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Vol. VII.

OCTOBER 1938

No. 10.

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VOL. VII

OCTOBER 1938

NO. 10

Original Article.

* PYORRHOEA ALVEOLARIS

(ITS CAUSES, DIAGNOSIS & TREATMENT.)

BY DR. S. K. GUPTA, B.D.Sc., B.O.

Dental Surgeon. (Pasrur).

Today we hear a great deal about the dental disease, the Pyorrhoea Alveolaris.

Many people think that it is purely a modern disease of the mouth confined to civilised people of the last two or three generations. This, however, is not the case. It is not a recent disease. Dental scientific research workers who have examined the skulls of ancient people have found clear indications that Pyorrhoea was present even in remote ages.

To give one interesting example that has been discovered in sarcophagus or rock tomb of Phoenician origin in Syria will throw a very illuminating light on the subject. It shows that a disease that still puzzles the dental profession today existed among those Eastern people in the fifth century B. C. and was treated, more or less successfully, by dentists some 2,500 years ago.

A male skeleton was found in one of those tombs, and it was authoritatively stated that the sarcophagus had remained unopened for 2,500 years. In the lower jaw of this skeleton there was found a gold appliance wrought by an ancient dentist to keep in place several teeth that apparently had become loosened by Pyorrhoea. An X-Ray examination of the mandible or lower jaw revealed the typical atrophy of pyorrhoea, and this seems to prove that the disease existed among those early civilised people. The appliance seems to have been highly successful, and was very skillfully made and inserted in the jaw. In those days, of course, the Phoenicians were famous for their skill in the arts and craft of metal work.

To-day the results of the disease can be observed upon the alveoli or bony process in which the teeth are imbedded, and the clinical appearance of the alveoli coincides with the ravages

• Exclusively contributed to the "Medico-Surgical Suggestions," Madras.

appearance of the jaws in ancient skulls which seems conclusive proof of the existence of Pyorrhoea in those days.

In the middle of the eighteenth century there were medical scientists who recorded their observations of the disease. They described it as a kind of scurvy that attacked the gums, the alveoli, and the teeth. They described the symptoms. The gums affected became livid, swollen, and inflamed. If a rather heavy finger pressure were applied to the gums from the top downward on the lower jaw, it would be found that a glutinous, yellowish white pus would issue from the junction of the teeth with the gums.

In those days scurvy was a very prevalent disease because men had not fully appreciated the fact that fresh fruits and vegetables were an essential factor in a diet that would prevent these bodily disorders.

Later in the eighteenth century another medical observer described it as an unknown kind of scurvy that seemed impossible to treat with any degree of success. Throughout the whole of the nineteenth century various other investigators concerned themselves with a study of Pyorrhoea. Many of them held different theories as to its cause and genesis, and different methods of treatment were employed. There were some who considered it was a disease of purely local origin. They contended that the tissues that connect the roots of the teeth to their bony sockets became infected by bacteria that set up inflammation, loosening the tooth from its attachment, and thereby causing the formation of pus during the process.

Others held an opposite view. They contended that the predisposing cause was not of local origin, but was entirely systemic. Some idiosyncrasy of the body in conjunction with some maladjustment of the metabolism was the cause of the trouble, and the disease of Pyorrhoea was a direct result of this disturbance.

To-day it is still a subject that is often discussed at the meetings of dental societies and forms the basis of many articles in the professional journals and magazines in all civilised countries. After many years of research and observation it is still a disease upon which there is a lot to be discovered.

In the first place it may be well to explain in simple words what is meant by Pyorrhoea. *Pyo*—means pus, and *pyorrhoea* means the discharge of pus. The alveolus is the hollow socket in the bone of the jaw in which the roots of the teeth are situated. The tissues that connect the roots of the teeth to the alveoli become infected with some particular organism or germ. Inflammation is set up. This results in the formation of pus. This pus is eventually discharged from a gum pocket or gingival trough formed around the neck of the tooth. As the disease progresses more and more tissue is destroyed. The gum becomes atrophied. As a result of this atrophy the roots of the teeth gradually become exposed and also

become loose in their sockets. As the alveoli becomes atrophied or wastes away the teeth eventually fall out.

During this state the affected tissues give rise to an offensive odour in the mouth, and the sufferer often experiences an unpleasant taste in the morning that appears of a metallic nature. And around the necks of pyorrhoeic teeth hard, brownish-black deposit of a calcareous nature begins to form. This clings to the cementum of the teeth, and seems to exert a corroding influence upon the adjacent tissues—the mucous membrane that covers the gums, the gum tissue itself, and the periodontal membrane or tissue that connects the tooth to the alveolus.

Strictly, there are three varieties of pyorrhoea, although the term is generally used to denote a morbid condition of the tissues around the necks of the teeth and about the gums. The dentist, however, usually classifies the disease under two headings—horizontal or marginal pyorrhoea and vertical or diffused pyorrhoea. The first variety can be recognised by inflamed gums that bleed easily and are tender to the touch. Often the necks of the teeth are covered with tartar of a yellowish-white substance. The vertical variety is distinguished by deep gum pockets around the teeth in which tartar of a brownish-black colour adheres to the roots and that causes suppuration and loosening of the teeth.

The exact cause of pyorrhoea is not yet definitely known. Disease is never the result of a single factor. It is a sequence depending upon many factors. If disease was the simple result of a single cause then the path of medicine would be a fairly plain and easy one. Disease, which is an abnormal condition of the normal functions of the organs or a series of organs of the body, is the result of complex causes and conditions—both physical and mental. There are so many things to upset the normal working of the body in civilised life. Age, sex, conditions of life, climate, habits, psychological influence, nutrition, physical and mental and temperamental idiosyncrasies, race and so forth, all have their effect upon the formation of a condition in which the body may become liable to disease or conversely may be able to resist disease.

Even when a disease is known to be the result of infection by a known micro-organism several factors play their part in the result. The germ may have become seated in the human tissues but it needs response from these tissues if it is to grow and spread its power of destruction. The response of these tissues may be influenced by any of the factors already given, and it requires very little imagination to comprehend the vast number of complexities that may occur in such circumstances.

Professor Herman Prinz, after much research, has summarised five principal theories that may be the cause of pyorrhoea, either separately or together. These are:

1. *The formation of salivary calculus or tartar around the roots of the teeth*
2. *Infection by some micro-organism.*
3. *Abnormal biting strain upon the teeth causing injury to the tissues around the roots of the teeth.*
4. *Constitutional disturbance of the body affecting the blood that feeds the periodontal tissues—i.e., the tissues around the teeth.*
5. *Senile atrophy of the alveoli or tooth sockets of the jaws.*

An interesting point is the age at which pyorrhoea is likely to attack a person. As a general statement it may be given that the middle years are the ones when a person is most prone to attack—forty to sixty years. Under twenty Pyorrhoea is not common. Between twenty and thirty the age incidence begins to increase. It increases most rapidly between thirty and forty, reaches its peak between forty and fifty, and after sixty shows signs of decreasing. This does not mean that the disease begins in one person, then gradually gets worse and then begins to clear up without treatment.

