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MEDICO, SURGICAL, SUBMISSIONS.

VOL-VI



NO. 10

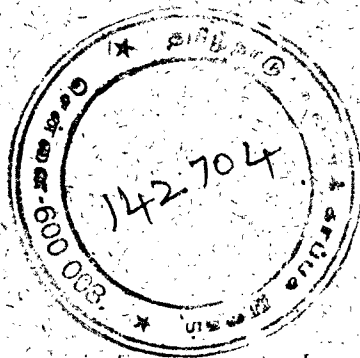
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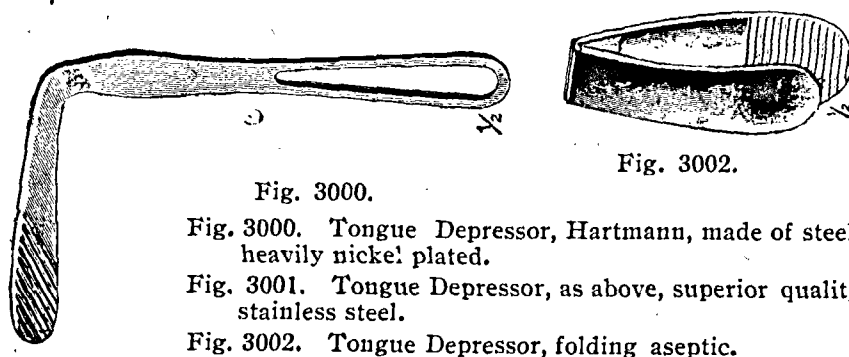


Fig. 3000.

Fig. 3000. Tongue Depressor, Hartmann, made of steel, heavily nickel plated.

Fig. 3001. Tongue Depressor, as above, superior quality stainless steel.

Fig. 3002. Tongue Depressor, folding aseptic.

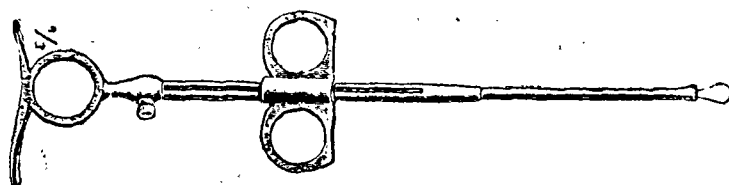


Fig. 3003.

Fig. 3003. Tonsil Snare, Eve's, heavy pattern, simple slide action.

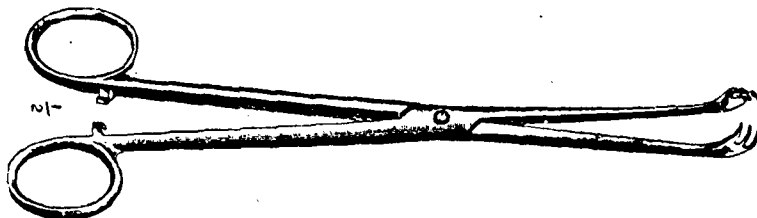


Fig. 3004.

Fig. 3004. Tonsil Vulsellum Forceps with side jaws and catch heavily nickel plated.

Fig. 3005. Tonsil Vulsellum Forceps as above. Stainless Steel.

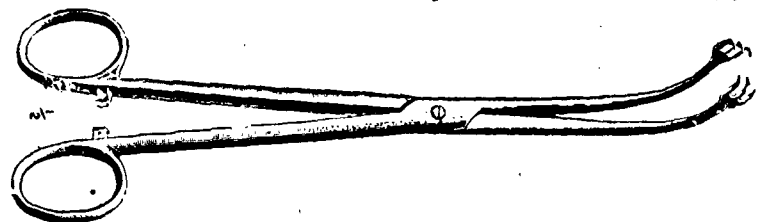


Fig. 3006.

Fig. 3006. Tonsil Vulsellum Forceps, curved on flat, steel nickel plated.

Fig. 3007. Tonsil Vulsellum Forceps, same as above. Stainless Steel.

Fig. 3008. Button Vulsellum Forceps as used in General Hospital. Steel Nickel plated.

Fig. 3009. Button Vulsellum Forceps, same as above. Stainless Steel.

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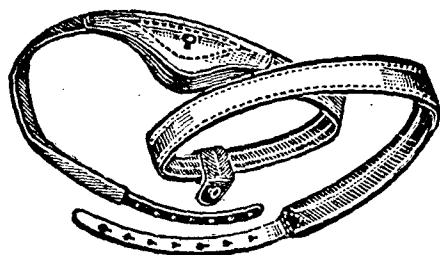
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MEDICO-SURGICAL SUGGESTIONS

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MODERN VIEWS OF NEPHRITIS.

BY DOUGLAS, J. THOMAS, M.D., D.M.R., M.R.C.P.,

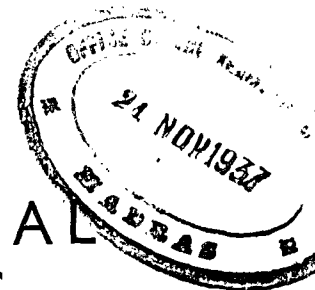
Hon: Physician to Out-patients, Royal Melbourne Hospital

In recent years we have seen the introduction of many classifications of nephritis. To many this has increased the confusion in a condition which is admittedly complex. I have no desire to add to the reader's difficulties, and for this reason I have tried to reduce the contentious points which arise in any discussion on nephritis to a minimum, and have avoided lengthy descriptions of microscopic anatomy and pathology.

Let us then in this article use a simple classification based on the acuteness or otherwise of the condition presenting. The terms acute, subacute, and chronic will be made to embrace all the types of nephritis described, although it will be necessary at certain points to define the relationship to former classifications.

We must briefly review the anatomy of the kidney. Essentially the kidney consists of *units*. Just as we use the term neuron in describing the structure of the central nervous system, so may we refer to the nephron—the fundamental functioning unit of the kidney. The nephron consists of a glomerulus with its associated tubule system and the blood vessels which serve both. It will be remembered that the greater part of the blood-supply of the tubule first passes through the corresponding glomerulus. The afferent branch of the interlobular artery which enters the glomerulus breaks up in the glomerulus and the efferent vessels from it are then (in the main) the afferent vessels to the corresponding tubules. When we visualize the possible routes taken by organisms (or clumps of organisms) and toxins from the blood stream into the kidney, the importance of this anatomical fact becomes very great.

The kidney then is composed of units or nephrons, of which there are possibly two million in each kidney. Probably all of them are not working at once. While some function, others rest, and it is likely that the contraction of the afferent artery determines to a large degree which units work and which rest. The fluid which is secreted by the glomeruli by a process of selective filtration contains



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sugar and sodium chloride, and has been described as "plasma without proteins." The membrane of Bowman's capsule is very fine, and its permeability is easily disturbed, so that albumin may escape from the blood stream. This capsular albumin may be added to by the detritus of degenerated tubular cells. It has been suggested that if a big protein molecule is traversing the capsule it may help to block the passage of smaller molecules. Possibly this is a factor in the causation of Na Cl retention. The first impact of toxins and organisms borne to the kidney by the blood-stream is upon the glomeruli, and it is here that the first mechanical obstruction to the passage of organisms is offered. Nephritis is never *exclusively* glomerular or tubular or interstitial, and I intend therefore to drop these terms in the basic classification employed, but reference to such terms as "glomerulonephritis" will be necessary.

ACUTE NEPHRITIS.

This may occur *during* an acute infective illness or *after* such illness has apparently terminated. If the nephritis develops during such an illness it is an acute focal nephritis. If it occurs after such illness it is an acute diffuse nephritis. In the former type we know that clumps of organisms become filtered out in the glomeruli, but in acute diffuse nephritis this probably does not occur. We therefore have been driven to admit that such acute diffuse nephritis may be toxic, or may even constitute an acute allergic reaction.

SUBACUTE NEPHRITIS.

Cases with onset as acute diffuse nephritis may drift on, as cases of subacute nephritis. But many cases of subacute nephritis begin in an *insidious* manner, and which seen first exhibit albuminuria, oedema, and *slight* rise in blood pressure. Let us now consider the clinical aspect of the three types of case already classified:— This is, acute focal nephritis, acute diffuse nephritis, and subacute nephritis (whether this is the later stage of acute nephritis or represents one with more insidious onset).

ACUTE FOCAL NEPHRITIS.

This is seen typically during some febrile illness, most commonly streptococcal in origin. If daily testing of the urine is not being done, attention is drawn to the presence of the condition by the change in colour of the urine, this suggesting the presence of blood. Examination of the urine then confirms the presence of albumin and blood, the latter in at least microscopic amount. These changes almost certainly mean that clumps of organisms have been caught in the glomeruli. The patient, however, exhibits no oedema, and the diagnosis is made on the urinary findings. If such a kidney is examined post-mortem, it is seen to be finely studded with haemorrhagic spots, but the greater number of the glomeruli are unaffected. This explains the absence of oedema. There is no rise of blood pressure. The prognosis is good, and the condition rapidly subsides.

Apart from the exhibition of free fluids as part of the general treatment of the toxic state accompanying the febrile illness, no special treatment directed to the kidney is necessary. Care is necessary in making the diagnosis. For example, a febrile case with haematuria and albuminuria may really be one of subacute bacterial endocarditis and not a passing infection with a passing focal nephritis. So that there is need for caution. But if the diagnosis is definitely that of focal nephritis occurring *during* febrile illness, the prognosis is good, and special renal treatment is unnecessary. But this is a very different state of affairs to that which is found in—

ACUTE DIFFUSE NEPHRITIS.

