The Intravenous Diet

F. Gerard Dolan, B.Sc., M.D.

Senior Interne, Camp Hill Hospital.

THE problem of disease as an anatomical and pathological complex has been broadened to include the physiological and biochemical alterations associated with the pathological state. These problems of metabolic alterations are a part of every disease process and frequently form the picture of the disease as it is presented to the physician. These metabolic complexes are a dissociation of equilibrium necessary for normal function and they produce very definite clinical pictures which demand treatment, and, if neglected, may jeopardize life even though the original pathology may be slight, as is seen in cases of infantile diarrhoea. Sodium, potassium, water and protein metabolism have recently come into the forefront of medical research. Other mineral and enzyme systems have yet to reach the stage where therapy can be applied readily. However what is at present known should be therapeutically applied in every case, for a patient in normal metabolic equilibrium is in better condition to recover from a disease process than one who is in a profound state of inequilibrium.

The purpose of this discussion is to review the problems relating to abnormal biochemical alterations, particularly those of water, sodium, potassium, protein and acid-base balance. At present the focus of medical research falls into the realm of biochemistry, and particularly on the above mentioned substances which have been proven to be valuable indicators in disease.

Although abnormalities of metabolism usually implicate all or most of these, they will be discussed individually as if pure alterations had taken place, although in certain sections the findings will be incorporated.

To divide the discussion of mineral, fluid and caloric metabolism, the discussion will follow these headings:

(1) Water and salt.

(2) Acidosis and alkalosis.

(3) Potassium.

(4) Protein.

(5) Summary of therapy.

(1) Water and Salt Metabolism..

The commonly quoted requirement of water in an adult under standard conditions is 3000 ml./day. This is calculated on the expected loss of 1500 ml. of urine per day, and a loss of 1500 ml. through other channels. The minimal expenditure may be much less—200-900 ml. This depends upon the amount and kind of solutes to be removed from the body. Under such conditions an intake of about 1500 ml. would be more than adequate.

The internal fluid invironment is distributed thus:

(1) Blood stream..... 7000 ml.
(2) Interstitial Fluid..... 10000ml.
(3) Intracellular Fluid..... 35000ml.

The association between the first two compartments is very close and they exist in a state of equilibrium as far as water and mineral content are concerned, and changes in one will tend to bring about compensation from the other. Thus in hemorrhage the blood loss is in part water loss and this loss is partially restored by fluid from the interstitial space. The intracellular water is more closely bound and is given up much less readily. But in time there occurs a definite dehydration of this space also. The reverse process may also occur when there is a general increase in body fluid. Very frequently, the movement of fluid also involves the movement of ions not only as a result but also as a cause. There is a balanced exchange between the capillaries and the interstitial space. Normally this is an equilibrium adjusted to maintain the exchange of metabolities in body organs and to distribute body fluid according to need.

In the process of fluid balance the role of the kidney is all important, for it is the disposal depot for most substances leaving the body. The kidney is also unique in its power to assess the fluid picture of the body and its related electrolyte content, and its ability to deal with it in such a way as to preserve the vital equilibrium necessary for most efficient function. However the renal activity is merely the crucial regulation in a complex process integrated by the regulation of the cardiovascular system, the pituitary, the neural and humoral control of blood pressure and the capillary bed, and the adrenal and other endocrine glands.

The control of the excretion of sodium and chloride determines the volume of extracellular fluid. 85% of the glomerular filtrate is reabsorbed. The rate of this reabsorption can be augmented in proportion to the load, which means according to the rate of glomerular filtration and the plasma concentration. The rate of water reabsorption in the distal tubule is under the control of the anti-diuretic hormone of the pituitary. Increases in the electrolyte content in extracellular fluids and diminution of body water leads to a decrease in urine volume through the action of the anti-diuretic hormone. The salt retaining hormone of the adrenal cortex increases the rate of reabsorption of sodium presumably in the distal tubule. High venous pressures increase the rate of reabsorption of sodium in the absence of changes in the rate of glomerular filtration, however this change is often present. The retention of sodium in such conditions as heart failure, hemorrhage, shock, hypo-proteinemic states, exercise and assumption of the erect posture may involve one or more of the following factors:

(1) Reduction of renal blood flow.
(2) Reduction of the glomerular filtration rate.
(3) Increase in venous pressure.
(4) Increase in action of the hormones of the adrenal cortex on the kidneys and circulation.
(5) Changes in blood pressure.

