simple	18
Tumours, malignant	31
Tumours, suspicious of malignancy	2
Other conditions	180
Tissues from 7 autopsies	24
	—255

Communicable Diseases Reported by the Medical Health Officers for the month of October, 1937.

County	Chickenpox	Diphtheria	Infantile Paralysis	Influenza	Measles	Mumps	Paratyphoid	Pneumonia	Scarlet Fever	Typhoid Fever	Tbc. Pulmonary	Tbc.-other Forms	V. D. G.	V. D. S.	Whooping Cough	Epidemic Jaundice	Catarrhal Jaundice	Impetigo	TOTAL
Annapolis	..	..	..	..	..	1	2	..	1	..	..	..	5	..	..	..	..	..	9
Antigonish	..	..	..	..	..	4	..	..	..	1	..	..	..	..	..	4	..	..	9
Cape Breton	1	2	..	..	..	..	..	..	25	..	..	..	..	..	12	..	..	..	40
Colchester	..	..	3	3	..	..	..	..	..	..	..	..	..	..	..	..	..	..	6
Cumberland	..	..	1	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	1
Digby	..	..	..	2	..	..	..	..	..	2	2	..	..	..	10	20	..	..	36
Guysboro	..	..	..	..	..	..	..	1	..	..	..	..	..	..	..	..	..	..	1
Halifax City	..	2	..	..	1	..	..	..	11	1	..	..	..	..	..	..	..	..	15
Halifax	..	..	..	3	..	3	..	..	..	..	..	..	..	..	..	..	..	..	6
Hants	1	..	..	..	..	1	..	..	1	..	..	..	3	1	2	..	..	..	9
Inverness	..	..	..	3	..	..	..	..	..	..	..	..	..	..	..	..	..	..	3
Kings	7	..	..	11	..	1	..	1	..	..	..	..	2	..	..	..	..	..	22
Lunenburg	10	..	1	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	11
Pictou	..	..	..	2	..	..	..	..	2	..	..	..	..	..	..	..	..	..	4
Queens	..	..	2	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	2
Richmond	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	...
Shelburne	..	..	..	..	13	..	..	..	2	..	1	..	2	1	..	..	..	..	19
Victoria	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	...
Yarmouth	..	..	..	..	25	..	..	..	1	..	..	..	..	..	1	..	4	..	31
TOTAL	19	4	7	24	39	10	2	2	43	4	3	..	12	2	25	24	4	..	224

Positive cases Tbc. reported by D. M H. O's. 47

RETURNS VITAL STATISTICS FOR SEPTEMBER, 1937.

County	Births		Marriages	Deaths		Stillbirths
	M	F		M	F	
Annapolis	9	7	28	10	7	0
Antigonish	20	18	13	5	5	1
Cape Breton	125	111	104	54	38	4
Colchester	24	16	21	5	4	1
Cumberland	31	34	54	18	16	4
Digby	24	16	40	19	9	3
Guysboro	20	9	14	5	2	1
Halifax	87	87	121	55	32	3
Hants	28	27	29	8	4	0
Inverness	22	17	18	14	12	1
Kings	20	21	34	11	6	0
Lunenburg	25	28	28	9	11	1
Pictou	24	19	36	10	7	0
Queens	13	4	15	4	6	0
Richmond	22	20	15	5	13	0
Shelburne	21	17	16	13	10	1
Victoria	5	4	6	1	1	0
Yarmouth	15	12	50	21	15	1
	535	467	642	267	198	21

Medical Library, Dalhousie University

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Epidemic of Paratyphoid B Fever in Liverpool and District by W. M. Fraser, B. T. J. Glover and V. Glass.
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Basal Anaesthesia for Short Operations by J. H. T. Challis.

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Physiological Bases of Nutrition by Stuart J. Cowell.
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The Action of Meat Extracts and Related Substances as Gastric Stimulants in Man by William Robert Boon.
Treatment of Spastic Paralysis by F. H. Mills.
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Medical Ethics and the Art and Practice of Medicine by Lincoln Davis.
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Treatment of the Menopause Syndrome by Irradiation of the Pituitary Gland by Robert Zollinger and Walter W. Vaughn.

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Personal Interest Notes

Dalhousie Medical School admitted into membership of the Association of American Medical Colleges.

Word has been received from Dalhousie University that at the meeting of the Association of American Medical Colleges held in San Francisco during the last week in October the Dalhousie Medical School was admitted into membership.