This is commonly seen *after* an acute infective illness, a period of ten days to three weeks having commonly elapsed before the nephritis develops. If daily examination of the urine is not being made, attention is drawn to the condition by the development of either oedema or haematuria. Examination of the urine will then show the presence of albuminuria, haematuria, and of casts. In focal nephritis there is no oedema, and no rise in blood pressure. In diffuse nephritis both occur. Probably the whole vascular system of the body is affected, and not merely the vessels in the kidney. The subject is generally young, the disease not often being seen after middle age. The patient is pale, and some anaemia rapidly appears. The change in colour of the patient is, however, due more to the accompanying vascular spasm than to anaemia. As the condition progresses, spasm may become less and the anaemia greater. The blood pressure will then drop to normal or near normal, but in any case we do not see very high manometer readings such as occur in cases of chronic nephritis. We should remember that even a continued rise to 150 mm. Hg. means (in a young adult confined to bed) a very serious circulatory disturbance.

Let us describe a typical case clinically. A schoolboy of sixteen comes home complaining of sore throat. He is found to have an acute tonsillitis, presumably caused by streptococci. He is put to bed, given fluids, aspirin, and possibly liquor hydrarg perchlor. His throat is very sore, and he is very miserable for three or four days. But then his temperature comes down, his throat clears up, and apart from some degree of debility he appears to have made a good recovery. He may even go back to school. Ten days or so later he is found, on waking one morning, to have oedema of the eyelids. Perhaps he goes to school that day. But when he gets home his ankles are slightly puffy. A hasty examination of the urine shows the presence of albumin and of at least a trace of blood. He is summarily returned to bed (if perchance he has been allowed up). He may have a headache and "stomach ache." Thereafter he runs a variable clinical course. Extremes are met from acute to very mild. The output of urine drops and the specific gravity rises. Except in very

severe cases there is no rise in the blood urea. *What is the prognosis?* Much more guarded than in a case of acute focal nephritis. He will probably recover completely. But while most recover completely others drift on into subacute nephritis, and a few die. Suppression of urine is a serious complication, but I have recently seen a case in which, after complete anuria for three days, the child made a complete recovery. It is well always in these cases to give a rather guarded prognosis. The daily output of urine must be measured, and the quantity of albumin estimated every two or three days. Such careful checking of the urine will warn you when the case is drifting on into one of subacute nephritis.

TREATMENT OF ACUTE DIFFUSE NEPHRITIS.

Put the patient to bed and keep him warm, nursing him with blankets next to his body. In this condition there is widespread vasoconstriction and surface temperatures must be carefully watched. The old idea (once fairly prevalent) of flushing the kidney by fluids has been utterly abandoned. *Restrict all intake (whether this be fluid or food)* to a minimum. The diet must be low in calories, and these be provided mainly from a carbohydrate source. This is necessary to rest, not only the kidneys, but the whole circulatory system. Dr. Horace Evans (to whom I am indebted personally for much help in this whole question of nephritis) begins this low diet treatment with a preliminary period of starvation. During this period no solid food is given. Fluid is given as orange-juice, one pint of this being taken in the twenty-four hours. The patient quickly learns to portion it out over the day and night. Generally speaking, three ounces are taken at a time. This period of "starvation" goes on for a week or ten days, at the end of which time the oedema and elevation of blood pressure have usually disappeared. If the blood remains elevated he frequently employs venesection, removing about 400 ccs. of blood in a child and 600 ccs. in an adult.

After return of blood pressure to normal, the diet is gradually increased by the addition of potatoes and vegetables (other than peas and beans), bread, butter, biscuits, custards, jellies, junket, tea with milk, and milk puddings. Later fish, rabbit, chicken, may be added. But the fluid intake is carefully restricted until all signs of oedema have disappeared. Salt in the food is kept to a minimum.

Anaemia should be treated actively. Evans feels that the presence of anaemia definitely delays healing. Half drachm doses of Ferri et Amon Cit may be given three times daily for weeks, or one of the iron pills may be given such as pill ferri (gr. 30 t.d.s.) Cardiac failure may occur, and immediate venesection should be undertaken if signs of cardiac distress appear. The bowels can be controlled by giving saline aperients. Marked suppression of urine is an exceedingly serious complication. No value attaches to giving large quantities of fluid by mouth when this complication exists, but a more liberal

allowance of fluid must be made than when no such complication has occurred. Intravenous injection of a pint of 5 per cent. glucose solution or of 100 ccs. of 15 per cent. saline may help. But the outlook is serious.

SUBACUTE NEPHRITIS.

As already stated, this may result from an attack of acute nephritis which has failed to be followed by healing, or may arise in a more insidious manner without pre-existing acute manifestations. Here the essential symptoms are albuminuria, elevation of blood pressure and some degree of oedema. Blood may be present in urine but commonly is absent. Attention should be drawn to the presence of rise in blood pressure. This is absent in nephrosis. I shall refer to the difference between subacute nephritis again. Most of us recognize subacute nephritis as one variety of our old friend, "the large pale kidney." The patient's main complaint is of the oedema. A pale, puffy countenance greets him every morning on waking. The matutinal shave reminds him (if he be an adult male) of his rather grotesque appearance. The urine shows the same changes as are found in acute diffuse nephritis, *but to a lesser degree*. The total output is reduced, the specific gravity is increased. But the quantity of protein in the urine may be very high (up to 35 grams per litre), and the blood protein may be correspondingly reduced. The clinical course varies greatly. The severe cases go rapidly downhill, and little or nothing can be done to help them. The less severe cases, in which haematuria and elevation of blood pressure are minimal, can be treated on the same lines as cases of *nephrosis*. Which statement leads us to the question, "What is nephrosis?" This question I can answer briefly by saying, "Nephrosis is a rare type of renal degeneration characterized by the *presence* of oedema and albuminuria, and by the *absence* of haematuria, hypertension, or increase of the blood urea. Blood cholesterol is generally raised, and the blood protein generally lowered. Some men deny the existence of nephrosis as an entity, and insist that these are merely special cases of subacute nephritis already described. But it is a fact that there are certain cases characterized by oedema and albuminuria that *do* run a special course. In these no failure of renal excretory function occurs. Retinitis, hypertension, and haematuria are never present. And complete recovery may occur.

The condition of nephrosis may appear at any age. Oedema represents a retention in the body of salt and water only, this being caused by the excessive loss of protein in the urine. The kidney is toxic, but the patient really is not. Once this fact is recognized the principles of treatment are easily grasped.

TREATMENT OF NEPHROSIS.

Restrict the intake of salt and water, reduce fats to a minimum, and give a liberal allowance of protein in the diet. A good feed of

steak will often produce a most copious diuresis. This shouldn't be hard for the patient to take. It really matters little in what form the protein is given. The fetish of white meats can be completely abandoned in these cases. The protein in the diet may be augmented by giving urea—say 15 grammes three times a day. I have used the mercurial diuretics in cases in which the oedema persists. My own preference is for salyrgan; 1 cc. or 2 ccs. may be given intramuscularly. My own experience has been that the addition of ammonium chloride is advantageous, but not really necessary. Still, in obstinate cases 90 grams of ammonium chloride may be given daily. There is no doubt that in some cases thyroid gland substance materially augments the action of the diuretics. Up to ten grams of thyroid extract may be given daily. It is surprising how well this comparatively large dose is tolerated.

The question of the treatment of septic foci crops up in all patients with nephritis. In cases of acute nephritis the removal (say) of tonsils may safely be left until convalescence is established. In cases of subacute nephritis and nephrosis I prefer to deal with this factor immediately. Tonsillectomy is done preferably under local anaesthesia. Nephrosis is on the whole a relatively malign condition. Many will disagree with this statement. The milder cases of subacute nephritis may be mistaken for nephrosis, and *vice-versa*. No case should be treated lightly, for we must remember that nephrosis is very often fatal. After the most amazing temporary recovery, the condition may recur in a much severer form, and the patient may die. But some of these cases recover completely, particularly if a recognizable causative septic focus has been adequately removed.

CHRONIC NEPHRITIS.

The term chronic nephritis embraces a wide group of cases, and covers the whole span of life from infancy to old age. But if we divide chronic nephritis into chronic parenchymatous and interstitial, we will find that the first type tends to occur before forty, and the second after this period.

I propose now to discuss chronic parenchymatous nephritis, and to reserve my remarks on chronic interstitial nephritis to the end of this paper, because of the necessity of reviewing certain types of arterial degeneration at the same time.

CHRONIC PARENCHYMATOUS NEPHRITIS.

Chronic parenchymatous nephritis occurs commonly before 40 years of age. I have often been asked, How can one distinguish between cases of subacute nephritis, nephrosis, and chronic parenchymatous nephritis? In all there is oedema and albuminuria, and the onset may be insidious in all three. Yes, but if the condition represents the late result of a former acute nephritis it may justifiably be called subacute nephritis.

The distinction between nephrosis and chronic parenchymatous nephritis is (to some at least) largely a matter of definition. But if in any case with oedema and albuminuria there is haematuria, elevation of blood pressure, or of the blood urea, the term nephrosis should not be used; the condition is a chronic nephritis. Admittedly the degree of chronicity varies widely. The outstanding clinical symptom in chronic parenchymatous nephritis is of course oedema. The urinary output is reduced, the specific gravity is raised. Haematuria may be present. Albumin and casts are present in the urine. The blood urea and blood pressure are often both increased. The prognosis is bad. My own experience has been that most of these cases die ultimately.

TREATMENT OF CHRONIC PARENCHYMATOUS NEPHRITIS.

Generally speaking, the treatment is the same as that already outlined under nephrosis. That is, restriction of salt and fluid intake, coupled with a high protein allowance, unless the blood urea is frankly raised. Protein, fat, and carbohydrate may be given in the ratio of 250-50-300 grammes per diem. I do not think that the renal lesion is adversely affected by such a dietary scale. Fluid should be restricted to somewhere about a litre and a half per day. In these cases, attempts to secure fluid elimination by routes other than the kidney are worth while. Obviously there are two main methods of attack. Purgation and sweating. This latter is best secured by the use of hot air baths, and the former by the use of salines. Mechanical means of relieving oedema (as, for example, Southey's tubes) I have abandoned. As in cases of nephrosis, urea may be given to reinforce the protein of the diet, in an attempt to decrease the oedema. Salyrgan may help some cases in which oedema is marked and persistent. But, ultimately, whatever treatment is employed, the condition generally proves fatal.