The most prevalent type is the horizontal type of pyorrhoea in which the gums become sore and inflamed and bleed easily, and in which masses of yellowish tartar are deposited around the teeth. The primary cause of this type of pyorrhoea is unhygienic oral habits. The thorough use of a tooth brush or a 'dantan' and the choice of a suitable diet will do much to prevent this type of pyorrhoea. Excess of sugary and starchy foods should be avoided because these tend to clog around the necks. When they are eaten they should be followed by substances of a fibrous, firm nature such as celery, vegetables, cheese, and fruits like apples and oranges.

When the tooth brush is employed it should be used to massage the gums as well as clean the teeth. A circular motion of the brush should be employed. When there is a predisposing for tartar to form easily around the teeth a visit should be paid at intervals to a well qualified local Dentist who will remove it by scaling and burring the teeth of the hard tartar. This type of pyorrhoea in the early stages can usually be treated very successfully.

The vertical type seems to be an essential accompaniment of many middle-aged people. It appears between the ages of twenty-five and sixty.

Because a person is affected with pyorrhoea it does not necessarily mean that the teeth should be extracted although this is a definite cure of the disease. In the early stages it can be treated, sometimes arrested, and cured.

The surgical or operative treatment that may be necessary cannot, of course, be done, by the reader. This is a job for the dentist but everyone can take measures of a prophylactic and dietetic nature that will help to prevent the disease. In the first place a suitable

tooth brush, *not too hard* in the bristles should be used. With it use a good and freshly prepared dentifrice which can be had from your well qualified Dental Surgeon.

In regard to diet this should be chosen so that the body is given a reasonable chance to remain healthy and also to prevent the formation of acids and gelatinous food debris likely to attack both the teeth and the soft structures of the gums.

Toasted bread is preferable to soft, new bread, and whole meal bread is preferable to white bread. Excess of sweet biscuit cakes and sticky confections should be avoided, and so should chocolates and coffee. Fish, bacon, meat and poultry are of a fibrous and cleansing nature. Vegetables such as lettuce, cress, celery and onions are preferable to cooked ones like cabbage and spinach. Fresh fruits like apples are good. Intelligent choice in these matters by the reader is the main thing needed.

THE USE AND ABUSE OF ERGOT AND PITUITARY.

Ergot and solution of posterior pituitary are the most important oxytocic drugs in the pharmacologic armamentarium, says M. Edward Davis in "Journal A.M.A." Their judicious use has been a boon to the practice of obstetrics. Properly used they are of inestimable value in the course of labour and the puerperium, often contributing directly or indirectly to the saving of human life. However, their indiscriminate use has led to disastrous results for mother and baby. In fact, many authorities have intimated that the undesirable consequences of the improper use of these oxytocic drugs have outweighed the good that they have accomplished. It is because of this serious indictment that the review of the proper indications for the use of these oxytocic in obstetrics may be timely.

ERGOT.

Ergot is a fungus that attacks rye and other grains, making them unfit for consumption. The crude drug contains many constituents, but it owes its oxytocic activity largely to the alkaloids which it contains. Many of the pharmacologic investigations of ergot concern themselves with the isolation and study of these alkaloidal constituents. Tanret in 1875 isolated ergotinine, which proved to be inert. Barger and Carr in 1906 isolated ergotoxine, which was found to have oxytocic activity. Stoll in 1918 isolated the isomers ergotaminine and ergotamine, only the latter alkaloid exhibiting oxytocic properties. Other alkaloids, namely, sensibamine and ergoclavine, have been described. It remained, however, for a

group of investigators in 1934 to isolate and describe a new water soluble alkaloid which for the first time satisfactorily accounted for most of the desirable oxytocic activity known to exist in ergot. The Council on Pharmacy and Chemistry of the American Medical Association has given the name of ergonovine to this new alkaloid.

The chemical and pharmacologic properties of this new substance have been carefully evaluated by a number of investigators, all of whom agree that this new water soluble, crystalline ergot base, which has the formula $C_{20}H_{23}N_3O_2$, is responsible for most of the oxytocic properties of active ergot preparations.

Although this new alkaloid is proving to be highly desirable as a therapeutic agent in obstetrics, the continued investigations on ergot have been productive of a great deal of new information. The extensive researches of Jacobs and Craig have demonstrated that lysergic acid is the only characteristic fission product common to the four pairs of alkaloids; thus, the final chapter in the chemistry and pharmacology of ergot still remains to be written.

The ergot preparation in the Pharmacopoeia of the United States is the fluid extract. When prepared according to official instructions it should contain most of the ergonovine present in the crude drug on which its oxytocic potency depends. The official assay by the U.S.P. cock's comb method will determine this potency. Ergonovine can best be assayed biologically by means of the isolated rabbit's uterus, the official cock's comb method and the isolated rabbit's intestine, and by a colorimetric procedure. There is considerable variation in the content of this potent alkaloid in the various samples of ergot obtained from one locality and a much greater variation in ergot obtained from different geographic locations.

The present status of ergot therapy has been considerably simplified. The official fluid extract, when properly prepared and stored, provides a good ergot preparation which can be taken by mouth in 30 to 60 minim (2 to 4 c.c.) doses two or three times daily. It occasionally provokes unpleasant reactions such as nausea and vomiting, in which case its use should be discontinued. Prolonged administration may result in the phenomenon of ergotism, which usually manifests itself in the form of a dry gangrene of the extremities. The drug should therefore not be continued beyond ten days.

The new ergot base ergonovine in the form of a salt may be used orally and parenterally because of its crystalline character. The malleate salt of the base is stable and satisfactory for therapy. For oral administration from 0.2 to 0.4 mg. can be given two or three times daily. It has an advantage over the fluid extract preparation in that it can be given intravenously in 0.2 mg. doses for its immediate effect when this is desirable. The parenteral routes of administration may be necessary in patients who are under anaesthesia or when the oral route is contra-indicated. Although as yet

no cases of ergotism have been ascribed to ergonovine, prolonged administration of huge doses in experimental animals have resulted in this phenomenon.

The characteristic response of ergot can best be seen in the immediate postpartum period. Some ten or fifteen minutes following the oral administration of the drug, the uterus exhibits increased activity. The normal uterine contractions are increased in rate and severity. The uterine musculature develops considerable tonicity, so that the organ does not completely relax between contractions. This increased muscular tone may persist for as long as an hour, but the rate and intensity of the contractions continue for one or even two hours after the uterine tetany has subsided. The increased rate and vigour of the contractions and the tetany are both desirable features during the puerperium, for they probably promote a more rapid uterine involution as well as control postpartum bleeding.

Ergonovine does not affect pulse, blood pressure or urinary output. It is therefore particularly indicated in patients who exhibit any of the vascular-renal manifestations of the toxæmias of late pregnancy. Fluid extract of ergot contains chemical constituents such as the amines, which may affect blood pressure.

SOLUTION OF POSTERIOR PITUITARY.

The oxytocic activity of extracts of the posterior pituitary gland was demonstrated by Dale in 1906. Blair Bell three years later introduced this most important therapeutic agent in obstetrics. It was not until 1928 that Kamm and his co-workers succeeded in separating posterior pituitary into two fractions, one containing the oxytocic principle, pitocin, and the second the diuretic and pressor principle, pitressin.