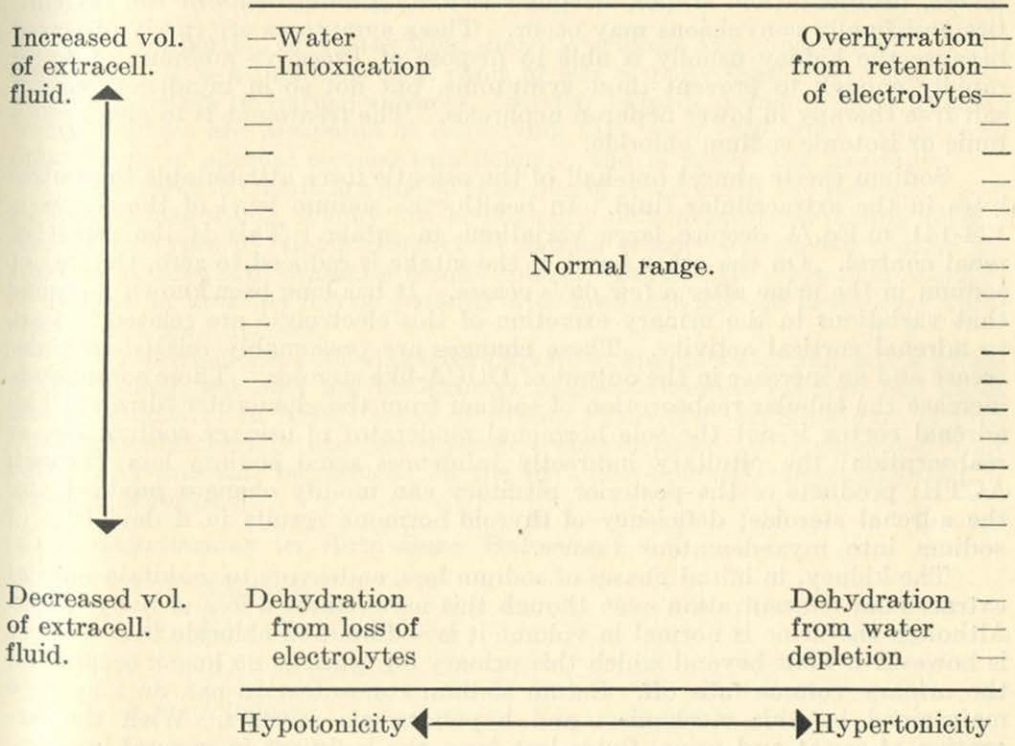
Water requirements vary greatly and extensive losses occur in a wide variety of conditions. In all such conditions the water contains sodium chloride and occasionally protein. A common example is the loss of gastro-intestinal secretions. The amount may reach several liters per day. A similar situation occurs in profuse sweating.

Large amounts of sodium chloride are lost when intestinal secretions are lost. These secretions are isotonic hence 5-9 gms. of sodium chloride are lost per liter loss. Similarly in profuse sweating sodium is lost but the concentration is much lower in sweat.

Disorders of water and salt balance may be simplified thus:

A. Dehydration.
   (1) Water deprivation.
   (2) Electrolyte loss.

B. Overhydration.
   (1) Retention of electrolyte.
   (2) Water intoxication.



In dehydration from water deprivation, there is hypertonic extracellular fluid as well as a diminished volume of this fluid. The symptoms are: thirst,

fever, dry skin, dry tongue, restlessness, feeble pulse, normal blood pressure and scanty urine of a concentrated type. The treatment is water.

In dehydration from electrolyte loss, there is a tendency toward low concentration of sodium and chloride. Symptoms are weakness, pallor, low blood pressure, absence of thirst and dilute urine that may be normal in volume. This syndrome is observed in many clinical states, including diabetic acidosis, Addisonian crisis, heat exhaustion, diarrhoea, vomiting and gastro-intestinal fistulas. In all of these there is a tendency to peripheral circulatory collapse. The treatment is saline.

Overhydration associated with a retention of sodium is characterized by a large volume of extracellular fluid and a normal or increased concentration of sodium. Edema is a frequent sign. This syndrome is seen in heart failure, nephritis and when an excessive amount of sodium chloride has been administered. In this type the treatment is salt restriction and fluids ad lib.

When overhydration is caused by the ingestion of very large amounts of water, the term water intoxication is applied. In this type there is usually not enough expansion of the extracellular compartment to produce edema that is visible, but there is definite hypotonicity. In mild degrees of water intoxication there is nervousness, salivation and vomiting. When more severe, disorientation, stupor, irregular twitching movements of the extremities and finally convulsions may occur. These symptoms are rarely observed because the kidney usually is able to dispose of excessive amounts of water rapidly enough to prevent these symptoms, but not so in injudicious use of salt free therapy in lower nephron nephrosis. The treatment is to give hypertonic or isotonic sodium chloride.

Sodium exerts almost one-half of the osmotic force attributable to electrolytes in the extracellular fluid. In health, the sodium level of the serum is 134-141 m.Eq./1 despite large variations in intake. This is the result of renal control. On the other hand, if the intake is reduced to zero, the loss of sodium in the urine after a few days ceases. It has long been known of course that variations in the urinary excretion of this electrolyte are related in part to adrenal cortical activity. These changes are presumably related to a decrease and an increase in the output of DOCA-like steroids. These compounds increase the tubular reabsorption of sodium from the glomerular filtrate. The adrenal cortex is not the sole hormonal moderator of urinary sodium loss or reabsorption; the pituitary indirectly influences renal sodium loss through ACTH: products of the posterior pituitary can modify changes produced by the adrenal steroids; deficiency of thyroid hormone results in a deviation of sodium into myxedematous tissues.