CHRONIC INTERSTITIAL NEPHRITIS.

I am afraid it is impossible to discuss chronic interstitial nephritis without defining certain degenerations of the arterial tree in which the kidney vessels are involved, but not primarily so. You will remember that arteries can be divided histologically into elastic and muscular. The aorta is an elastic artery. The cerebral vessels and the body arteries generally are muscular. If a condition of diffuse myo-hypertrophy exists throughout the muscular arteries (including the renal vessels), then the condition is really one of hyperpiesia with cardio-vascular hypertrophy as originally described by Allbutt. Such hyperpiesia is one cause of hyperpiesis. What then is the relation to atheroma? Simply this: Atheroma is not a cause of high blood pressure, but high blood pressure may prematurely induce atheroma. Atheroma is an essential part of Allbutt's "decreased arteriosclerosis." This arteriosclerosis is a generalized disease, but there is neither rise of blood pressure nor myo-hypertrophy

unless such diffuse myo-hypertrophy has been a factor in the *production* of such "decreascent arteriosclerosis." This arteriosclerosis may result in cerebral or coronary thrombosis or thrombosis in a renal vessel (with resulting ischaemia of the portion of kidney concerned) but it is not a cause of renal failure.

Looking more particularly at the renal vessels, we find then that they may show diffuse myo-hypertrophy (in which case we have present a high blood pressure but no impairment of renal function), or be the seat of decreascent arteriosclerosis with or without accompanying rise in blood pressure. The urine may in both cases show a trace of albumin. Neither of these conditions constitute chronic interstitial nephritis. This latter is our old friend, the red granular kidney.

Post mortem we find the kidneys to be small and red. (You will remember that in chronic parenchymatous nephritis they are large and pale.) The patient is over forty years of age. There is no oedema. The condition is often discovered in routine insurance examinations or because the patient comes for check-up complaining of headache, giddiness, or failure to concentrate when doing his work. Examination quickly discloses rise in the blood pressure, with accompanying cardiac enlargement. The urinary output is increased to 70 or 80 ounces per day. The patient has to get up at night. A mild degree of albuminuria is present. The urine is pale, and the specific gravity low. The patient is often breathless on exertion, the lungs may be emphysematous. Later on, as the condition progresses, oedema of the ankles may appear. Examination of the fundi will often disclose hypertensive retinitis. After a complete urinary and cardiovascular survey the diagnosis is rarely in doubt. And yet such a patient may, if he be fortunate, live on with a fair measure of health for years. In fact, this period of survival varies so greatly that it becomes very difficult to lay down general rules of conduct and treatment. The whole mode of life of the individual must be reviewed. If he be an industrial worker, his occupational stress and exposure must be carefully analyzed. The Wassermann reaction should be done.

TREATMENT OF CHRONIC INTERSTITIAL NEPHRITIS

If I were asked to give one general rule for the conduct of these cases, I would say, "Let the patient be moderate in all things." Let him exercise, eat, and drink with restraint. I believe that drugs have very little effect on the course of the disease. If heart failure should occur this will be treated on the normal orthodox lines.

In conclusion, let me say a few words concerning uraemia (the ultimate expression of renal failure), and the so-called pseudo-uraemia due to hypertensive cerebral attacks. The symptoms of these cerebral attacks has been described as those of "episodic, angio-spastic cerebral phenomena." The condition may closely

simulate true uraemia. In pseudo-uraemia, we may have a slight rise in blood urea; in true uraemia there is always severe nitrogenous retention. But such chemical examination may be the only means of distinguishing the two conditions.

Hypertensive attacks are usually ushered in with headache and vomiting. Coma and convulsions may occur, but the "hissing" type of respiration seen in true uraemia is absent. During these attacks the patient is given water and fruit juice only. The bowels are cleared by enema, and 4 ounces of a half saturated solution of Mag. Sulph. injected. Morphia gr. $\frac{1}{4}$ should be given if the headache is severe. Venesection should be undertaken in asthenic case. Lumbar puncture may help to relieve the headache. It is presumed that the pathological condition underlying these attacks is one of cerebral oedema with accompanying vascular spasm, resulting from the prolonged hypertension. Such attacks may often be distinguished from true uraemia by the rapidity of onset. Uraemia develops more slowly, and is less dramatic in its arrival. Whenever uraemia is suspected, the blood urea should be estimated. It will invariably be very high, the figure being 100 milligrammes per 100 ccs. of blood, or over. Here again the patient is given only water and fruit juice (or glucose). Mag. Sulph. solution should be given per rectum, as in pseudo-uraemia, and the patient lumbar-punctured. If there is vomiting, the fluid intake should be reinforced by the intravenous or rectal injection of glucose solution. A 10 per cent. solution is employed. Given rectally, a pint may be taken. But by the intravenous route the amount should not exceed half a litre. In both uraemia and pseudo-uraemia chloral hydrate may be used to control excitability. My own practice is to give 20 grains rectally when the patient weighs somewhere about ten stone, but 30 grains should be given in heavier patients.

This paper has been kept as brief as possible. I realize that I have only given a very limited account of the pathology of the various types of nephritis, and I have omitted many of the rarer and less important sub-varieties. I trust that brevity has not been attained at the expense of clarity, and that my attempt to give a concise review of the more common types of nephritis seen in general practice has not been wholly unsuccessful.

(*The General Practitioner April 15, 1937.*)

CESAREAN SECTION.

BY O. R. THOMPSON, M.D.

Macon.

In a study of obstetric literature one finds that the incidence of cesarean section in the United States varies from 1 to 10 to 1 in 500. The indications may be found to be anything from a contracted pelvis to the patient's desire for delivery by section rather than through the natural passage. The variation in incidence and indications show that there is either a great difference in opinion as to the indications for the operation, or a marked difference in the anatomic structure of the women in different sections of the country and in different clinics in the same community. The latter assumption is ridiculous and the former cannot be explained on the basis of a difference of opinion, but is due to the fact that the technic of the procedure has been developed so that there is a sense of security experienced by all parties which leads to the performance of entirely too many operations of this type.

The astounding mortality of mothers and babies revealed by an analysis of cesarean sections performed in different localities of this country should convince anyone that this is not an operation to be taken lightly, and that there is something indicated beyond surgical skill and the desire to operate—and that is sound obstetric judgment. Obstetrics is not an adjunct of general surgery and the time will never come when the best way for the birth of a baby is the abdominal route and not through the natural passage. The high mortality rate is largely due to the failure of the attending physician to have a thorough knowledge of his patient, and to know his own limitations. He must be able to recognize and handle the borderline cases in such a way that, when necessary, operative measures will be safe. A woman who is fatigued from long labor, has had many vaginal examinations, has fever, and upon whom attempts have been made to deliver the baby by other means, is not a good risk for cesarean operation.

The time of the operation in respect to labor is an important factor in the mortality rate. According to Miller: "When it is an elective procedure and when every circumstance is favourable, it has a minimum mortality of at least 2 per cent. In average hands—and these it must be remembered, are the hands that do most of our surgery—it carries a mortality of from 10 to 12 per cent. The death rate increases approximately 1 per cent with each hour of labor and each vaginal examination, especially after the membranes have ruptured; it increases 10 to 15 per cent with each attempt at delivery; and it reaches 50 per cent after attempted craniotomy. In plain words, this means that the mother's life is being placed in

jeopardy for the sake of a child whose chances are frankly rather more dubious than her own. For it must not be forgotten that the child's danger increases in like ratio with the mother's, and that the fetal and infant mortality, in these days of casual section, is quite as serious a consideration as the maternal death rate."

Much of the waste of human lives and health could be saved if pregnant women would submit to frequent periodic examinations by competent physicians. With careful supervision the physician in charge could plan and would not be forced into performing emergency operations. Cesarean operations are always attended with some risk, even though they are performed by the most experienced surgeons. After the operation has been performed and the result is satisfactory to all concerned, there remains a scar in the uterus that will remain a menace to future childbearing. This fact should be borne in mind before the decision is made to operate.

"The preservation of maternal life and health and of fetal life" constitutes the only justifiable indication for cesarean section. There is a general agreement as to the absolute indication, which is confined to a situation where there is an obstructing tumor or the pelvis is contracted so that a living or dead fetus cannot be delivered through the natural passage. In the group of relative indications, each operator must rely upon his own skill, judgment and experience, all guided by that intangible something we call "obstetric conscience."

Spontaneous delivery may be expected in 80 per cent of all cases of pelvic contraction. In each patient there should be a careful study of the pelvic measurements, the shape of the pelvic cavity, and the size, position, and presentation of the fetus before the onset of labor. A roentgenologic study, preferably a stereo, is of great importance in determining the exact proportion between the presenting part and the pelvic inlet. During the labor rigid asepsis must be maintained, vaginal examinations should not be made, and, if possible, the membranes should be held intact. The patient's strength should be maintained by forcing food and fluids; alkalies should be given to prevent the development of an acidosis; rest and sleep should be obtained by the use of morphine, scopolamin and one of the barbiturates. The progress of labor and the patient's strength should be observed and not the clock. A standard "test of labor" cannot be made applicable to all patients and I should like to emphasize the danger of allowing the patient to suffer too long and become exhausted before resorting to some operative procedure. The dehydrated, exhausted and infected patients give us the highest mortality rate following cesarean operation.

Cesarean section is rapidly becoming the method of choice in delivering patients of central and partial placenta previa. In those patients, primipara as well as multipara, with thick, rigid, and

undilatable cervixes, the method of delivery, in interest of both mother and baby, is the abdominal route. The delay and shock necessary to dilate the cervix and deliver is certainly a more serious ordeal for the mother and carries a higher death rate for the baby.