Solution of posterior pituitary U.S.P. contains both principles, and is the most commonly used pituitary preparation in obstetrics. It is standardized as to oxytocic activity. The oxytocic standard is such that each cubic centimetre contains not less than 8 and not more than 12 international units. The international unit, as adopted by the League of Nations, is the amount of oxytocic activity obtained by 0.5 mg. of an international standard powder. The pressor principle is so adjusted as to be of the same magnitude as that produced by a comparable solution of a standard pituitary powder.

The pharmacologic action of posterior pituitary is quite uniform. It is entirely inactive when given by mouth. Its intramuscular or subcutaneous administration causes the initiation of uterine motility in from three to five minutes. The uterus contracts vigorously, remaining in this tonic state for several minutes, and, as the tonus diminishes, increasingly vigorous contractions occur. The action of the drug lasts for five or ten minutes and rapidly disappears, to be reinitiated by another dose. The intravenous administration of

small doses, 3 minims (0.2 cm.), provokes an immediate response. However, the general reaction to this mode of therapy is sometimes quite marked. The patient may develop a marked circumoral pallor, a sense of constriction in the chest, pain in the back of the head, marked palpitation, nausea, and occasionally vomiting, simulating shock. The reaction may last several minutes or longer and gradually disappears without any serious effects.

Solution of posterior pituitary has a variable effect on blood pressure of normal persons. Usually they show little or no elevation of blood pressure after therapeutic doses. The pallor which may develop is not an index of a change in blood pressure. However, in patients with hypertension a marked elevation occurs after intravenous or intramuscular administration of the drug. The rise in pressure is transitory and is followed by a return to normal. Solution of posterior pituitary also causes a diminution in the urinary output, which likewise becomes more marked in patients with kidney damage. A temporary anuria may occur in patients with toxæmia of pregnancy. These abnormally marked effects of pituitary solutions on blood pressure and urinary output in patients exhibiting toxæmia of pregnancy make the use of this drug undesirable in this group.

Dieckmann and Michel noted that parenteral injections of extracts of the posterior lobe of the pituitary were followed by a marked decrease in the volume of urine, an increase in the chloride concentration, and an average rise in the systolic pressure of 51 mm. of mercury pressure in a group of patients with pre-eclamptic toxæmia studied during pregnancy. Less marked changes were noted in normal gestation. In addition to warning of the inadvisability of using solution of posterior pituitary as an oxytocic drug in patients exhibiting evidences of toxæmia, these authors suggest that the changes induced by the drug may be of some diagnostic and prognostic value in the management of these cases.

INDICATIONS FOR OXYTOMIC DRUGS

Non-Pregnant Patients—Although solution of posterior pituitary will cause some contractions and tonicity of the uterus of the non-pregnant person, it is rarely indicated. The pathologic cause for the bleeding should be determined and treated. Any increased bleeding at the time of the menstrual periods or bleeding occurring between periods is sufficiently serious to warrant a careful examination and a correct diagnosis. It is never justifiable to give ergot or pituitary extract as a temporary expedient in the hope that this will control the bleeding unless one is absolutely certain that the underlying pathologic process causing the abnormal bleeding can await treatment.

Therapeutic Abortion.—Oxytocic drugs will not terminate a normal gestation in the first trimester of pregnancy. The uterus is not responsive to ergot and pituitary during this period. Experi-

mental evidence would indicate that the corpus luteum of pregnancy inhibits uterine response to posterior pituitary extracts. As term approaches the uterus becomes more and more susceptible to the action of this substance. Near term one can cause the uterus to contract vigorously after the administration of the drug.

Although oxytocic drugs fail to initiate an abortion in a normal pregnancy, ergot and pituitary may be used to hasten or to complete the process once it has begun. In inevitable and incomplete abortions, solution of posterior pituitary can be used in 0.5 to 1 cc. doses intramuscularly with benefit to the patient. This will cause increased uterine motility which may result in a complete and rapid termination of the process or the evacuation of portions of the products of conception still in the uterus. Profuse bleeding should be controlled by curettage and packing if necessary. Following the completion of an abortion, ergot can be given orally for several days to limit the bleeding and favour normal involution.

Induction of Labour.—The medicinal induction of labour is rarely successful until term is approached. This is probably due to a lack of sensitivity on the part of the uterine musculature to oxytocic stimuli. If, therefore, it is necessary to induce labour for some complication prior to term, one usually has to resort to mechanical means. The method of medicinal induction of labour at or near term at the Chicago Lying in Hospital consists of the following procedure: Castor oil, 1½ ounces (42 cc.), is given early in the morning, followed two hours later by quinine in 3 grain (0.2 gm.) dose at hourly or half-hourly intervals for four or five doses, followed by graduated doses of solution of posterior pituitary, beginning with half a minim (0.03 cc.) and increasing half a minim at each dose fifteen minutes apart until 3 minims (0.02 cc.) is given. The latter dose can be repeated until a total of 1 cc. is used. The uterus should be carefully observed, and if it exhibits tetanic contractions so that the foetal heart tones are interfered with solution of posterior pituitary is discontinued. In the rare event that the continued tonic state of the uterus so seriously interferes with the uteroplacental circulation that the foetal heart rate is dangerously slowed, it may be necessary to administer ether to the patient for several minutes to relax the uterus. The administration of the drug is stopped when regular rhythmic contractions are initiated in the course of the induction.

Labour.—The oxytocic drugs are almost never indicated during the first and second stages of labour. It is dangerous to interfere with the normal uterine motility. Solution of posterior pituitary given during the course of labour initiates a marked tetany of the uterus. As this tone diminishes, the uterine contractions increase in severity and frequency. This abnormal uterine action may result in an interference with the placental circulation resulting in foetal asphyxia and even death. It may likewise result in an interference

with the uterine circulation and subsequent damage to the uterine musculature. The force of the uterine contractions driving the presenting part against an incompletely prepared passageway may result in serious lacerations of the cervix, the vagina or perineum or, in the event that the resistance offered by these structures is too great, the uterus may rupture. Furthermore, the abnormal stimulus provided by the solution of posterior pituitary may completely alter the normal course of labour so that serious operative interventions may be necessitated in an attempt to save a baby in jeopardy or to conclude an abnormal labour.

- Occasionally uterine inertia develops as a result of a lack of tone on the part of the uterine musculature or because of the general physical condition of the patient. Oxytocic drugs are rarely indicated in stimulating uterine contractions. The underlying condition that is responsible for the inertia should receive proper treatment. Thus, physical exhaustion on the part of the patient from a long, arduous labour may be best treated by sedatives. A period of complete rest will often result in a recurrence of normal uterine activity and a rapid completion of the labour. In some cases rupture of the membranes under proper conditions provides a satisfactory stimulus to poor and ineffective pains. At other times a hot enema may do the same thing. Rarely is it advisable to give solution of posterior pituitary. If this is done, the dosage should be limited to 1 or 2 minims subcutaneously. This should not be repeated many times. If there is any tendency to uterine tetany or to abnormal uterine motility, or if the foetal heart rate or rhythm is disturbed, the drug should be immediately discontinued and the uterus calmed with a narcotic.