The kidney, in initial phases of sodium loss, endeavors to maintain normal extracellular concentration even though this necessitates a loss of body water. Although the urine is normal in volume it is sodium and chloride free. There is however a limit beyond which this urinary adjustment no longer occurs and the urinary volume falls off. Serum sodium concentration can no longer be maintained by this mechanism and hyponatremia appears. With the exception of sweat and urine, fluids lost from the body are in general isotonic; this per se should not produce hypotonicitv. Therefore its presence points either to:

(1) Loss of sodium without water i.e. through the use of low sodium fluids in gastric and intestinal lavage, peritoneal irrigations or in an artificial kidney.

(2) Losses of sodium with water followed by replacement of water alone. With either type, decreases in the bicarbonate content of the plasma or actual acidosis may be observed depending on the magnitude of the chloride loss relative to the sodium deficit.

What is the importance of the hypotonicity and of the decrease in the total amount of the extracellular sodium in the circulatory collapse which follows depletion of sodium? The former derangement can be readily corrected in the experimental situation by inducing diuresis in which considerable amounts of water but very little salt is lost. This occurs after the intravenous injection of urea. In this way the serum levels can be restored to normal, but the total amount of sodium is not replaced. So in sodium therapy you must consider:

(a) the concentration of sodium.

(b) the volume of extracellular fluid.

(c) the total amount of extracellular sodium.

A certain proportion of deaths from diabetic acidosis and coma are due to salt depletion. It is a factor in fatal cases of loss of body fluids, as in diarrhoea, vomiting, fistula or burned surfaces. Similar hazards occur in those patients whose kidneys are incapable of conserving salt, as in Addison's disease and other forms of adrenal cortical insufficiency, and in some renal disorders.

Most cases of edema are due to retention of sodium. If the increment is of superficial magnitude, it may not be clinically demonstrable as pitting edema. Almost all types of edema respond to sodium restriction. When sodium restriction is adequate dramatic results follow. Further, the administration of 30-40 gms. of urea per day in patients with normal NPN will prove very valuable in inducing diuresis.

The ability of the post-operative patient to handle water and salt is greatly diminished, not only are his kidneys unable to excrete sodium, but water also is retained. In Ariel's series of twenty patients given 3,500 ml./24 hrs., three patients actually had post-operative convulsions. It is suggested that both water and salt be reduced on the first post-operative days until normal function is regained.

(2) Disturbances in Acid-Base Balance.

A. Alkalosis.

(1) Metabolic

(2) Respiratory.

B. Alkalosis.

(1) Metabolic

(2) Respiratory

Condition	PH	CO_2 Comb. Power.
Metabolic Alkalosis.	Increased	Increased.
Respiratory Alkalosis.	Increased.	Decreased.
Metabolic Acidosis.	Decreased.	Decreased.
Respiratory Acidosis.	Decreased	Increased.

In most varieties of acidosis that are encountered, such as diabetic acidosis, the CO_2 combining power of plasma is decreased and of course the pH tends to decrease also. A less familiar variety of acidosis is seen whenever there are pulmonary factors, such as emphysema, which prevents the free excretion of CO_2, thereby increased acidity of body fluids occurs. In the presence of excess CO_2 there is an increased CO_2 combing power in the plasma.

In ordinary varieties of alkalosis, i.e. that seen in pyloric obstruction, the CO_2 combining power increases, but when there is an accelerated loss of CO_2 such as occurs in chronic hyperventilation arising from encephalitis or salicylate intoxication, the alkalosis is associated with a decreased CO_4 combining power.

The most important organs associated with the control acidbase balance are the lungs and the kidneys. The lungs act through their ability to blow off CO2, an acid radicle or by reduction of the rate and depth of respirations in order to conserve the acid product.

The role of the kidney is of fundamental importance and it will be discussed at some length as a summary of Pitts recent work.

NaC1 and $NaHCO_3$ are the major salts of the blood plasma and interstitial fluid of man. These fluids are alkaline. The degree of alkalinity of all is tempered or buffered by a relatively fixed concentration of carbonic acid (dissolved CO_2). The reaction of the blood plasma and the interstitial fluid of the body must of necessity be actively and continuously stabilized by respiratory and renal homeostatic mechanisms which preserve respectively, constancy of carbonic acid concentration and constancy of bicarbonate concentration. Were either to fail, alterations of cell permeability and ionic composition would occur so as to alter vital enzyme systems.