In patients who have premature separation of the placenta, in the primipara with an undilated cervix and in the multipara with an undilatable cervix, the procedure of choice is delivery by section. The child may be saved and the operator is able to control the uterine hemorrhage. If the hemorrhage is concealed and has been going on for some time, there may have developed an extravasation of blood in the uterine wall, destroying its contractile power, making a supravaginal hysterectomy necessary before the uterine hemorrhage can be controlled.

Eclampsia is not an indication for cesarean section. Toxemia of pregnancy and eclampsia are medical conditions and not surgical. With our present knowledge of the toxemias of pregnancy and the excellent results obtained with the conservative treatment, one is hardly justified in doing a section on an eclamptic patient unless the toxemia has resisted all treatment and the patient is rapidly growing worse. Should a section be necessary for obstetric reasons, the patient should be treated and prepared for the added strain of an operative delivery.

Patients with a moderately contracted pelvis and giving a history of one or more labors ending disastrously for the baby, should receive a most careful study. If there is any doubt as to what to do, labor should be induced at eight and one-half months or the patient allowed to go to term and sectioned immediately after the onset of labor.

Cesarean section is the preferred method of delivery in cases of stenosis of the cervix due to congenital abnormalities or the end results of previous operations, injury, or disease. I was compelled to perform a section on a patient with marked fibrosis of the cervix following the use of radium for the treatment of uterine bleeding. Sections are indicated in cases of carcinoma of the cervix or neoplasms involving the adnexa. Ovarian cysts large enough to cause complications during labor should be removed during the early months of pregnancy. Fibroid tumors may obstruct the birth canal but usually come out of the pelvis during the last months of pregnancy and do not as a rule complicate the delivery. Patients with ventral fixation of the uterus should be sectioned, and the operation should be seriously considered on those patients who have had extensive myomectomy or a successfully repaired third degree laceration. Patients showing disproportion and cervical dystocia in whom there is an arrest of the head at the pelvic inlet and the cervix undilated after a satisfactory test of labor, should be delivered by

section. Manually dilating the cervix and the use of forceps on a floating head or delivery by version usually means disaster to the baby and much damage to the mother. Medical complications, such as cardiac disease or tuberculosis, when associated with a contracted pelvis or other conditions which insure a difficult labor, may justify cesarean section. The decision must depend upon the judgment of the operator.

Authorities do not agree as to the necessity of repeating the cesarean operation in subsequent pregnancies. "Once a cesarean, always a cesarean," is a dictum that has been accepted by many of the profession. I do not subscribe to the dictum, because many women give birth safely to children in the natural way after a cesarean operation has been previously performed. The method of delivery in subsequent pregnancies should be determined by the indications for the previous section, type of operation performed and the postoperative convalescence.

A patient should not be subjected to the dangers of a cesarean section to deliver a dead fetus unless her pelvis is so contracted or deformed that an embryotomy cannot be performed without great damage to her tissues. Embryotomy is a loathsome operation, but its use in delivering some of the badly managed and neglected cases would certainly lower the high maternal mortality and probably not increase the infant mortality. The wider use of this operation would mean more living mothers, and, ultimately, other, better managed pregnancies for them. An added justification lies in the fact that in practically every average series of cesarean section the mortality is dual in 25 per cent of all fatalities.

There is an apparent tendency to exalt the child's life at the expense of the mother's. The ideal of obstetrics is to save two lives, but occasionally a decision has to be made, and in deciding the course I share the opinion of Miller: "If the woman is a primipara, her present child must be sacrificed to her future generative possibilities. If she is a multipara, with living children, her present child must be sacrificed to her existing responsibilities. And under no circumstances, be she primipara or multipara, should her life be jeopardized for the sake of a child whose chances, no matter for what reason, are in anyway dubious."

The best results following cesarean section will be obtained if the operation is performed on the estimated date of labor or a few hours after its onset, before the muscular efforts of labor have been sufficient to lower the vitality of the patient and before repeated vaginal examinations have given rise to uterine infection. Just before or immediately after the onset of labor, the mother is in the best possible condition for the operation. The increasing irritability of the uterus at the end of pregnancy will insure the proper contraction and retraction necessary for the control of the hemorrhage

during and after the operation. The size of the child should be carefully estimated by x-ray examination, and if it is unduly small, the date of operation should be deferred, to avoid the possibility of delivering a premature infant. When labor has begun, or if in the doubtful cases a longer or shorter test of labor is advisable, the operation should be performed as soon as the indication is recognized as positive. Rectal examinations should be used instead of vaginals, and the membranes, if possible, should be kept intact. Every hour of active labor renders the prognosis more serious and, therefore, no delay should be permitted after the need of the operation is recognized. The cesarean operation is never advisable after serious attempts at delivery from below have been made.

The obstetric surgeon should be familiar with the various operative procedures and choose the technic that will be safest for the individual patient. The classic section should be reserved for operations of election and clean cases when a rapid delivery is indicated. These include premature separation of the placenta, central and partial placenta previa, and repeat sections where classic section has been performed previously. I wish to recommend the low cervical operation as developed by Beck and DeLee as having many advantages over the other procedures, and its technic offer no difficulties to one trained in pelvic surgery. It has a lower mortality and morbidity rate and may be performed with safety after a relatively long test of labor. The advantages of low cervical section are: The incision is made in the thin, non-contractile part of the uterus, and when closed, is completely covered over by the bladder and peritoneum. Rupture of the scar during subsequent pregnancies is less likely to occur because the incision is in the lower segment, which is the resting, non-involuting part of the uterus during the puerperium, and which plays a late and passive part in the stretching incident to pregnancy and labor. The Porro operation should be limited to patients who have conditions that demand hysterectomy after delivery of the child. These include: multiple fibromyomas which cannot be removed by myomectomy; malignant disease of the uterus; premature separation of the placenta with marked destruction of the uterine musculature; the presence of incurable and extreme cicatrization of uterus and vagina, and uterine infection from contamination during labor. The range of usefulness of the vaginal section has become limited since the perfection of the abdominal cervical operation. The low cervical section is technically less difficult and offers almost as much protection against infection as the vaginal section. The vaginal section is recommended when one of the following conditions demand the rapid emptying of the uterus before the eighth month of pregnancy; (1) lesions of the heart, lungs, and kidneys; (2) toxemia of pregnancy in the presence of impending convulsions and in a patient who did not

improve under the conservative treatment; (3) pernicious vomiting of pregnancy in a patient who is dehydrated and would not stand long labor; (4) premature separation of the normally implanted placenta in its milder forms.

CONCLUSION.

Every case should be conducted as if cesarean section were contemplated; which means strict asepsis; strength of the patient maintained, and an early recognition of the necessity to operate in the interest of the mother's life and health and the life of the baby.

Since every justifiable obstetric operation represents at a failure on the part of nature, it behooves us to take due care that the results obtained following any obstetric operation do not reflect on our judgment and skill as physicians.

(The Journal of the Medical Assn. of Georgia.)

THE OCCURRENCE OF CARCINOMA IN CLINICALLY BENIGN PROSTATIC OBSTRUCTIONS

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Pueblo

For many years we have been taught and led to believe that prostatic hypertrophy and carcinoma of the prostate were entirely separate conditions. They were not supposed to exist together except in very rare instances, and it was generally believed that hypertrophy never degenerated into a malignancy.

In 1922 at the Pueblo Clinic we began the routine pathological examination of all prostatic tissue removed at operation. This included both the gross and microscopic examination. At first only a few sections of the gland were examined microscopically, but as our percentage of malignancies increased the examination became more detailed and now our pathologist makes numerous sections from all portions of the removed tissue. The results have been astounding and prompted this study. Only cases of apparently benign prostatic obstruction in which there was no clinical evidence of carcinoma, and which were operated either by suprapubic prostatectomy or cautery punch resection are included. Cases of carcinoma that were diagnosed or were suspected by examination, and cases of benign prostatic obstruction not operated are excluded.

Clinically, carcinoma of the prostate may be divided into two groups, those that don't give urinary symptoms and those that do. The first group is of high grade malignancy, and metastasize so early that the symptoms are usually caused in remote portions of the body

without any suspicion that the prostatic lesion is primary. Routine rectal examination reveals the prostatic involvement. Treatment is highly unsatisfactory. The cases are usually doomed and often all that can be done is to relieve their pain. The second group is usually of low-grade malignancy, may remain localized for a long time and tends to metastasize late or not at all. This group would also include the type of prostatic carcinoma that coexists with prostatic hypertrophy but is a separate and distinct lesion. Treatment here is much more satisfactory, and if the lesion can be entirely removed, cure may result; if not, the urinary obstruction may be relieved and the patient given a period of relative comfort. It is this second group that includes all the cases of our series.

Seventy-five cases have been analyzed. Of these, fifty-three (70.6 per cent) showed no evidence of any malignant change. Twenty-two (29.4 per cent) either showed definite evidence of malignancy at the time of operation (pathological examination) or later developed carcinoma in the prostatic area. This is a very high percentage of malignancies when the type of case under study is considered. It is generally conceded that about 20 per cent of all cases of prostatic obstruction are carcinomatous, but recent reports in the literature suggest the possibility of much greater incidence. Two excellent postmortem studies have recently appeared in which routine sections of prostates removed at autopsy were studied. The first by A. R. Rich comprised 292 cases of men over 50 years of age dying of all causes. Definite carcinoma was found in 14 per cent. There was a decided tendency for the early tumors to be situated laterally or posteriorly in the tissue near the capsule of the gland. Rich concludes that cancer may also arise within the newly formed tissue of a nodule of hypertrophy. The second, by R. A. Moore, was a study of 375 prostates of all male patients over 20 years of age autopsied during a period of one year, in Erdheim's Clinic in Vienna. Frank carcinoma was found in 16.7 per cent, although it was not recognized clinically in any case. He concluded from this study that carcinoma of the prostate is predominantly a lesion of the posterior lobe but that it may occur in any portion of the gland, that it is intimately associated with senile atrophy and rarely occurs in acini already hyperplastic. He feels that carcinoma and benign enlargement of the prostate are two separate diseases which occur independently. The work of Geraghty and Young seemed to establish the fact that a large number of prostatic carcinomata started in a single focus in the posterior lobe, and extended beyond the capsule between the fascia of the bladder base and the fascia of Denonvilliers. Randall and Ferguson in their anatomical studies dispute this point of origin and believe that the posterior lobe is not a common site of origin of carcinoma but that it may start in any of the lobes and that it may have multiple foci of origin. So the

argument as to the point of origin and the relationship of hypertrophy to carcinoma of the prostate is far from being settled.