Solution of posterior pituitary and ergot find their greatest usefulness in the third stage of labour. Used judiciously and with the proper indications, the oxytocic drugs may often prevent the occurrence of postpartum haemorrhage and the serious results that may be caused by it.

In the normal conduct of the third stage of labour it is usually advisable to await the complete separation of the placenta, after which its expulsion can be aided. Immediately after the birth of the child, 1 cc. of solution of posterior pituitary can be given intramuscularly. In the event that the patient has had no anaesthetic, fluid extract of ergot can be given by mouth or ergonovine can be given parenterally. These oxytocics should help to maintain the uterus in a contracted state avoiding any unnecessary bleeding.

If excessive bleeding occurs after the delivery of the baby and prior to the placental separation, one should administer 1 cc. of solution of posterior pituitary intramuscularly. The placenta can then be expressed by Crede's method. If this procedure fails to separate and expel the placenta, one should consider its manual removal in the face of continued haemorrhage.

Should excessive bleeding occur after the expulsion of the placenta, one must determine the cause for the bleeding. Uterine atony is only one of the causes for postpartum haemorrhages. Pastore recently reported that it accounted for only 22 per cent. of the haemorrhages occurring in his series of cases. Portions of the placenta may be retained in the uterus, resulting in a failure of the uterus to remain contracted. These fragments must be manually removed under proper aseptic conditions, after which oxytocic drugs will usually control further bleeding. Trauma of the soft parts, such as deep cervical tears and uterine rupture, may lead to serious and even fatal postpartum haemorrhage, which, needless to say, no oxytocic drugs will control. A careful diagnosis must always be made before the indiscriminate use of drugs is resorted to. When the haemorrhage is the result of uterine atony, solution of posterior pituitary in 1 cc. doses should be given subcutaneously or intramuscularly. Small doses, from 2 to 3 minims, can be administered intravenously in an emergency regardless of the possible reaction. Pastore reports five cases of pituitary reactions in his series of ninety-six postpartum haemorrhages, with one fatal case possibly due to the use of pituitary. Ergonovine is the ideal drug because of the rapidity of its action and the sustaining character of the effect. It can be given in 0.2 mg. doses intravenously. If the patient is awake, fluid extract of ergot can be given by mouth.

Postpartum haemorrhage accounts for 10 to 12 per cent. of the maternal deaths that occur every year. It is therefore a serious contributing factor to the higher maternal mortality. It is a complication that is ever before the mind of the obstetric attendant. The rapidity of its onset, its magnitude and the rapid development of the serious symptoms often overwhelm the accoucheur. The careful and orderly management of this complication and the proper use of oxytocic drugs will usually result in a favourable outcome.

PUERPERIUM.

The use of oxytocic drugs in the puerperium has enjoyed almost universal popularity. Ergot has been the therapeutic bulwark for the period. It has been credited with hastening normal involution, decreasing the likelihood of late postpartum bleeding, limiting the probable spread of intra uterine infection if present, and helping to maintain the genital organs in the best state possible.

Normal uterine involution is the physiologic process by which the puerperal uterus is rapidly reduced to about one-twentieth its size and returned to the normal non-pregnant state. Many factors may influence this important function and result in a large, boggy uterus. This subinvolution of the uterus predisposes to infection, to late postpartum bleeding and to subsequent malpositions. Davis, Adair and Pearl demonstrated the fact that improper lochial drainage as a result of lack of muscular tone often contributes

to a retardation of normal involution. Oxytocic drugs stimulate uterine contractions and tone, thereby favouring a more rapid involution.

Infection of the uterus or its appendages seriously delays involution. The relaxed uterus favours the retention of infected clots in its cavity and a spread of this infection in the genital organs and to environmental structures. A lack of uterine tone may result in an extension of infected thrombi from the vessels in the placental site. Theory and practice favour the maintenance of the uterus in a state of tone by the use of oxytocic drugs.

It has been the accepted practice at the Chicago Lying-in Hospital to prescribe ergot in the puerperium for delayed involution. Fluid extract of ergot, from 30 to 60 minims three times daily, or ergonovine maleate, from 0.2 to 0.4 mg. three times daily, can be given to patients who have had a postpartum haemorrhage, a difficult forceps delivery, intra-uterine manipulation, abnormal lochia, fever regardless of the cause, or delayed involution without cause. This therapy is kept up for at least three days or as long as is necessary; this medication should be rarely continued beyond a week or ten days, however, because of the danger of ergotism. If uterine contractions become too painful, the dose of the drug is reduced or entirely omitted. The prophylactic use of ergot to assure a normal involution is probably of some value, although further studies will be necessary to confirm this impression.

THE ABUSE OF PITUITARY

Posterior pituitary preparations have been woefully misused in the conduct of obstetric cases. It has been given indiscriminately in the first and second stages of labour to increase the intensity of uterine action and to speed up the normal processes. Normally, uterine motility is a well regulated, physiologic action resulting in steady even, though at times slow, progress of labour. However, this progress is accomplished with a minimum of danger to the birth passageway and the passenger. During the first stage the uterine contractions progressively increase in frequency, duration and intensity. However, normally this uterine activity skilfully produces effacement and dilatation of the cervix. The uterus, like all muscular organs, relaxes between contractions to allow for recovery of its musculature and for the proper continuation of an adequate placental circulation. The second stage, during which the baby is gradually pushed through the birth canal, is likewise orderly. Now the baby's head or presenting part is carefully driven against a resisting pelvic floor which must be slowly stretched to allow for the ultimate passage of the baby.

Solution of posterior pituitary upsets this carefully synchronized mechanism of labour. Violent uterine contractions initiated by oxytocics drive the baby's head against a partially effaced and dilated

cervix. Soft tissues are battered, bruised or torn. The intensity of the contractions often results in deep cervical lacerations or, when the resistance offered by these structures is too great, in ruptures of the contractile and isthmic portions of the uterus. The tetany of the uterus interferes with the placental circulation, and asphyxia or death of the baby is the inevitable consequence. In the second stage the violent uterine contractions initiated by pituitary may result in extensive, irreparable damage to the soft tissues and the pelvic floor, in brain haemorrhage in the baby. Babies are literally driven through unprepared passageways at a terrific cost to them and to their mothers. De Lee says he feels certain that the indiscriminate use of pituitary preparations is one of the four major causes responsible for the persistently high maternal and foetal death rates in the United States.

Recent years have seen the introduction of a number of oxytocic preparations in which posterior pituitary is combined with some form of thymus gland extract. This combination was heralded as the ideal oxytocic for labour, resulting in an increase in the effectiveness of the uterine contractions without the inherent tonicity and unbridled activity. Thus, they are supposed to speed up labour along its normal physiologic course. This utopian pharmacologic action so far has proved to be non-existent for these preparations represent pituitary solutions diluted with inactive thymus gland substance, retaining all the undesirable and dangerous action of pituitary. It can thus be concluded that the physician who wishes to serve womankind conscientiously and honestly must allow labour to take its normal course, rarely resorting to expedients which might hasten the process but which inevitably carry in their wake injury, mild or serious, and even death to mother and baby.

TREATMENT OF EMPYEMA.

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Phoenix, Arizona.

I know of nothing new in the treatment of empyema of the pleura. The value in talking about it lies in the emphasizing of adequate early treatment so that the illness may not be prolonged for months or years into a state of chronic empyema.