The body tends to stabilize base 25-27 m. Eq./L above the sum of all fixed non-volitile anions. The kidneys have a dual problem; the first is to salvage large quantities of bicarbonate which enters the glomerular filtrate per day. About 5,000 m. Eq. of bicarbonate enters the tubule but usually only 1-2 m. Eq. are lost daily. Excessive bicarbonate, as in an alkaline ash diet, is excreted however. The second renal problem arises from the fact that non-volitile acids, i.e. H_2SO_4 and H_3PO_4 are formed in considerable quantities in the metabolism of phospholipids and proteins. These acids tend to deplete body bicarbonate which neutralizes them. Two renal mechanisms are called into play: 1) The excretion of titratable acids and 2) the excretion of ammonia. In excreting titratable acids, neutral salts enter the tubule and are converted into acid salts or free acids. The alkaline portion is then reabsorbed into the blood stream.

In Blood:

H_3PO_2	$2NaHCO_3$	—	$Na_2HPO_2 + 2H_2CO_3$
Acid Metabolite	Buffer System		Neutral Salt Weak Acid.

In Kidney:

$$Na_2HPO_2 + H_2CO_3 = M\ NaH_4PO_2\ (\text{Excreted})\ M\ NaHCO_3\ (\text{Absorbed})$$

The reaction is driven to the right because sodium bicarbonate is continually absorbed. The capacity of the kidney to perform this operation is somewhat limited. Thus the kidney can salvage one of the two sodium ions. To salvage the second the kidney would require the ability to produce urine at a pH of 2.5. Actually the best it can do is 4.5. For this reason it can excrete only negligable quantities of strong acids. These strong acids must be eliminated fully neutralized but the kidney accomplishes this without sacrifice of sodium ion by replacing them with ammonium ions.

$$Na_2SO_4 + (N_2H)_2CO_3 = (NH_2)_2SO_4 + NaHCO_3$$

Under ordinary conditions, the kidney balances its acid base budget more by the latter than by the former mechanism.

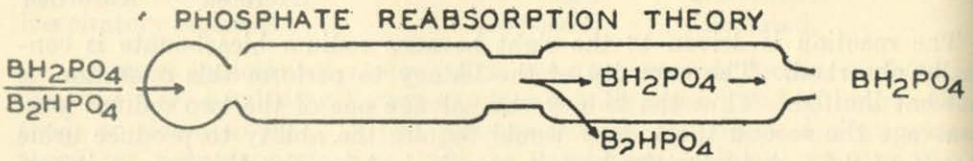
A normal individual on an average diet excretes the equivalent of 300 to 500 mls. of 0.1 N acid/ day combined with ammonia and 100 to 300 mls. as titratable acid. The uncontrolled diabetic produces much more metabolic acid not only from protein and lipid metabolism but also by hydroxybuteric acid and acetoacetic acid. In this case the equivalent of 3000 to 5000 mls. of 0.1N acid is excreted with ammonia while 760 to 5000 mls are excreted as titratable acid and even then equilibrium is never quite reached.

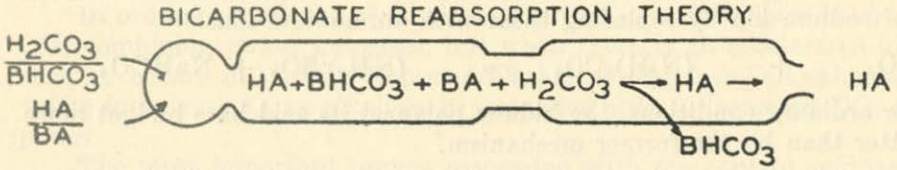
In nephritis, the production of metabolic acid is within normal limits, or even reduced if the patient is on a low protein low fat diet. However, the renal capacity to eliminate acid without loss of base is limited and the more severe the renal disease the greater is this incapacity.

The most alkaline a urine the kidney can produce has a pH of about 8.0. This is found when the patient has ingested soda bicarbonate. It has been found that the renal threshold for bicarbonate is 25-27 m.Eq./1. This threshold is fairly stable, however, these reservations must be made. In hyperventilation, the threshold is reduced; why this is so is unknown, for both the cause and effect tend to lower plasma bicarbonate and pH. In perniceous vomiting or prolonged gastric drainage or following ingestion of large amounts of soda bicarbonate, the concentration of chloride in the body fluids is reduced and that of bicarbonate is proportionately elevated. So long as chloride is reduced the kidney will conserve bicarbonate.

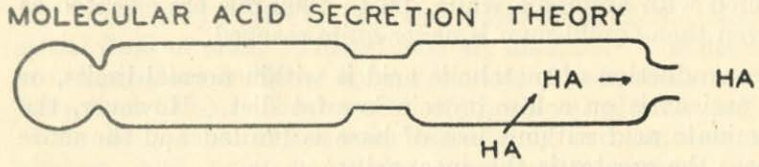
Plasma enters the kidney at a certain pH. It passes through the glomerulus and the proximal loop at the same pH. In the distal segment of the renal tubule acidification takes place. To do this the tubule could absorb base or excrete acid but it could also do both. Only two buffer mixtures enter the glomerular filtrate in significant quantities, namely monobasic and dibasic phosphate and carbonic acid and carbonate.