Hess states in a recent article on genito-urinary malignancy that in his experience 25 per cent of the so-called simple adenoma of the prostate showed central evidence of malignant degeneration.

In analyzing our series of seventy-five cases, as to the operative procedure there were forty-six supra-pubic prostatectomies and twenty-nine transurethral resections. Of the twenty-two cases where evidence of malignancy was found, twenty were prostatectomies and two resections. This brings up the point as to the value of sections removed by resection, as to the presence or absence of carcinoma. Caulk believes that such sections will show a high percentage of carcinoma, if present, and he has had years of experience with resection. However, here is another point open to argument. There is no doubt that the study of the gland removed by prostatectomy is much more accurate in determining early malignancy than the study of sections removed by resection, many of which are markedly altered by the high frequency current or heat used in removing them. So of the twenty-seven cases resected and found benign only time will tell the later appearance of carcinoma, and the percentage may be still higher than that at present recorded.

Twenty of the twenty-two malignant cases were diagnosed either at operation or by the pathological examination immediately afterward. Two were not discovered by either of these methods but later appeared clinically in the patient at the site of operation. Both these cases were operated before multiple sections were taken, so undoubtedly the malignant area was overlooked. Nine cases showed well developed carcinoma of the adenocarcinoma type. Eleven cases showed early or very early malignant change.

Four of the twenty-two cases were diagnosed at operation due to extension of the lesion to the capsule, the others were found by the pathologist (sixteen) or appeared later clinically. Eighteen of the cases were relatively simple prostatectomies, the gland being easily and completely enucleated and no extension to the capsule or surrounding tissue being present. In fact in all these cases we felt that we had a simple hypertrophy until informed otherwise by the pathologist.

As to the origin of the carcinoma in the prostate, we have no accurate data, but we feel that no single lobe predominated. In the future a careful effort will be made in all cases to locate accurately the origin of the lesion in the gland.

As to the results in the series: There were three operative deaths, giving a mortality of 4 per cent—two from postoperative embolism and one from surgical shock and uremia. Subsequent examinations have been attempted in all cases, and check-up

examinations in those cases that showed malignancy at regular intervals. Either the patient or some close member of his family was informed of the pathological findings, and the need for follow-up examination emphasized. We have had good co-operation from most of the patients, of the fifty-three benign cases there have been eight deaths as far as we can determine. In none of them was carcinoma a factor. In the group of nine cases showing well developed carcinoma there have been six deaths. Of these one was an operative death from embolism, another from pneumonia four years after operation, having shown no evidence of recurrence of the malignancy, and the other four from carcinoma and metastasis. The period between operation and death in the four cases was as follows: (a) one year and six months, (b) three years, (c) four years, (d) five years. Three cases are living and well, one ten years after operation, another seven years and the third three years. They have all been carefully followed up and no evidence of recurrence has been found or reported. The results in this group show a high cancer mortality, as would be expected. However, the fact that five of this group went four years or more, relieved of their urinary symptoms and comfortable, is encouraging.

In the group of eleven cases showing very early malignant change, the results are much better. There are nine cases living and well, and two deaths. Of the cases living, one has gone twelve years; one ten years; one eight years; one six years; one four years; two two years; and two one year. All of these have been followed up and show no recurrence. One case died four years after operation, of nephritis; the other six months later of uremia. In none of this group so far has carcinoma recurred. All of these patients had suprapubic prostatectomies. The two cases that were not found by the pathological examination both developed local recurrences and metastasis, one four years after operation and lived another year before death. The other metastasis developed six years after operation; the patient lived two more years before his death.

CONCLUSION.

1. Carcinoma of the prostate was found in a high percentage of the cases of apparently clinically benign prostatic obstruction studied (29.4 per cent).

2. The earlier the malignant lesion is discovered the better the end results. Those cases that showed well developed carcinoma gave a high cancer mortality following operation; those that showed a very early involvement have so far given very good results.

3. A careful pathological examination is very important in all removed prostatic tissue. Numerous microscopic sections should be taken from every portion of the gland.

4. Prostatectomy holds an advantage over trans-urethral resection in removing early malignant involvement and giving a chance for ultimate cure instead of simple relief from obstruction.

(Colorado Medicine, April 1937).

POINTS IN THE DIAGNOSIS OF CANCER

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Durant.

Definition and General Signs. Before attempting to diagnose cancer, it is well to call to mind a working definition of the disease.

Cancer begins as a single body cell or group of cells, which for some unknown reason begins to grow independently of the other body tissues, and serves no useful purpose.

Before beginning a detailed discussion of the diagnosis of cancer in certain of the commoner locations of the body, a few general signs are given below, that are more or less common to all:

1. Loss of weight.
2. Temperature.
3. Leukocytosis.
4. Bleeding from any body cavity.
5. Pain, usually a late symptom.
6. Cachexia, a late sign.

Any of the above signs may be associated with many other diseases, but we should be cancer minded and suspect every one when these signs occur in our patients. Remember also, that a small percentage of cancer patients exhibit a positive Wasserman. Again, an undiagnosed fever calls for a Widal test, and this is positive occasionally. In one case this was so, and the patient died of carcinoma of the body of the uterus.

Carcinoma of the stomach occurs too often in men, and usually when diagnosed is too late to offer any hope of permanent cure.

Gaither, in his article based upon the study of two hundred forty-five cases, gives the following early diagnostic signs:

1. Average loss of weight, twenty-nine pounds.
2. Loss of appetite, fifty-five per cent.
3. Pain (Epigastric), seventy-one per cent.
4. Occult blood in stools, seventy-six per cent.
5. Nausea and vomiting, sixty-seven per cent.
6. Anemia, sixty-three per cent.
7. Motor function, obstruction, sixty-four per cent.
8. Achylia, sixty-four per cent.

There are other early diagnostic signs, but those listed above have occurred in the largest per cent. of the cases that have come under his observation.

Loss of Weight: This is a fairly constant finding and should always be regarded with suspicion.

Loss of Appetite: Is variable. One patient with an advanced carcinoma and marked emaciation, volunteered that her appetite was excellent.

Pain: May be of every possible kind; sharp, shooting, boring, burning, twisting, cramp-like, aching, and pressing.

Occult Blood in the Stool: Is a most valuable finding.

Nausea and Vomiting: Occur quite often and are associated, as a rule, with sour stomach.

Anemia: Low readings are seen occasionally early, but a large number of cases present color indices near normal.

Motor Function—Obstruction: Is noted in a large per cent. of cases, with all sorts of irregularities, usually in the region of the pyloric antrum.

Achylia: Associated with obstructive x-ray findings is decided evidence toward cancer. Remember, also, to look for Oppler Boas bacilli. If found along with the above findings, a diagnosis of carcinoma of the stomach may be made without fear of contradiction.

CARCINOMA OF THE FEMALE BREAST.

Carcinoma of the female breast takes its heavy toll yearly, and some of the early diagnostic signs adopted by the American Society for the Control of Cancer are:

1. Beginning fixity of the nipple.
2. Retraction of the nipple on the affected side upward and outward.
3. Bloody discharge from the nipple.
4. Deformity of the breast contour.
5. Presence of a lump in the breast.
6. The lump is hard in consistency.
7. The lump is painless in three-fourths of all cases.
8. Skin dimpling or attachment.
9. Cancer of the breast is opaque by transillumination.
10. Aspiration of punch biopsy may establish the diagnosis.

Transillumination of the breast serves a very useful purpose when in doubt, and should be used by every practitioner and surgeon.

The basic principle of transillumination is the difference in the transmission of light by tissues of different density and composition.

Three types of transmission are recognized:

1. Solid tissue on transillumination causes a shadow of moderate density.
2. Blood casts a very intense shadow of a much greater opacity per unit volume than solid tissue.

3. Clear fluid is translucent, casting no shadow.

CARCINOMA OF THE CERVIX UTERI.

Carcinoma of the cervix uteri calls for diligent investigation, and every woman is entitled to a pelvic examination, particularly if she has borne children.

The use of Lugol's solution on the mucous membrane of the vagina and cervix may establish in most cases the absence of early cancer. The Lugol solution is applied to the cervix by a cotton swab.

In the normal living tissue of the vagina, the epithelium is stained, in a few seconds, a deep mahogany brown. When the entire area takes this stain, the test is specific for determining the absence of cancer of the portio and vagina.

A white area on the cervix or vagina following Lugol's solution means either:

1. Early carcinoma (edges sharply defined).
2. Erosion or ulcer.
3. Loss of surface epithelium due to recent trauma.
4. Eversion of glandular epithelium of the endocervix (rare).
5. In areas of chronic cervicitis a light brown color blends with the deeply stained normal tissue (precancer, our condition).
6. Luetic or tuberculous ulceration.
7. Leucoplakia (white spot disease), precancerous condition.

The diagnosis of carcinoma of the body of the uterus is very difficult and frequently, when finally diagnosed, it is too late.

1. A sound passed into the uterine cavity, returning bloody, should call forth for curettement and microscopic study.
2. Novak's presentation of the suction curette should bring to light more early cases of this kind.

CARCINOMA OF THE RECTUM.