Various types of empyema occur. Tuberculous empyema I will not discuss, as it is a subject by itself, nor the empyemas due to the various types of fungus organisms. The ordinary empyema is of the

streptococcic or pneumococcic or rarely of staphylococcic etiology. It occurs most commonly following pneumonia less often following abscess of the lung, or following trauma to the chest wall.

In a typical case the recovery from pneumonia is interrupted by an exacerbation of fever and the picture of sepsis. Routine examination will usually disclose the presence of fluid in the chest, and this can readily be verified by simple aspiration.

TREATMENT

At this point aspiration is the most important treatment. Pneumonia is sometimes followed by the formation of clear, sterile, pleural fluid. This fluid may disappear following aspiration, or on subsequent aspirations it may show infection. I think in one case I may have traumatized the lung during aspiration and caused a pneumococcic empyema to develop. However, fluid may be sterile at one aspiration and infected at the next without trauma.

Aspiration should be repeated at daily or frequent intervals if the patient is febrile and the pus is thin. Some authors have reported that empyema in children may be cured by repeated aspiration. However, this has not been my experience, possible due to the fact that I shrink from repeated aspiration of small children.

The physiology of the chest is such that operation early in the formation of empyema is dangerous. Opening of the chest allows air to enter. Many a person has a mediastinum which is quite mobile. In this type of chest the addition of air, and especially free opening of the chest to the outside causes a shifting of the mediastinum toward the opposite side of the chest. This in turn embarrasses the respiration of the opposite lung and the action of the heart. Therefore, aspirations of the chest should be continued until the early thin, watery pus becomes thick and is filled with fibrin. Coincidentally with the appearance of fibrin there is an inflammatory reaction in the pleura of the mediastinum which causes the mediastinum to become fixed. It is then able to withstand the admission of air without shifting.

SURGICAL ASPECTS

At this point we are ready for operation usually removal of a small piece of rib. In my experience I believe there are three principal points of importance about operation. First the lowest point in the pleural cavity must be chosen for the drainage site. The second is that a sufficiently large tube be used for drainage. In a recent case I found that even the large-sized tube which I used became plugged with fibrin frequently and the tube had to be removed at least twice and cleaned before adequate continuous drainage was established. However, we do see cases at times which may be drained with a catheter. One such patient was a physician's wife who did not wish to have her rib cut and we inserted a tube between the ribs. This tube did not become plugged and the patient recovered

However, in the average case a large enough tube should be inserted at operation so that it will not have to be changed or cleaned out frequently in order to insure perfect drainage. Dr. Alexander recommended two tubes some years ago, one for drainage, the other an interspace above for instillation of irrigation solution.

The other point is not universally agreed upon; that is, that closed drainage should be instituted. My reason for insisting on closed drainage is that we can in this way aid the lung to expand. It is stated that the lung expands whether the drainage is closed or not, and I have had the experience of doing open drainage and having the lung expand satisfactorily. However, in those cases I have seen which did not heal and which developed a condition of chronic empyema, open drainage had been used. Since general surgeons customarily treat empyema, I have handled only about fifty cases, I believe, but in no case as yet have I failed to see the lung expand perfectly under closed drainage.

Empyema of the pleura cannot heal until the pleural space is obliterated and no area remains in which pus can form. This means that the lung must expand fully and usually become adherent to the chest wall. The parietal and visceral layers of pleura are then in apposition and no space exists in which empyema may continue.

A number of tubes are made which are suitable for establishing closed drainage. I have used for a number of years the tube of Tudoe Edwards of London. This tube has an inner flange which rests upon the parietal pleura, an outer flange which can be attached to the skin of the outer chest wall, and within the cuff, next to the drainage tube, there is a small tube inserted through which one may irrigate the chest if desired. The drainage tube is attached to a long rubber hose which passes downward by the bedside and into a jar of sterile water. The end of the tube rests beneath the level of the water and air can pass from the chest and bubble out through the water but no air can enter the tube from the outside because of the water. There is a small column of water sucked up into the tube by the negative pressure of the chest and this in turn exerts a slight suction on the air in the pleura and therefore tends to aid the lung to expand.

When closed drainage has been established I favor irrigation of the chest, at first three or four times daily and later less often with salt solution. Dakin's solution may also be used at first in small quantities, and later freely. If irrigation is carried on through a single drainage tube, one must be careful not to over-distend the empyema pocket when injecting the irrigation solution. However, I do not know that irrigation is essential. I believe the chief purpose of it is to remove more completely the pus from the pleural surfaces and to keep the fibrin well washed out so that it may not plug the tube. No doubt most cases will recover well if no irrigation

is used, provided free drainage is maintained. The temperature should come down in a day or two when drainage is well established.

COMPLICATIONS

A simple complication of empyema is the presence of a small pneumothorax, even when no abscess of the lung is present. Usually this pneumothorax disappears and the opening in the lung closes with the use of simple closed drainage. However, if the bronchial fistula persists thoracoplasty may eventually be necessary to close the empyema cavity. If abscess is present, drainage or lobectomy may need to be done.

Chronic empyema occurs when the lung has not been fully re-expanded during drainage of the empyema. One of the first cases which I saw was that of a woman about 25 years old who had had pneumonia followed by empyema 19 years before at the age of six. She was treated by tube drainage but evidently not completely healed, for in the intervening nineteen years she had had six additional operations for the empyema which kept recurring. However, her chest wall was healed. She had been aspirated repeatedly during the two or three months prior to my seeing her. Were I to see this case again I should attempt closed drainage, for it might have been possible even then to re-expand the lung sufficiently so that the visceral pleura would have become adherent to the parietal pleura. I think that many cases of chronic empyema might be treated in this way and more extensive operation avoided. However, in this case (and eventually in many cases the treatment would be the same) I removed the two or three ribs immediately over the pus pocket together with the parietal pleura, periosteum, and intercostal bundles, leaving the pocket entirely exposed to the air. The skin was sewn to the edges of the parietal pleura where it had been cut. In a few weeks this pocket filled in by granulation tissue and eventually was covered by skin. The patient recovered entirely. However, this woman had a great deal of illness during nineteen years and was left with a chest wall defect because of the fact that the empyema was not completely drained and the lung expanded originally.

A man 50 years of age gave a history of empyema at the age of 18, which was treated by rib resection and either open or tube drainage. His chest wall healed after a period of time, but he was never in good health afterwards. About a year prior to the time I saw him he began to have periods of fever followed by cough and expectoration of a large amount of purulent and greenish sputum. This would stop and after a few weeks or less he would again have fever with pain from which he would recover after a few days of cough and expectoration. At the time I saw him he had developed a superficial abscess in the left lower anterior chest wall. This was opened and bromopin injected. There was extensive deformity of

the chest which I presume was caused by the long standing pleural infection which he had. There was a chronic empyema cavity present and portrayed by the bromopin. This man also was treated by resection of ribs overlying his empyema cavity and by the removal of sheaths of calcified parietal pleura in addition to the periosteum and intercostal bundles.