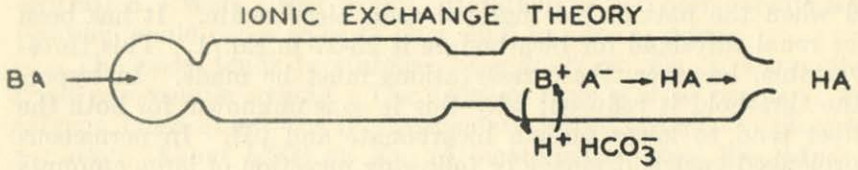
What happens then in the distal tubule? Here are a few theories:





These two offer theories if the acid is present in the glomerular filtrate. If not, then there are two more theories:





The excretion of ammonia is also a function of the distal tubule. Where does the ammonia come from? It was thought to be from urea but now glutamine is suspected.

The acidosis of diabetic ketosis is primarily a consequence of overloading of the circulation and the kidney is unable to handle the excess acid. On the other hand, the acidosis of chronic renal disease is primarily a consequence of the inability of the damaged renal tubules to exchange $H+$ and NH_4+ for base at a normal rate.

(To be continued)

COMMITTEE ON TRAUMA
AMERICAN COLLEGE OF SURGEONS

Management Of Acute Head Injuries

HEAD injuries may be classified as (A) open and (B) closed. Open injuries incur the risks of (1) hemorrhage and (2) wound infection. Closed injuries involve the risks associated with (1) immediate destruction of brain tissue (contusion, laceration, etc.) and (2) progressive damage secondary to (a) cerebral compression due to intracranial hemorrhage or edema, and (b) anoxia resulting from an inadequate airway.

For clarity, the treatment of open and closed wounds will be considered separately in this bulletin, but it is to be emphasized that both often are seen in the same patient and require consideration jointly.

Treatment Of Open Wounds

The proper treatment of *lacerations of the scalp* consists of thorough cleansing, débridement and snug closure. As a first-aid measure, a compression bandage should be applied to control bleeding. Externally protruding foreign bodies should not be manipulated. When the patient's general condition permits and adequate equipment is available, the following procedure should be undertaken:

(1) Shave scalp widely about the wound.

(2) Cleanse wound with soap and water and irrigate thoroughly with copious amounts of solution (saline or water).

(3) Infiltrate margins of scalp wounds with procaine.

(4) Remove dirt and excise devitalized portion of scalp.

(5) Explore skull for fracture.

(6) If no fracture, close wound snugly in one or two layers with interrupted sutures of nonabsorbable material.

(7) Give antitetanic serum or tetanus toxoid.

Simple lacerations may be closed in the Emergency Room by a single operator, but it is more satisfactory if the operator has an assistant to compress the margins of the wound to control bleeding.

Where the *laceration overlies a fracture*, surgical correction should be undertaken only in the Operating Room, for it may be necessary to remove dirt, hair, or debris from the fracture line or to remove bone fragments. One should be prepared to deal with bleeding from the dura and brain.

If the *brain is lacerated* or *foreign bodies are retained* within the brain substance, all macerated brain tissue should be removed, the foreign bodies removed if possible, particularly if they are nonmetallic, all bleeding controlled and the dura and scalp closed snugly. Surgical treatment of these wounds requires experience in brain surgery and special equipment (suction,

lighting, electrocoagulation, etc.). With the use of chemotherapeutic and antibiotic agents, it is permissable to defer operative closure of wounds for many hours pending improvement in the patient's general condition and analysis of his neurologic status.

Leakage of cerebrospinal fluid from the nose or ear constitutes a special problem. Any cerebrospinal fluid fistula invites the risk of meningitis. Now, with an adequate screen of antibiotic and chemotherapeutic agents, one is justified in waiting ten to twelve days pending spontaneous closure. If the leak persists longer than this, surgical closure of the fistula should be considered. Some feel that lowering of the spinal fluid pressure to 90 mm. of water by spinal drainage every twelve hours promotes spontaneous closure. Fortunately most fistulas do close spontaneously.

Closed Wounds

One of the most frequent causes of death after head injury is progressive cerebral damage due to anoxia. Many factors contribute to this:

(1) The capacity of the contused brain to utilize oxygen is diminished.

(2) The cerebral blood flow is reduced, even though blood pressure may be elevated.

(3) The oxygen concentration of the blood is diminished as the result of a disturbed respiratory center, and obstruction of the respiratory tract (tongue, mucus, pneumonitis).

The treatment in such cases is the maintenance of an adequate airway and the administration of oxygen. Patients frequently improve remarkably after clearing the airway and administering oxygen. The most satisfactory way of giving oxygen is by nasal catheter. Oxygen tents and masks interfere with attempts to keep the airway patent. Constant nursing attention, with a suction apparatus for the removal of mucus, is necessary. This may be supplemented by postural drainage (head down on face). Tracheotomy is indicated in certain cases.