The Association of American Medical Colleges is a voluntary organization made up of representatives from accredited medical schools in the United States and Canada. It has been in existence now for over forty years and during that time has devoted its energies to the improvement of medical education. It works in co-operation with the Council on Medical Education of the American Medical Association, the Canadian Medical Association, the Medical Council of Great Britain, and other important bodies dealing with medical education and licensure. To become a member of the Association of American Medical Colleges certain minimum standards are required. There must be so many full time well trained teachers according to the number of students; the school must have proper facilities and equipment for teaching fundamental subjects, such as anatomy, physiology, biochemistry and pathology. The affiliation with hospitals must be such as to allow of sound clinical teaching to the students. In attaining membership the Dalhousie Medical School has come up to all these standards.

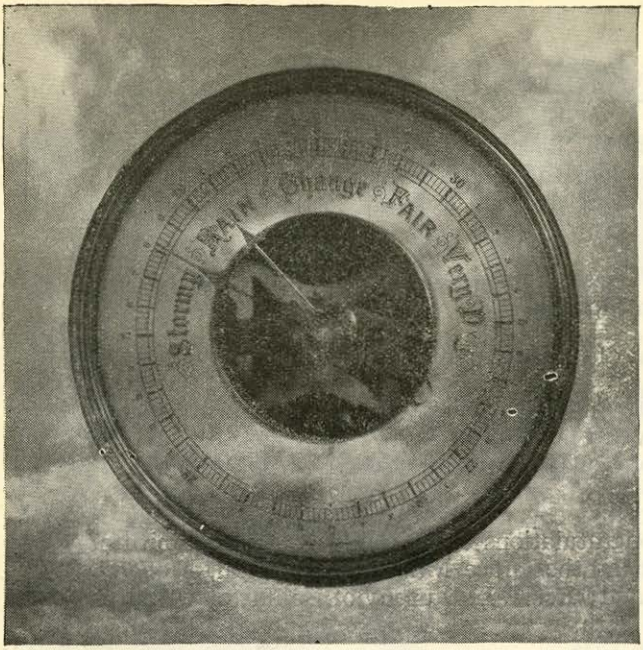
The benefits of membership in the Association of American Medical Colleges are many. First, there is the honour attached to membership which is comparable in many ways to the Grade "A" rating of the Council on Medical Education of the American Medical Association. The staff benefits by receiving the Journal on Medical Education which is issued by the Association, the only journal on Medical Education published anywhere. And there are benefits to the students. Dalhousie will continue to be known as a first class medical school and students attending Dalhousie will have equal opportunity with those attending other good schools in obtaining good appointments in post-graduate medical education.

Pictou Hospital receives splendid Gift.

Retiring from active operative work, Dr. Alexander Primrose of Toronto, has sent all his surgical instruments to Pictou Memorial Hospital. The distinguished surgeon, a native of Pictou, will make the presentation through Dr. G. A. Dunn of Pictou.

Dr. Dunn announced the gift would be a valuable addition to the institution's equipment as it included a number of instruments the hospital board could not afford to purchase. In all there are about two hundred and eighty instruments.

Born in Pictou in 1861, Dr. Primrose studied at Pictou Academy. Following his graduation from Edinburgh University he went to Toronto and has remained there ever since. He is a former Dean of the Faculty of Medicine of the University of Toronto.



Stormy Weather Ahead

The anticipation of winter . . . pleasant thoughts to many . . . but consider those to whom winter means a series of colds, with the possibility of other and more severe respiratory infections. Those persons whose resistance is low are naturally predisposed to such infections but the accumulation of clinical evidence shows that much can be accomplished by prophylactic measures.

ALPHAMETTES . . . Cod liver oil therapy, instituted early, will do much to lower the incidence of these respiratory infections and in Alphamettes—a simple concentrate of defatted cod liver oil—is found a most convenient medium for this therapy. Each small 3-minim capsule exhibits the full vitamin A and D value of over 4½ teaspoonfuls of cod liver oil (U.S.P. XI).

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MONTREAL CANADA

729

Dr. J. B. Reid of Truro has left for an extended vacation extending until after Christmas. The Doctor plans to spend a few weeks at his old home in Middle Musquodoboit and following that will study at Boston, New York and Chicago.

Dr. D. W. N. Zwicker of Chester is at present in New York where he is taking post-graduate study in medicine.

Dr. and Mrs. W. A. Hewat of Lunenburg are visiting in New York.