Brindley states that approximately sixty per cent. of all intestinal cancers are in the rectum, usually as a local lesion in the beginning, and amenable to surgical removal with a good prospect of permanent cure. The most frequent signs of the disease are.

1. Blood in stools.
2. Pain.
3. Disturbance of bowel function.

The above three points, associated with general signs, warrant a thorough examination including digital, procto-sigmoidoscopic, and x-ray studies.

No attempt has been made to cover the whole field of diagnosis in this paper. Accordingly, the parts of the body in which cancer occurs most frequently have been reviewed and their early diagnostic points indicated.

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Extracts.

CYANOSIS OF THE NEW-BORN.

E. A. MORGAN, M.B., AND ALAN BROWN, M.D., *Toronto, Ont.*
J. A. M. A., Vol. 105, No. 14, October 5, 1935.

Cyanosis of the new-born infant is a problem which should challenge the obstetrician, general practitioner, as well as the pediatrician, because no other condition which occurs so frequently suffers from more slipshod methods of prevention, inaccuracies of diagnosis, and uncertainties of prognosis. Unfortunately, much of the supposed successful progress made in overcoming the condition in recent years is probably due to the large percentage of spontaneous recoveries.

The authors list of commonly accepted causes of cyanosis are listed below, and their comments on those that are not self-explanatory, noted:

CAUSES DUE TO ACCIDENTS OF LABOUR.

1. Aspiration of mucus.
2. Atelectasis.
3. Prolapsed cord or cord around the neck.
4. Early separation of the placenta and low implantations of the placenta.
5. Prolonged difficult labor.
6. Breech presentation with difficulty in delivering of after-coming head.
7. Severe circulatory or toxic conditions of the mother, or drugs, particularly morphine, administered to the mother during labor.
8. Cerebral edema.
9. Intracranial hemorrhage (traumatic).

CAUSES DUE TO PATHOLOGIC CONDITIONS OF THE INFANT.

1. Prematurity.
2. Persistent thymus.
3. Diaphragmatic hernia.
4. Tracheo-esophageal fistula.
5. Congenital cardiac malformation.
6. Tongue swallowing
7. Pneumonia of the new-born.
8. Tetany of the new-born.
9. Sepsis of the new-born.
10. Intracranial hemorrhage (spontaneous).

The authors list Aspiration of Mucus, and Atelectasis separately, but discuss them simultaneously, because, as they say, they are

nothing more than cause and effect. The mucus closes the alveolar passages, impeding the relief of the atelectasis, and acting in the same manner as if one deliberately would choke the infant by compressing the trachea with the fingers. Dunham has shown that x-ray greatly simplifies the usual difficult diagnosis of atelectasis.

Prevention of cyanosis due to aspiration of mucus, is not difficult if a metal suction-tip with smooth surfaces, bent to conform to the curve of the infant's mouth and pharynx, is used. However delay in attempting to remove the mucus, use of coarse gauze to wipe out the secretions, or too vigorous use of a hard rubber catheter should be avoided. Care should be taken to avoid introducing pyogenic bacteria into the infant's respiratory tract, by inserting a tube which has not been sterilized.

Cerebral edema probably occurs more frequently than it is supposed, and resembles an intracranial hemorrhage in history of long and difficult labor and delivery, but differs from it in that the symptoms of cyanosis, vomiting, refusing to nurse, twitching of extremities, and even convulsions are delayed until eighteen to twenty-four hours after delivery. Moreover, the examination of spinal fluid is usually negative except for a few red blood cells appearing microscopically. Substantiating pathological evidence in support of this clinical theory is difficult to obtain, unfortunately. The theory depends on the evidence that the brain receives sufficient traumatism to produce a reactionary edema.

There is probably no condition more difficult to diagnose and prognose than intracranial hemorrhage, probably due to the fact that the hemorrhages vary so greatly in size, and because physical signs such as reflexes, muscle tone, and ocular manifestations cannot be depended upon as aids in diagnosis. Moreover, it is difficult to ascertain from the laboratory examination whether gross blood found in the spinal fluid is a result of intracranial hemorrhage or imperfect technique in the lumbar puncture; and even if the blood can be definitely shown to be of cerebral origin, it does not have the prognostic significance that has been attributed to it, because as Ford has shown, complete recovery has been made in numbers of these cases. The following factors should be considered strongly suggestive, at least, of intracranial hemorrhages: Asphyxia pallid, difficulty in deglutition, stertorous breathing, full and boggy fontanel, convulsions—occurring after a difficult or instrument delivery or in a premature infant. The demonstration of gross blood in the cisternal fluid and to a lesser extent in the spinal fluid. An accurate prognosis cannot be given in the first few days, but if the hemorrhage is not subtentorial, in which case the infant usually succumbs in the first week, the chance for complete recovery is not as hopeless as has been heretofore believed.

Recent improvements in roentgenological technique which make the diagnosis of a greater number of cases of atelectasis possible, and advances in blood chemistry which make possible the diagnosis of tetany in the new-born, have greatly decreased the number of deaths that have previously been attributed to thymic dysfunction. The diagnosis of these cases of thymic hypertrophy were very logical since often they were greatly improved by roentgen-ray therapy, but it must be recognized that there was never any actual scientific proof that an internal secretion of the gland was responsible. And although we are still unable to account for some causes of sudden death, except by the theory of thymic dysfunction we should remember that it has never been shown by actual proof to be a cause of cyanosis in the new-born infant.

Tetany has been receiving a great deal of attention as a cause of cyanosis of the new-born. The clinical picture is similar to intracranial hemorrhage or cerebral edema except that the symptoms may occur any time within the first few weeks of life. The clinical symptoms are verified by low blood serum calcium. Faulty diet of the mother during pregnancy, hypoparathyroidism in the infant—possibly due to trauma, and alkalosis in the new-born have all been suggested as possible causes of tetany in the new-born infant.

The author's analysis of present knowledge concerning cyanosis in the new-born infant is certainly a challenge to discussion and careful consideration of this important problem in order that a greater uniformity in the conception of the etiology of cyanosis and in the diagnostic methods employed may be reached.

ACUTE SYPHILITIC MENINGITIS.

ROMA AMYOT, *Union med. du Canada, Montreal.*

Dec. 1936, 65: 1161.

Acute syphilitic meningitis is rare. In 70 per cent of the cases of syphilis there is some infection of the meninges during the secondary stage but fortunately this infection is generally slight and transitory and shown only by examination of the spinal fluid. Therefore the practitioner is not generally prepared to think of syphilis as a cause of meningitis. Moreover the symptoms differ considerably from those of meningococcus of tuberculous meningitis and in some cases, especially if associated with chronic suppurative otitis, may suggest cerebral abscess.

The author describes such a case in a young man 23 years of age who came to the hospital with such intense headache that rest and sleep were impossible. He had a history of chronic suppurative otitis on both sides for two years and hearing was impaired on the left side. There was a considerable meningeal reaction of the lymphocytic type without the typical clinical signs of meningitis but with marked signs of intracranial hyperpressure; the patient was in very bad general condition and had a trace of left hemiplegia. A cerebral abscess of the right temporal region originating from the otitis was suspected. Ventriculography however showed the absence of abscess. In the meantime the report from the laboratory on the lumbar puncture had come in showing positive Wassermann, benzoin, and colloidal gold reactions. The patient had become able to give a history and said that about nine months before he had a small swelling on the glans and enlargement of the inguinal glands. He had been given three series of intravenous injections, probably of novarsenobenzol; it was three weeks after the last of these that the meningitis began.

Another patient, a woman of 47, was admitted to hospital with violent headache. The previous month she had had three attacks with loss of consciousness, tonic convulsions, and blueness of the lips. There was no history of sinusitis, otitis, coryza, or tonsillitis. She too had signs of intracranial hypertension with bilateral choked discs, the spinal fluid showed a lymphocytic type of inflammation without bacteria, and slight paresis of the left limbs as a sign of localization on the right side. These signs too suggested cerebral abscess but remembering the previous case the blood and spinal fluid tests were made at once and both were strongly positive. It was evidently a case of syphilitic meningitis. The patient did not remember having had any skin eruption and she had never had any treatment.

In these cases of syphilitic meningitis the meningeal infiltration interferes with the flow of spinal fluid and so brings about the signs of intracranial hypertension. The typical signs of acute meningitis such as are seen in tuberculous meningitis—Kernig's sign, rigidity of the neck, photophobia, hyperesthesia of the skin, and retraction of the abdomen—are absent or slight. There may be considerable rise of temperature.

The clinical signs of this syphilitic meningitis recede quite rapidly under specific treatment. Unfortunately the spinal fluid reaction may persist and tertiary syphilis or even general paralysis may develop. These patients with resistant spinal fluid should be given malaria treatment to prevent the development of general paralysis.

GONORRHEAL INFLAMMATION OF THE BONES IN INFANTS.

JAUN P. GARRAHAN AND CARLOS RUIZ, *Semana med.*,
Buenos Aires, Dec. 10, 1936, 43 : 1620.

A case is described in an infant born Apr. 4, 1934. The mother had a gonorrheal discharge and at the age of 7 days the child developed gonorrheal conjunctivitis. At the age of two weeks the left elbow was swollen and red. The redness extended down the forearm to the roots of the fingers. This was accompanied by soft edema around the elbow. Movement and palpation of the joint were painful. There was no fever. The swelling and redness of the joint receded within three weeks but the same condition developed in the right elbow. This recovered in even less time than the affection on the left side. The child's general condition remained quite good.

Roentgenograms of the arms showed a picture that looked exactly like that of congenital syphilis of the bones. There was hypertrophic periostitis and in the bones of the forearm bands of calcareous condensation like those seen in syphilis. This suggested the possibility of congenital syphilis but the serologic reactions were negative in both mother and child. The clinical signs disappeared within a few months but some roentgen signs of the bone involvement persisted for about a year and a half. The child was kept under observation for two years and during that time did not develop any clinical signs of congenital syphilis.