The *differential diagnosis* between surgical and nonsurgical lesions is of primary importance in the management of acute head injuries and usually depends upon the patient's course. Hence, a carefully taken history, particularly as relates to the onset and duration of unconsciousness, and frequent and repeated neurological examinations are necessary.

Intracranial clot such as extradural, subdural intracerebral hematoma, usually is incompatible with life and requires surgical removal. Most patients with these lesions show progressive stupor, convulsions, focal paralyses and disturbance of the vital signs, such as alteration in pulse, respirations, etc. If the patient has been conscious and then loses consciousness, an expanding intracranial lesion is probably present. The lucid interval may be prolonged (days or weeks) with subdural hematoma. If the patient has remained continuously unconscious from the moment of impact, the coma may be due in part to contusion of the brain and in part to an expanding lesion. In such

instances, it may be impossible to make a differential diagnosis clinically, and cranial exploration then is necessary if the patient's condition is growing worse.

The *surgical management* of these lesions is not within the scope of this paper.

Special Examinations and Treatments

(1) *X-rays.* X-rays of the skull rarely influence the course of treatment, yet the procedure of taking films involves considerable manipulation and wastes time, both of which are detimental to the ill patient. Hence, it is advisable that x-ray examination of the skull be deferred until such time as the patient is co-operative and his condition stabilized. Attention should always be given to the possibility of injury elsewhere, particularly to the cervical spine.

(2) **Position of Patient.** The position of election is with the head elevated, since this reduces venous congestion. The advantages of this position, however, are far outweighed by the disadvantages which might accrue from an obstructed airway, and if the patient's airway cannot be kept completely open with the head elevated, this position should be changed to one which best promotes an adequate airway.

(3) **Shock.** Surgical shock (peripheral vascular collapse) is rare in head injuries per se, but, when present, the usual measures (Trendelenburg position, intravenous fluids, heat, etc.) should be employed. Shock usually indicates associated injuries and demands primary consideration.

(4) **Fluid Balance.** In an effort to reduce or limit cerebral swelling after head trauma, certain writers have advocated rigid dehydration by withholding fluids, administration of hypertonic solutions intravenously, the use of magnesium sulphate, enemas, etc. Rigid dehydration does harm rather than good to an ill patient, as the other vital body functions are interfered with. Fifteen hundred to 2000 cc. daily are necessary to keep the patient in fluid balance.

It is necessary in this connection to keep close watch upon the protein metabolism. Coma, stupor and prolonged confusion often result from protein depletion and can be prevented or corrected by the administration of protein hydrolysates, plasma or whole blood. If coma is prolonged, gastric feedings by nasal catheter are indicated, using a high protein diet.

(5) **Spinal puncture.** Considerable controversy prevails concerning the indications for and the merit of spinal puncture in the diagnosis and treatment of head injuries. Some advocate daily spinal punctures as routine in the treatment of head injuries, but this is not generally accepted. The authors of this bulletin consider the indiscriminate use of spinal punctures dangerous. The following are considered proper indications for spinal puncture.

(A) Diagnostic—

(a) To determine pressure where intracranial clot is suspected.

(b) To determine the presence and/or degree of bleeding.

(B) Therapeutic—

(a) To lessen intracranial pressure by withdrawing fluid as a temporary expedient pending measures that provide more lasting control of increased intracranial tension, such as surgical evacuation of clot.

(b) Evacuation of bloody fluid when signs of meningisimus appear, usually four to eight days. Evacuation of fluid at this time usually relieves headache and speeds recovery.

Technique. When indicated, a spinal puncture should be done with the patient in the lateral recumbent position, using a standard spinal puncture needle.

The operator should make the following determinations: color of the fluid, initial pressure, final pressure and the amount withdrawn.

Jugular compression tests should not be carried out unless one suspects injury to the spinal column. These tests give no information of value with reference to the brain, and the sudden rise in spinal fluid pressure which follows jugular compression may be harmful after head injury.

Spinal puncture should not be attempted if the patient is unco-operative for the information obtained is unreliable, and struggling against resistance may be harmful.

(6) **Control of Restlessness.** Patients who are restless and confused constitute a difficult nursing problem. and sedative medication may be necessary. Paraldehyde administered rectally or barbiturates—sodium amytal, sodium luminal—intramuscularly or intravenously are satisfactory. The latter are particularly indicated for the control of convulsions. Morphine and codeine are contraindicated because of depression of respirations, edema of the larynx and alteration of pupils (diagnostic).

(7) **Convalescence.** Early ambulation is recommended after head injury. The patient should receive physical therapy in the form of active and passive exercises early and should be gotten out of bed into a chair as soon as he is able and willing to co-operate. Prolonged bedrest is conducive to traumatic neurosis.

Lacerations of the brain, such as are asscoiated with depressed fractures and penetrating wounds, are frequently followed by a convulsive disorder. These patients should be given anticonvulsive medicaion for a period of several months after injury. The drug of choice is Dilantin Sodium gr. 1½ three times daily.