At the recent meeting of the American College of Surgeons held at Chicago the following physicians received Fellowships: Dr. Frank Mack, Dr. H. W. Schwartz and Dr. A. L. Murphy of Halifax, and also Dr. V. D. Schaffner of Kentville.

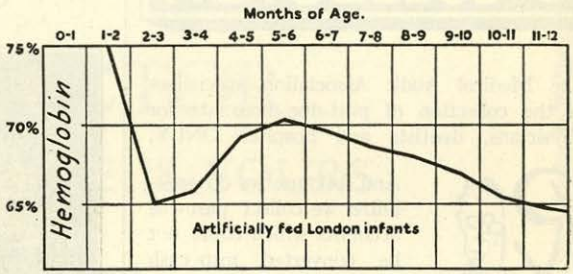
The BULLETIN extends congratulations to Dr. and Mrs. R. P. Smith of Halifax on the birth of a son at the Grace Maternity Hospital, Monday, October 25th, and also to Dr. and Mrs. A. L. Murphy of Halifax on the birth of a son on Tuesday, October 26th.

The marriage took place at Montgomery, Alabama, on Saturday, October 2nd, of Miss Dorothy Belden Davison, youngest daughter of the late Mr. and Mrs. George C. Davison of Moncton to Dr. George Edward Maddison, also formerly of Moncton. Dr. Maddison is a graduate of Dalhousie, '37, and is at present Health Officer for Monroe County, Alabama.

The BULLETIN extends congratulations to Dr. Victor O. Mader of Halifax who was recently admitted into Fellowship of the Royal College of Physicians and Surgeons of Canada.

The marriage of Miss Irma Jackson of Birmingham, Alabama and Dr. John Henry Budd son of Mr. and Mrs. Fred W. Budd of Halifax took place on November 4th at Cleveland, Ohio. The bride has been for two years a member of the staff of Deaconess Hospital as laboratory and X-ray technician. Dr. Budd, who was a graduate of Dalhousie Medical College in 1933, has been doing post graduate work in surgery for three years in Cleveland hospitals and has recently established a practice there. Immediately following the ceremony Dr. and Mrs. Budd left for a short honeymoon in Bermuda and after December first, they will reside in Cleveland.

Nutritional Anemia in Infants



The accompanying chart of the hemoglobin level in the blood of infants is based on more than 1,000 clinical cases studied by Mackay. The sharp drop in hemoglobin during the early months of life has also been reported by a number of other authorities. It is noteworthy that this fall in hemoglobin has been found to parallel closely that of diminishing iron reserve in the infant's liver. The usual milk formula of infants in early life further contributes to this anemia because milk is notably low in iron. It is now possible, however, to increase significantly the iron intake of bottle-fed infants from birth by feeding Dextri-Maltose With Vitamin B in the milk formula. After the third month Pablum as the first solid food offers substantial amounts of iron for both breast- and bottle-fed babies.

Reasons for Early Pablum Feedings

1. The iron stored in the infant's liver at birth is rapidly depleted during the first months of life. (Mackay,[1] Elvehjem.[2])

2. During this period the infant's diet contains very little iron—1.44 mg. per day from the average bottle formulae of 20 ounces, or possibly 1.7 mg. per day from 28 ounces of breast milk. (Holt.[3])

For these reasons, and also because of the low hemoglobin values so frequent among pregnant and nursing mothers (Coons,[4] Galloway[5]), the pediatric trend is constantly toward the addition of iron-containing foods at an earlier age, as early as the third or fourth month. (Blatt,[6] Glazier,[7] Lynch[8]).

The Choice of the Iron-Containing Food

1. Many foods reputed to be high in iron actually add very few milligrams to the diet because much of the iron is lost in cooking or because the amount fed is necessarily small or because the food has a high percentage of water. Strained spinach, for instance, contains only 1 to 1.4 mg. of iron per 100 gm. (Bridges.[9])

2. To be effective, food iron should be in soluble form. Some foods fairly high in total iron are low in soluble iron. (Summerfeldt.[10])

3. **Pablum is high both in total iron (30 mg. per 100 gm.) and soluble iron (7.8 mg. per 100 gm.) and can be fed in significant amounts without digestive upsets as early as the third month, before the initial store of iron in the liver is depleted. Pablum also forms an iron-valuable addition to the diet of pregnant and nursing mothers.**

Pablum (Mead's Cereal thoroughly cooked and dried) consists of wheatmeal, oatmeal, cornmeal, wheat embryo, brewers' yeast, alfalfa leaf, beef bone, iron salt and sodium chloride.

[1-10] Bibliography on request.

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