A boy 25 years old with a history of empyema at the age of 15, gave a similar history. This boy was cured by unroofing of the empyema cavity and the granulating in of his pleural pocket. In this case I attempted to cure the condition by instituting closed drainage, but a small bronchial fistula was present which prevented the lung from expanding. The fistula healed after the empyema pocket was unroofed.

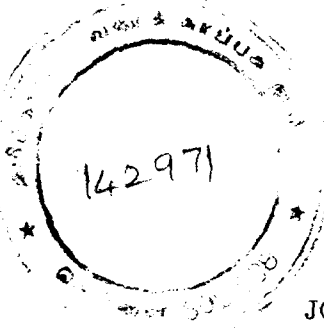
If abscess is present the patient will not recover until the abscess is drained. A case in point was a man 47 years old who had pneumonia sixteen months before I saw him. Rib resection was done for empyema after the pneumonia, the tube being in about four months, but after the tube was removed and the chest healed, he was aspirated repeatedly. A year later he had a severe hemorrhage and continued to expectorate blood at intervals during the following month or two. At the time I saw him I felt he had a chronic empyema and operated with the expectation of finding this. However, he had also an abscess of the lung. This was drained and recovery was without incident.

I could cite many similar cases of chronic empyema. The period following the original empyema varies from a few months to in one case thirty-two years. These chronic empyemas point ever so strongly to the need for early adequate drainage of acute empyemas of the pleura and re-expansion of the undiseased lung.

SUMMARY

1. The treatment of non-tuberculous empyema is discussed.
2. The drainage site must be at the lowest point in the pleural cavity.
3. A large enough tube must be used to insure continuous drainage.
4. Closed drainage is to be preferred.
5. Irrigation is of some value.
6. Chronic empyema occurs when the lung has not been fully re-expanded during drainage of the acute empyema.
7. Chronic empyema may be cured by "unroofing" if bronchial fistula is not present.
8. Chronic empyema should be avoided by proper drainage of acute empyemas and re-expansion of the underlying lung.

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ENTERITIS.

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Pickneyville, ill.

Enteritis may be defined as inflammatory disease of the small intestine. It may or may not be accompanied by colitis, duodenitis, cholecystitis, or gastric pathology. It may be acute or chronic.

The etiology of chronic enteritis, which we shall discuss, is not entirely settled. It may, however, be:

1. Mechanical: as by irritating foods or ingested substances such as bran.
2. Chemical:
 - (a) hematogenous, as by mercury which is excreted in part through the small intestine;
 - (b) direct, by drugs and cathartics which act on the small bowel following gastroenterostomy, where acid contents of the stomach are thrown directly into the jejunum.
3. Bacterial:
 - (a) specific, such as typhoid bacillus in the ileum, also vibrios not rare.
 - (b) non-specific, which occurs in achylia duodenitis, following gastro-enterostomy.

Probably enteritis always descends from above except when associated with a chronic colitis. It descends in passive congestion where the small bowel is readily infected or irritated.

Morbid anatomy: In newer cases there is hypertrophy of the mucosa, with increase in size and number of mucous glands. Hyperemia of mucosa is also present. Later there is a thinning of the bowel wall, with atrophy of the mucous membrane and involvement of the serosa.

Symptoms: 1. Subjective. The symptomatology of chronic enteritis is confusing, inasmuch as it may simulate gastritis, peptic ulcer, gall-bladder disease, chronic colitis without diarrhea, or appendicitis. Many patients who suffer from this disease are grouped as "nervous dyspeptics" or as hypochondriacs. Often there are "heartburns," occasionally chronic unexplainable vomiting.

Pain is sometimes present, coming on after meals as in peptic ulcers. It is usually radiating to the left hypochondrium, is colicky in nature, and is much shorter in duration than ulcer pains. It is very infrequent for this pain, however, to be relieved by alkalies, as is ulcer pain.

A feeling of "unquietness" in the abdomen, due to the speed of passage of food through an irritated small intestine and to the violence of the peristaltic waves, is often complained of.

Flatulence is an almost universal phenomenon. This does not occur in the small bowel, but in the colon, and is due to the fermentation of the large amounts of undigested starch that are thrown into the cecum.

Finally, there is an indescribable feeling of nervousness after food, most often after the evening meal. There may be sweating, hot flashes, vertigo, a feeling of extreme weakness, or even fainting due to the mesenteric engorgement (so-called abdominal apoplexy). These cases have been misdiagnosed epilepsy very frequently and account for some of the "cures" which occur spontaneously in this disease. Often, when a patient comes in with an abdominal complaint he will inform us his appendix is sore, or that he has pain in the region of the liver. Not so with the sufferer from enteritis. His pains are indefinitely located and flitting, although they may at times be severe. He is nervous, his heart pounds so he must walk the floor at night; he feels he is going to die when the gas crowds his heart. Never does he come in saying his small gut is out of kilter.

2. Objective: The most important objective symptom of enteritis is the presence of a tender area to the left of the umbilicus, often midway between umbilicus and costal margin, about two inches to the left of the midline. The patient is usually thin.

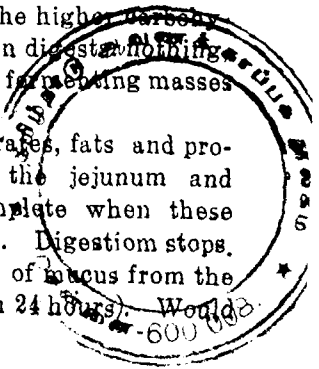
The ileo-cecal region may also be tender, as a result of the inflammation about the cecum. It must be remembered in enteritis as in all other bowel infections that there is no deep tenderness unless, by direct extension or extension through the lymphatics of the bowel wall, the peritoneum is involved.

Most important are the laboratory findings. The x-ray gives but little information as to the small bowel except in obstructions. In enteritis there is usually observed a rapid emptying of the stomach, with hyper-peristalsis, and barium taken after a full meal is frequently present in the colon within two hours after ingestion. The x-ray is not necessary to establish a diagnosis, but may be of value in ruling out gall-bladder or stomach pathology.

The feces examination is of the utmost importance. In order to see why, let us briefly review our physiology of the small bowel.

Excepting the small amount of absorption which takes place in the stomach, all digestion of fats, proteins and the higher carbohydrates takes place in the small bowel. The colon does nothing but is an excellent incubator for the putrefying and fermenting masses which enter at the cecum.

In chronic enteritis the passage of carbohydrates, fats and proteins (in the form of meat fibers) through the jejunum and ileum has been very rapid. Digestion is not complete when these half-dissolved particles are thrown into the cecum. Digestion stops. With the food materials is an enormous amount of mucus from the ileum (estimated in severe cases at three quarts in 24 hours). Would



we expect mucus and undigested starch granules in the feces? Not unless there was an accompanying colitis with rapid passage through the colon. Due to the fact that the colon holds this undigested material for 12 to 36 hours, the mucus and meat fibers putrefy, the starch ferments, causing the gas mentioned under symptomology and the sole remaining pathological finding in the feces is the enormous increase in fat globules, fatty acid crystals and soaps.

The feces may be whitish; they are nearly always light in color due to the soaps. The microscope may show many starch granules, especially if the colon has also been irritable and emptied before the fermentation had completed. Under these circumstances the meat fibers may also be present, but are often square-cut, instead of presenting the rounded appearance of a piece of melting ice.