This case shows that gonorrheal arthritis does occur in the newborn infant though it is not common and therefore is the more apt to be mistaken for congenital syphilis, particularly as the roentgen picture may be very much like that of syphilis. The roentgen picture of gonorrheal arthritis in the newborn is very varied and there is no sign that is pathognomonic of the disease. Gonorrheal arthritis is apt to develop within the first 30 days of life when the child has gonorrheal ophthalmia and again after two years of age when the period of gonorrheal vulvovaginitis begins. It is important for practitioners to know that even a mild gonorrhea may cause arthritis and osteitis and that care must be exercised in making a differentiation between such bone affections and those caused by syphilis because of the difference in the treatment required.

DELAYED DEATH FROM PULMONARY EMBOLISM, M. W.

SHULMAN, M. D. *American Journal of Surgery*, November, 1935.

Sudden death from massive pulmonary embolism following venous thrombosis in the lower extremities is by no means uncommon. In general, there are two groups of cases: The first comprises those in which, following trauma of the lower extremities, thrombosis of the femoral vein occurs. This may or may not be associated with infection. The second is made up of those in which thrombosis of the femoral vein occurs after obstetrical procedures, operations in the pelvis or elsewhere in the abdomen. This usually happens in clean cases and is generally attributed to stasis. Death in such cases is usually sudden and occurs when the thrombus breaks off and is swept through to the lungs. This often happens when the patient is recovering from the effects of the operation or injury and seems to be convalescing in a satisfactory way.

However, massive pulmonary embolism is not always an immediately fatal disease. H. E. Robertson (*American Journal of Surgery*, October, 1934) has shown that both pulmonary arteries may be almost completely occluded without sudden death. He states, however, that death occurs within a few hours at the most. Post-mortem evidence confirms this when the embolus is found to be organized and adherent to the intima of the pulmonary artery. Yet this takes time.

Two cases are reported in this article where the clinical history and the pathological findings show that massive embolism may occur and still the patient live for some days after. These patients lived seven days after the release of the thrombus. Reports of this type of embolism are rare.

At autopsy these cases showed almost complete occlusion of the pulmonary arteries and their larger branches. The first case was a result of stasis in the femoral vein plus infection. The second case was a result of the stasis alone.

The medico-legal aspect is pointed out. Death has often occurred after a considerable time has elapsed and in these instances it is essential to ascertain whether the actual cause of death is legally the primary cause. This applies particularly in cases of violence where the victim dies sometimes after the initial injury. The question frequently arises whether the patient succumbed to the initial injury or not.

Medical Notes and News

RE-ORGANISATION OF I.M.S.

PROTEST BY PUNJAB CONFERENCE.

Demand for raising the standard of medical education was among the recommendations of the 7th Punjab Provincial Medical Conference which concluded at Rawalpindi. The Conference was presided over by Dr. K. R. Chaudhri. Among the resolutions passed were :—

1. Resolved that this Conference of the Punjab Provincial Branch of the Indian Medical Association records its emphatic protest against the scheme for the re-organisation of the I.M.S. as announced in the communique of the Government of India on 25th March 1937 and considers the scheme as most retrograde, reactionary and against the professed policy of Indianisation of the service. In practice this policy means the perpetuation of the European element in the service and the elimination of the Indian element. This Conference hereby demands the complete abolition of the civil side of the I.M.S.

2. This Conference resolves that the Government be requested to take early steps to amend the Punjab Medical Registration Act of 1916 as it does not meet the present-day requirements, (b) that in the amended Act the system of nomination of members and of having a nominated president be done away with and (c) to remove the invidious distinction between the various constituencies of the electorate, (d) it further resolves that safeguards should be provided against quackery.

3. Resolved that all fees earned by Government medical men, attached to Government hospitals, be credited to the Government especially those earned from laboratory, X-Ray and private medical wards which run at the Government expenses or using Government material.

STANDARD OF MEDICAL EDUCATION.

4. Resolved that in the opinion of this Conference it is high-time that the standard of education in medical schools be suitably raised in order to conform to the minimum standard of qualification for registration as laid down by the Indian Medical Council.

5. Resolved that this Conference requests the Government to institute a National Health Insurance Scheme without any further delay for the benefit of the public and the provincial conference is requested to formulate the scheme.

6. This Conference urges upon the Government to allow the nationals of those countries only to practise in India which have

reciprocity to allow nationals holding Indian medical degrees to practice in their country.

7. This Conference is of opinion that certificates signed by a registered medical practitioner should be recognised without a counter signature. Further resolved that to keep up its prestige the Punjab Medical Council should take early steps to represent the matter to the Punjab Government.

8. Resolved that the Government be approached to make provision for a post graduate course as is done in other countries.

9. Resolved that this Conference urges its members to prescribe Indian made drugs as far as possible and further calls upon the Provincial Government to instruct its various departments to purchase medical supplies manufactured in India.

10. Resolved that the Central and Provincial Red Cross Executive Council be requested to employ and associate more of medical men as it will enhance the efficiency of that department.

THE TINNEVELLY DISTRICT MEDICAL ASSOCIATION, PALAMCOTTAH.

(BRANCH OF THE INDIAN MEDICAL ASSOCIATION).

A Special Meeting of the Tinnevelly District Medical Association was held on Saturday the 16th of October 1937, at 6 P.M. in the premises of the Government Women and Children Hospital, Vannarpet, to meet Dr. M. Subramania Iyer, M. B. & C. M., President of the South Indian Medical Union, Madras, and a vice-Chairman of the Reception Committee of the All India Medical Conference. Before the meeting the gathering was entertained to light refreshments. Twenty-seven members of the Headquarters were present on the occasion.

Lieut. Col., T. S. Shastri, I.M.S., who presided over the meeting said that they had all met there to welcome Dr. M. Subramania Iyer, and made an appreciative reference about him and his career.

There was a free discussion in which most of the members took part, about the present position of the Medical Profession in our Province, Medical Education, and Medical Associations. Dr. Subramania Iyer advised the formation of a Medical Association in Madras, which should have its branches in all the district and which should have autonomy for itself while it may work in co-operation with any All Indian Medical Association and exhorted that as many members as could afford it, should join the Reception Committee and attend the forthcoming All India Medical Conference in Madras, and press their views on the rest of the Profession assembled there. Dr. N. Isaac opposed the abolition of the L.M.P. Course.

There was general agreement among the members on the following points :—

(1) Every member of the Profession in our Province must support the Reception Committee of the Indian Medical Conference,

and make the Conference a great success, (2) every member of the Profession should join the Indian Medical Association and make it the one spokesman of the Profession whether for the Province or for the whole of India, (3) all the practitioners of Madras City, should join the Madras Branch of the Indian Medical Association (4) all the other sectional medical associations like the South Indian Medical Union, and the Licentiates' Association working in Madras should be *forthwith* amalgamated with the Madras Branch of the Indian Medical Association, as the Indian Medical Association could serve every legitimate interest of the Profession much more effectively than any of them, and (5) the L.M.P. course should be abolished.

Dr. T. N. Govinda Iyer, M.B.&C.M., the Vice-president proposed a hearty vote of thanks to the respected visitor, which was carried with acclamation.

ALL INDIA ANTI-INDIAN MEDICAL COUNCIL DAY.

Extracts from the proceedings of the Protest Meeting of the Bombay Medical profession held under the joint auspices of the All India Medical Licentiates' Association Bombay Branch, Indian Medical Association Bombay Branch and the Medical Association Bombay North, under the Presidentship of Dr. S. Sanjivarao, M.B.B.S. (Madras) at Dr. Sardesai's Eye Hospital, Sandhurst Road, Bombay on 20th September 1937.

The President at the outset thanked the House for the honour done to him by electing him as the president of the meeting and than briefly explained to the house the purpose and the significance of the meeting and requested Dr. U. B. Narayan Rao to speak on the subject, *viz.*, the Indian Medical Council Act 1933. Dr. Narayan Rao in his speech briefly referred to the shortcomings in the Indian Medical Council Act 1933, which was enacted in spite of country-wide protests. The Indian Medical Council, he said is official-ridden and a large section of the Indian Medical profession, *viz.*, the Medical Licentiates, are totally excluded from the purview of the Act. He further observed, the Medical profession of India have got to agitate and agitate until the Act is radically changed so as to bring it to the lines of the present day requirements.

Dr. M. C. Bilpodiwala then moved the following resolution in a short speech :—

“Resolved that this meeting condemns the Indian Medical Council Act 1933, and requested the members of the Indian Legislative Assembly to take early steps as to provide therein amongst other things a more suitable arrangement for reciprocity and the inclusion of the Licentiates within its purview.”

* Then Drs. Jivraj, N. Mehta, B. Y. Paranjpe, U. Shesha Rao and G. G. Wadhvani spoke supporting the resolution.

The resolution was passed unanimously.

Dr. U. Shesha Rao was authorised to send copies of the Resolution to (i) The Secretary I. M. Council (ii) The Secretary The Indian Legislative Assembly, and (iii) to the Medical Press.

THE TINNEVELLY DISTRICT MEDICAL ASSOCIATION.

Held its monthly meeting on Saturday the 25th of September 1937, at Sankarankoil. Members numbering 45 were present from all parts of the District.

After Tea Party and music, the meeting began under the Presidentship of Lieut. Col., T. S. Shastry, I.M.S. The Secretary Dr. K. Rama Ayyar, M.B.B.S., read the minutes of the monthly meetings held at Courtallam and Headquarters in July and August which were passed.

1. After detailed and full discussion the following resolution was passed unanimously :—

“The object of the Indian Medical Association started on the model of the British Medical Association, being to develop medical and allied sciences, to improve the public health, to maintain the honour and dignity as well as the interests of the Medical Profession, to bring about the unification of all Branch of the Medical Profession in India and to improve *esprit d' corps* among them.

And the Indian Medical Association being the only Association in India which provides a common platform for all classes and strata of allopathic Medical Practitioners practising in India.