Many patients complain of residual headache, particularly in the posterior part of the head, and of dizziness. These symptoms generally are classified as posttraumatic neuroses. Recent observations indicate that they may result from trauma to the cervical roots and at times are relieved by cervical traction.

Note: The foregoing is one of several articles distributed by the Commitee on Trauma, American College of Surgeons, through its Regional Committees.

Twenty-Six Hospitals in Province "Approved"

Twenty-six Nova Scotia Hospital were included in the list of 3,352 institutions approved or provisionally approved by the American College of Surgeons in its 34th annual survey ending December 31, 1951.

The survey list in 1951 included 4,111 hospitals of 25 or more beds, of which 81.5% are approved. Of the 3,352 total approvals, 2,991 or 72.7% are fully approved and 361 or 8.7% are provisionally approved.

The approved hospitals in this province follow:

Amherst: Highland View Hospital, General, 76 beds, 12 bassinets. Antigonish: St. Martha's Hospital, General; 175 beds, 25 bassinets. Cornwallis: Royal Canadian Naval Hospital, General, 100 beds. Dartmouth: Nova Scotia Hospital, Mental, 450 beds. Glace Bay: Glace Bay General Hospital, General, 133 beds, 29 bassinets; St. Joseph's Hospital, General, 155 beds, 26 bassinets.

Halifax: Camp Hill Hospital, General, 600 beds. Children's Hospital, Children, 98 beds. Grace Maternity Hospital, Maternity, 67 beds, 67 bassinets. Halifax Infirmary, General, 228 beds, 60 bassinets. Halifax Tuberculosis Hospital, Tuberculosis, 120 beds. Victoria General Hospital, General, 430 beds. Royal Canadian Naval Hospital, General, 255 beds.

Inverness: St. Mary's Hospital, General, 44 beds, 10 bassinets.

Kentville: Blanchard-Fraser Memorial Hospital, General, 50 beds, 11 bassinets. Nova Scotia Sanatorium, Tuberculosis, 400 beds.

Liverpool: Queens General Hospital, General, 40 beds, 10 bassinets.

New Glasgow: Aberdeen Hospital, General, 144 beds, 35 bassinets.

New Waterford: New Waterford General Hospital, General, 70 beds, 10 bassinets.

North Sydney: Hamilton Memorial Hospital, General, 58 beds, 20 bassinets.

Sydney: City of Sydney Hospital, General, 163 beds, 24 bassinets. Point Edward Hospital, Tuberculosis, 178 beds, 2 bassinets.

Sydney Mines: Harbour View Hospital, General, 49 beds, 2 bassinets.

Truro: Colchester County Hospital, General, 105 beds, 21 bassinets.

Windsor: Payzant Memorial Hospital, General, 82 beds, 18 bassinets.

Wolfville: Eastern Kings Memorial Hospital, General, 29 beds, 8 bassinets.

Personal Interest Notes

Doctor C. Miller Ballem, Montreal, M.D.,C.M., Dal. 1945, M.Sc., McGill, received his certificate in Internal Medicine at the recent examinations in Montreal and Toronto.

Doctor Donald F. Sutherland, New Glasgow, M.D.,C.M., Dal. 1943, received his certification in Obstetrics and Gynaecology. He is practising his specialty in Saint John, N. B.

Doctor and Mrs. H. E. Wilson have left Ottawa for Vancouver where they will take up residence. Before going to Ottawa, Doctor Wilson practised at Ship Harbour.

Doctor Fraser Nicholson, Assistant Professor of Psychiatry, at Dalhousie University and Consultant Psychiatrist at the Nova Scotia Hospital in Dartmouth, conducted a two-day institute for parents and teachers at the Yarmouth Memorial High School the latter part of February.

Doctor William N. Watt, B.Sc., M.B., Ch.B., St. Andrew's, has opened a practice at Spryfield in Halifax County.

Doctor A. J. Myrden, Dal. 1950, of Halifax, has been appointed a Research Fellow in Surgery at the Harvard Medical School and assistant in surgery at the Peter Bent Brigham Hospital, Boston, and will take up his new duties in September.

Dr, J. Avery Vaughan, Dal. 1947, now practising in Windsor, recently passed the examinations of the Royal College of Physicians and Surgeons of Canada for certification in the specialty of general surgery.

The Bulletin extends congratulations to Doctor and Mrs. F. L. Akin of Windsor on the birth of a daughter, Jean Lynne, on January 25th; to Doctor and Mrs. N. A. Morrison of Musquodoboit Harbour on the birth of a daughter, on March 18th; and to Doctor and Mrs. A. J. MacLeod (Catherine MacDonald) of Moser River on the birth of a son, Alan David, on April 2nd.

Doctor A. L. Murphy of Halifax attended a meeting of the Board of Governors of the American College of Surgeons in Quebec in February, and addressed a clinical session of the college.