The diagnosis, therefore, is established on the basis of the fecal content.

The blood and urine are not of necessity changed by chronic enteritis. While, at times, a cecitis may cause an increase in leucocyte count to 9 or 10,000, there is little rise due to the enteritis itself.

Complications. The most frequent complication of enteritis is, of course, inflammation of the cecum, followed by colitis. This is often heralded by repeated attacks of mild diarrhea.

Appendectomy frequently complicates the picture about this stage, due to the tenderness about the cecum. The patient has digestive symptoms, loss of weight, and a hundred? Presto! Out comes his appendix, in spite of his low blood count. Ten days in bed, with starvation diet for the first three, makes him feel fine. In a month or two he is back, with his symptoms sufficiently altered that he never realizes his operation has been useless. Appendectomy never cures these cases, and is sometimes very embarrassing to the surgeon.

Finally, after the lapse of months or years, a true colitis with diarrhea will probably supervene. When this happens, we no longer are dealing with enteritis, but with enterocolitis.

Due to the scant time for absorption hypochromic anemias are frequent complicators of these cases, especially in cases associated with achylia. These may be mis-diagnosed pernicious anemia, which they greatly resemble. The color index will make the distinction.

Hunter's glossitis is frequent in chronic enteritis, and is not necessarily associated with achylia, as is generally understood. Fever may be present, with the other toxic symptoms, leading to diagnosis of tuberculosis. They may have backaches, leg pains and other toxic symptoms. Chronic urticaria is not infrequent, due to the increased permeability of the injured intestinal wall to proteins.

Gall-stones may be induced by entry of germs or toxins through the portal circulation. At any rate, many chronic sufferers from

enteritis develop gallstones after a number of years. This is well known to be true with typhoid; why not in non-specific infections of the bowel?

Differential Diagnosis. 1. Appendicitis. Low blood-count, tenderness to left of umbilicus, with hyperesthesia of abdominal wall at that point. Chronicity, pains colicky and transient.

2. Peptic ulcer may have pains very similar, but again we have the relief by alkalies in ulcer and the greater tenacity of ulcer pains.

3. Cholecystitis may present a more difficult problem, but the location of tenderness, the X-ray findings and the stools will make diagnosis possible.

Treatment. 1. Dietetic.

Diet must be one to give the inflamed small intestine rest. As in all chronic diseases, there must be no period of starvation or starvation diet.

Cellulose in any form must be prohibited, not because of the mechanical irritation but because of the chemical one. We have no cellulose-splitting enzymes, but the fermentative bacteria in the large bowel have, and will form large quantities of gas with the undigested fibers of cabbage, beans, corn, etc.

When achylia is also present we forbid potatoes and peas. Meat should be taken very sparingly; should be boiled or baked, and must be lean.

No spices of any sort are permitted.

Patient can eat milk, cream, cheese, potatoes, porridge, tea, coffee, cocoa, juices of fruits and vegetables, and various pastries. In severe cases only mashed and liquid food may be taken but this diet may not be continued for long.

The diet closely follows Dr. Alvarez' smooth diet

2. So far as medicines are concerned, it is very important not to use laxatives, since they can only pour fuel on the fire. They always irritate.

If constipation is troublesome, milk is laxative without irritation, due to the lactose present. Better yet is Beta-lactose, which does not ferment with the production of gas. A table-spoonful before meals will often aid very much in establishing normal defecation. Fruit juices are also of benefit.

Mineral oil may also be used, especially if the astringent drugs are prescribed.

Astringents: (a) Tannigen, or other forms of acetyltannic acid, are of value when treatment is first started, in dosage of five to ten grains before meals of about two weeks. (b) Bismuth subcarbonate or subgallate are useful if there be hyperchlorhydria.

Absorbents, such as charcoal, are useless, since the gas in the bowel is in the colon, and the wet charcoal is incapable of absorbing gas.

Antiseptics are also useless and in many cases irritating. They can only do harm.

Vaccines have not been shown to be of value in uncomplicated enteritis.

Diet, then, is the method of treatment which gives most chance of cure. It must be carefully followed for many months, since backsets are as frequent as in colitis, and cure cannot be certain for several months after the cessation of all symptoms.

(*Illinois Medical Journal*, April 1938.)

Extract.

TREATMENT OF SIMPLE VAGINITIS.

The fact that leucorrhoea may have a simple aetiological background, writes L. Goldstein, in "Med. Record" for May 5, 1937, is likely to be overlooked in the undue emphasis on the trichomonas and monilia as causative agents; the majority of patients complaining of leucorrhoea who present themselves to the physician show no pathological manifestation other than a simple vaginitis.

The normal vaginal secretion is bluish white in colour, resembles in appearance and consistence ordinary skimmed milk, and possesses an acid reaction. This reaction has been attributed to the presence of the vaginal bacilli of Doderlein, but the glyco-genic activity of the vaginal epithelium may be an important factor. In inflammatory conditions of the vagina, there is usually an associated disturbance in the metabolic and chemical activity of the cells.

The normal secretion of the cervical mucous membrane is colourless and only assumes a whitish or greenish hue when it comes in contact with the vaginal secretion. Abnormal processes in the cervix and vagina usually manifest themselves by an over-productive amount of secretion which produces the chief symptom—leucorrhoea. The type of leucorrhoea depends upon its primary cause. That is, in simple vaginitis the discharge is usually watery, thin, or catarrhal or suppurative in character. The discharge of gonorrhoeal infection is usually profuse, thick and pyogenic. The discharge of trichomonas vaginitis manifests itself as a profuse, bubbly, free yellowish creamy discharge possessing an offensive odour. In carcinoma, involving either the cervix or vagina, the discharge is of a serosanguineous nature, and possesses an offensive odour.

The symptom of leucorrhoea is encountered in many patients who are free of pathological processes in the pelvic organs, gonorrhoea, and parasitic invasion (monilia and trichomonas), and in whom no definite cause for the vaginitis can be uncovered. In a number of these patients the vaginitis is associated with some degree of erosion of the cervix.

Various types of bacteria are encountered in the vaginal tract in nonspecific vaginitis. In addition to the normal Doderlein's bacilli, the vaginal flora consists of many kinds of organisms such as diphtheroids, enterococci, gram negative diplococci and coliform bacilli. Almost always a profuse and purulent discharge is associated with this kind of flora. In vaginitis of mild to moderate severity, Doderlein's bacilli are usually present along with other bacteria.

It is difficult to determine the underlying organic basis of vaginitis in cases where the trichomonas and monilia are absent, and no abnormalities in the pelvic organs are present. Excluding general conditions such as hyperthyroidism, cardiac disease and debilitating diseases which may be associated with the symptoms of leucorrhoea, local irritations induced by douches, chemicals, antiseptics, etc., may be the causative factors.

No medication should be applied to the vagina without first making a search for the cause. In obscure cases of irritative vaginitis, mild antiseptic applications of preparations containing boric acid and glycerine are beneficial. Forty-seven patients with simple non-gonorrhoeal vaginitis were successfully treated with applications of a simple preparation consisting of boric acid 2 per cent., glycerine 14.8 per cent., and oxyquinolin sulphate 0.02 per cent. The medicament was incorporated in a water-dispersible jelly in a vegetable gum base. Routine vaginal and cervical smears were taken on all patients under observation. The vaginal tract was first cleansed with a solution of tincture of green soap and water and dried. About 2 drachms of the preparation were applied to the entire vaginal vault in one treatment.