The monthly Meeting of the Tinnevelly District Medical Association, assembled at Sankarankoil, on the 25th of September 1937, hereby unanimously resolves, after a full discussion, that the Tinnevelly District Medical Association should be converted into a proper Branch of the Indian Medical Association with effect from the 1st of October next.”

2. The following resolutions were also passed unanimously :—

(a) An extra subscription of One Rupee per year per head be levied in consideration of the conversion of our Association into a Branch of the Indian Medical Association to meet the Association expenses.

(b) The Tinnevelly District Medical Association strongly supports the Bill to amend the Madras Medical Registration Act of 1914 proposed to be moved by Dr. P. S. Srinivasan, M.L.A., in the current Session of the Madras Legislative Assembly and urges on the Minister for Public Health and the Legislative Assembly Members to help in passing the same at an early date.

(c) In view of the fact that there has been a persistent demand for the last 25 years from the different Medical Associations and the Medical School students in the country, and in view of the fact that

such a change is imperatively necessary to set up a recognised and efficient standard of Medical education, this meeting urges on the Minister for Public Health to make the I.Sc., the minimum educational qualification for eligibility for admission into the Medical Schools under the control of the Government of Madras.

Dr. P. S. Srinivasan, Sub-Assistant Surgeon of the Government Headquarters Hospital, Palamcottah, read out clinical notes on the following interesting cases which were treated at the Government Headquarters Hospital, Palamcottah :—

1. Two cases of severe Burns.
2. A case of Empyema of Maxillary Antrum.
3. A case of carcinoma of Thyroid.

Dr. Janardhana Rao, L. M. & S., read out interesting notes of a case of "Serum Sickness."

A case of Empyema of the Maxillary Antrum successfully operated on by Lieut. Col., T. S. Shastry, I.M.S., was exhibited to the members. The specimens of Necrosed Duodenum and Ante-mortem clots in the Heart derived from two fatal cases of Burns were also shown to the members.

Thereafter Col. Shastry, summed up the salient and important features of all the cases and offered his thanks to Dr. T.R. Srinivasa Iyengar, for the splendid arrangements. Thereafter an excellent Dinner brought the happy gathering to a close late at night.

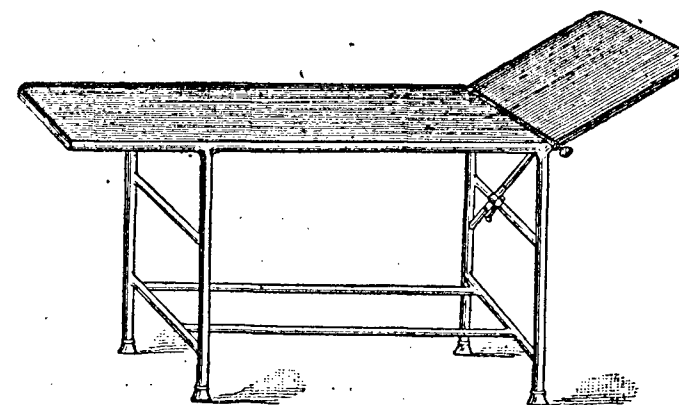
Under the auspices of the Tinnevely District Medical-Association, in connection with its monthly meeting at Sankarankoil, a Health Exhibition, was arranged at the Board High School, Sankarankoil, on Saturday the 25th of September 1937, at 10 a.m. under the auspices of V. Ramakrishna Esq., I.C.S., Collector of Tinnevely.

Lieut. Col., T. S. Shastry, I.M.S., the President of the Association, welcomed the guests, explained the great need for sustained propaganda to instil into our people habits of personal, domestic and rural sanitation and on behalf of Nutrition campaign to improve the resisting power of the people, and requested Mr. V. Ramakrishna to open the Health Exhibition.

Mr. V. Ramakrishna, in declaring the Exhibition open, commended the efforts of the Tinnevely District Medical Association to promote the health of our people, said that such Exhibitions are very useful, instructive, and beneficial to the public and school children.

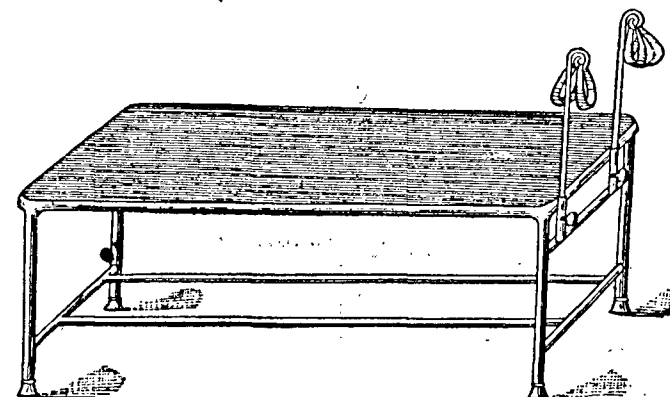
Mr. Mathuranayagam, the Health Inspector, explained the posters on prevention and causation of Tuberculosis, Flies, and Cholera.

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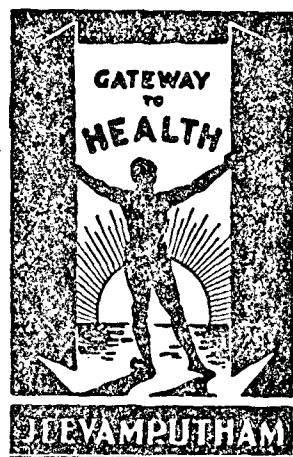
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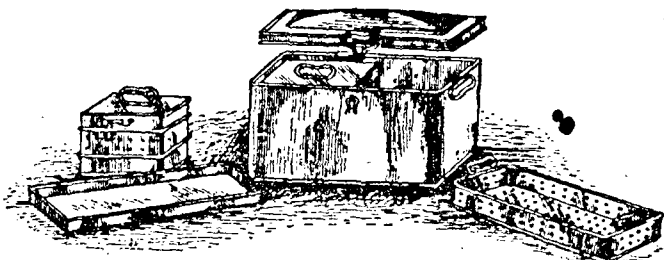
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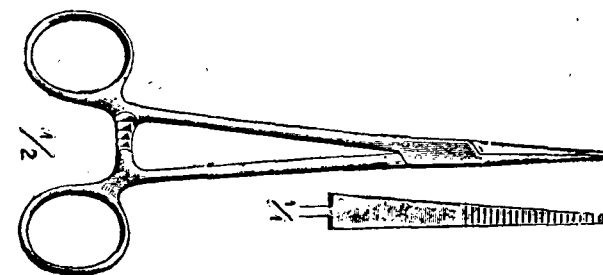


Fig. 3010.

Fig. 3010. Tonsil artery Forceps Berket's as used in General Hospital. Steel nickel plated.

Fig. 3011. Tonsil artery Forceps Berket's Stainless Steel.

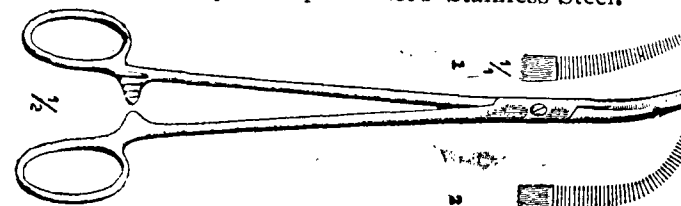


Fig. 3012.

Fig. 3012. Tonsil artery Forceps curved on flat, 1 or 2. Steel nickel plated.

Fig. 3013. Tonsil artery Forceps curved on Flat, 1 or 2. Stainless Steel.



Fig. 3014.

Fig. 3014. Tonsil Dissector, Durham, fine double ended, Steel nickel plated.

Fig. 3015. Tonsil Dissector same as above, Stainless Steel.



Fig. 3016.

Fig. 3016. Tonsil Dissector and pillar retractor, Steel nickel plated.

Fig. 3017. Tonsil Dissector and pillar retractor, same as above. Stainless Steel.



Fig. 3018.

Fig. 3018. Tonsil Dissector Forceps, Waughs—Steel, nickel plated 8" long.

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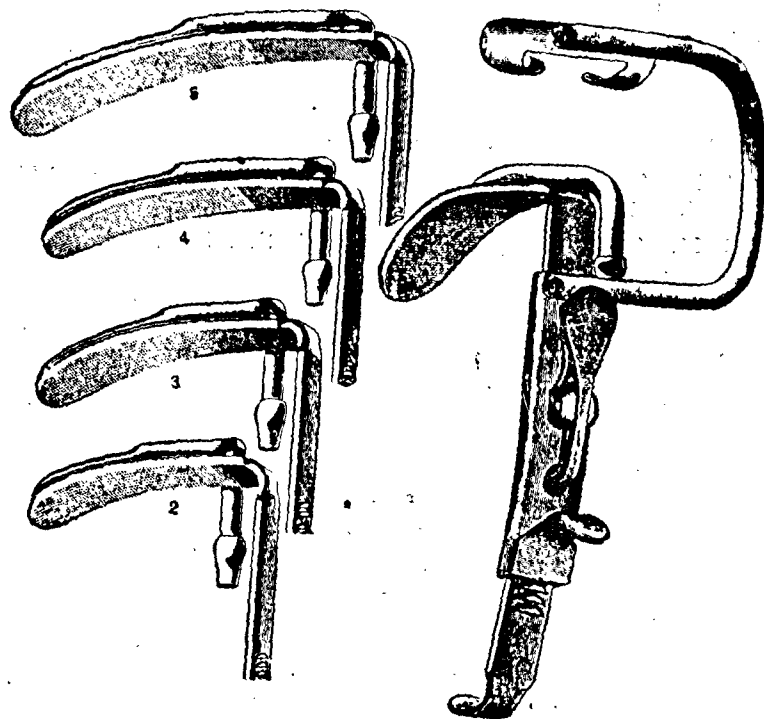


Fig. 3020.

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