Doctor G. McK. Saunders, Dal. 1947, of Stellarton, was recently named Fellow of the Royal College of Physicians and Surgeons of Canada, following successful examinations. Doctor Saunders has been appointed surgeon to the Sackville Medical Centre at Sackville, N. B.

Doctor E. M. Curtis, Dal. 1932, of Truro, radiologist at the Colchester County Hospital in Truro, was successful in receiving his certification by recent examinations held in Toronto by the Royal College of Physicians and Surgeons of Canada.

Doctor J. K. Beer, Dal. 1941, and Doctor J. B. Downing, Dal. 1943, both practising in Summerside, P. E. I., have been awarded certificates as

specialists by the Royal College of Physicians and Surgeons of Canada, Doctor Beer in general surgery and Doctor Downing in internal medicine.

Approximately thirty doctors from Western Nova Scotia met at the Grand Hotel in Yarmouth in February for a series of lectures sponsored by the post-graduate committee of the Faculty of Medicine of Dalhousie University. The meeting was under the chairmanship of Doctor W. C. O'Brien, President of the Western Nova Scotia Medical Society. Doctor W. I. Morse, of Halifax gave an illustrated lecture on the modern treatment of the anaemias. Doctor R. W. Reed of Halifax conducted the evening round-table discussion on Penicillin and related drugs.

Doctor and Mrs. P. S. Cochrane of Wolfville were in Montreal in March where the former attended the 75th anniversary of the Grand Chapter of the Royal Arch Masons of Quebec.

Society Meetings

The Valley Medical Society held its monthly meeting at the District Community Centre in Annapolis on February 20th. Doctor W. I. Morse of Halifax of the Department of Medicine of Dalhousie University and Doctor R. W. Reed of the Department of Bacteriology of Dalhousie University, gave interesting and instructive papers on "Recent Advances in the Diagnosis and Management of the Anaemias" and "Review of the Present Status of the Antibiotics." Prior to the meeting the Society was entertained at dinner by the Order of Good Cheer.

At a meeting of the Pictou County Medical Society held at New Glasgow on February 24th two members of the post-graduate committee of the Faculty of Medicine, Dalhousie University, gave interesting papers to the members. Doctor R. M. MacDonald spoke on "Functional Bowel Disorders" and Doctor R. L. Aikens on "Chronic Pulmonary Diseases."

BOOK EARLY

The Annual Meeting of the Nova Scotia Division will be held at the Lakeside Inn at Yarmouth Sept. 3rd, 4th, 5th, 6th. Accommodation at the Inn is limited so members should make **RESERVATIONS EARLY** through

Dr. D. F. Macdonald,

Yarmouth, N. S.

Letters requesting reservations should state clearly arrival and departure date and time.

Obituary

Dr. Anthony Ivan Mader

The last of a notable group of Halifax medical men, Doctor A. I. Mader, died at the Victoria General Hospital, on April 5 at the advanced age of ninety.

He was born in New Canada, Lunenburg County, Nova Scotia in 1862. After preliminary education in Lunenburg and Halifax he spent two years as a student in the Halifax Medical College. Following an interval of employment in the New England States he entered McGill University where he was graduated in Medicine in 1891. He at once returned to Nova Scotia and was appointed to the staff of the Victoria General Hospital and as an instructor on the staff of the Halifax Medical College, both of which he continued to serve until 1910. In 1909 he was made a Fellow of the Royal College of Surgeons of Edinburgh.

About this time Doctor Mader established a private hospital on Coburg Road, Halifax, which he conducted successfully for many years. He was one of the pioneers in the use of radium in the treatment of cancer in Nova Scotia. Some twenty years ago the hospital was transferred to the Sisters of Charity who conducted a maternity unit there until the Halifax Infirmary was completed on Queen Street.

During his later years Doctor Mader carried on private practice until ill health and extreme age limited his activities. He was a skilful and enterprising practitioner and for many years occupied a prominent place in the Halifax medical profession. His death marks the passing of the last member of the teaching faculty of the Halifax Medical College which in affiliation with Dalhousie University did all the medical teaching in the Atlantic Provinces of Canada from 1873 to 1911.

In 1899 Doctor Mader married Eva Anderson Waddell of Halifax, who predeceased him. His three cildren are all members of the medical profession. Doctor Eva Mader MacDonald of Toronto, Doctor Ivan Mader of Hackensack, New Jersey, and Doctor Victor O. Mader of Halifax. One brother, Victor, is living in the United States.

Funeral services were held on April 8, with interment in Fairview Cemetery.

The Bulletin extends sympathy toDoctor C. M.Harlow of Halifax on the death of his father, Dr. L. C. Harlow, one of Canada's foremost authorities on the chemical treatment of farm soil, which occurred in Truro, after a lingering illness on January 16th, at the age of seventy-nine. Also to Doctor E. T. Granville of Halifax and Doctor F. J. Granville of Stellarton on the death of their father, Mr. James M. Granville, retired commercial traveller and crier at the Halifax County Court, on March 24th after a brief illness, at the age of eighty-eight.