The patient usually received two or three treatments a week. In the ordinary case of vaginitis, three to five treatments bring about such a degree of relief that the patient is inclined not to report for further treatment. As improvement occurs, the number of Doderlein's bacilli increases in the bacterial flora whereas the number of other strains of organisms becomes markedly reduced. There is also a noticeable reduction in the number of pus cells.

The preparation of boric acid and glycerine can be easily applied to all the vaginal surfaces, and can easily be removed with water. The lubricant feature of such a preparation tends to allay the irritation and discomfort of these patients, and assists materially in reducing the amount of discharge. This method of treatment

has the advantage over douches in that the antiseptic preparation remains in contact with the inflamed vaginal surfaces until removed.

MORPHINE AS AN AID IN DIAGNOSING ACUTE ABDOMINAL AFFECTIONS.

Harry A. Singer, writing in "The American Journal of Surgery," October, 1936, says that it is universally taught that morphine should not be administered to a patient with an acute abdominal affection until the diagnosis has been established or at least until a decision as to whether or not to operate has been reached. The objection to the drug during the diagnostic period is that the narcotic "masks" the clinical picture. As a matter of fact the only sign, symptom or laboratory test which is affected by the morphine is the pain and its related manifestations, principally rigidity and hyperaesthesia.

By virtue of the fact that the alkaloid relieves or moderates pain and removes thereby, in part at least, the abdominal wall resistance and the hyperaesthesia, morphine is often most helpful in the diagnosis of acute abdominal affections. When the suffering of the patient is great, a trustworthy and adequate history cannot be obtained without first employing a narcotic to assuage the agony. When the physical signs are difficult to elicit or are equivocal the administration of morphine permits a much more satisfactory examination. After its administration a small or moderate size mass in the presence of diffuse rigidity accompanied by hyperaesthesia can be made out; the point of localized tenderness can be more accurately determined; that portion of the abdominal wall which is most rigid can be determined; with relief of the spontaneous pain the hyperaesthesia; is more or less removed permitting the examiner to determine the true degree of tenderness which is proportionate, other things being equal; to the severity of the inflammation present.

A disadvantage of the method is admitted by the author to be danger that a patient may be deceived by relief from pain. For this reason he thinks that care should be taken to apprise the patient of the selective effect of morphine on the pain and of its failure to influence the course of the disease. Another objection which might be raised against the use of morphine in the pre-diagnosis period is in the cases of intestinal obstruction.

•The procedure of the author is as follows: "The ordinary case is handled in the customary manner. If, however, the

patient is unable to co-operate in the taking of the history or in the making of the physical examination on account of the severity of the pain, a sketchy history is obtained and a preliminary examination made and the observations recorded. Morphine grains $\frac{1}{4}$, or its equivalent, dilaudid grains 1.20, is injected intravenously. The effect is very prompt and striking. An adequate history is now obtained and a complete, careful physical examination can be done. Observations made before and after morphine can then be compared."

In addition to aiding the examiner in arriving at a correct interpretation of symptoms, morphine administered early in the diagnosis period spares the patient the unnecessary suffering and renders him a better operative risk. Morphine is conceded to be one of the most effective therapeutic agents in counteracting the effects of "shock" haemorrhage and also peritonitis. From what has been said it is quite clear that the author has not deprived himself and his patients with acute abdominal affections of the benefits of morphine, and in the words of Moynihan: "Many of us have been frightened of the drug, quite needlessly, for properly used its value is beyond question."

RAW FRUIT TREATMENT OF ACUTE DIARRHOEA.

Th. Baumann of the Paediatric Clinic, Univ. of Basel, in "Hippokrates," No. 35, 1937, says that the raw fruit treatment with apples and bananas is based on the three pillars of every therapeutic diet: one sided nourishment, hunger-diet, and finest dispersion, as with all acid diets, i.e. large surface on which the digestive ferments can work, so that the digestive process proceeds without any difficulty. To this must also be added the great capacity of the pectins of raw fruit to swell up and absorb, as a result of which the alimentary canal is mechanically cleansed, bacteria and toxins adsorbed. On the other hand, the tannic acid content of the raw fruits and the acidity produced by the apple-diet are quite secondary in importance. Cooked fruits do not act curatively, but rather make the intestinal disease more severe, because the active form of pectin is destroyed by cooking.

During the first nine months of life a diet of raw fruits offers no advantages as compared to the other common, adequate, therapeutic diet; it may even be harmful since the pectins, and especially the raw fibres, irritate the very delicate intestinal mucous membrane. This also holds for Bircher-Benner's "fruit-milk equivalent to

human milk." Starting with the tenth month, however raw fruit diet is decidedly superior to all therapeutic foods ("protein milk," butter milk, lactic acid milk) for the infant it is far better than the ordinary purging and subsequent gruel zwieback diet or a diet rich in proteins. The only exceptions are chronic dyspepsias of a constitutional type. Here, too, one attains good raw fruit stools, but even with careful shifting to a therapeutic diet richer in proteins relapses occur. Herter Heubner's disease reacts best to bananas and a subsequent curative diet of proteins. The duration of attacks of colitis membranacea or eosinophile intestinal catarrh based on a constitutional disturbance is shortened by raw fruits from 10-14 days to 4-5 days. For cases of acute and subacute, febrile or afebrile gastroenterocolitis of older children 3-4 raw fruit-days are successful with typhoid or paratyphoid fever.

Bananas are to be preferred to apples because they satisfy the appetite much better and because they are richer in salt and calories (45 to 60 cal). Besides this bananas are preferred by the infant; they produce firm stool in a shorter time. On a banana diet one hardly loses weight, if at all. The nitrogen balance is much more rarely negative; the mineral balance is partly even positive. Apple diet is always followed by more or less sudden, intense loss of weight accompanied by a certain hunger-acidosis due to its low caloric value and content in salt. Only the poverty in salts, and with it the precipitate loss in weight, can be combated by administering Noll's combination of 200 gm. apple in 500 gm. tea and 200 gm. Ringer's solution.

TECHNIQUE OF THE BANANA CURE.

After a tea-pause of 6-12 hours (4 tablets of sweetening substance per litre tea), the infant is given 5 to 8 bananas daily in 5 portions. After removing the peel the banana is beaten with a fork until it is quite foamy or well whisked and given to the child with a spoon. A little tea may be given after this. This diet is maintained exclusively for 3-4 days. Then the child is given $\frac{1}{2}$ litre whole milk with 2-3 beaten bananas, for 2-3 days, and then full diet in milder cases; in more severe cases, lactic or citric acid milk (1.5 gm. acid citr. to $\frac{1}{2}$ litre milk) and 2-3 bananas, then gradual introduction of full diet, but avoiding fat; with severest cases one continues to give 4-5 bananas daily and gradually introduces sour milk. Infants are given 6-8-10 bananas a day with as much black tea as they desire; after 3-4 days suddenly go over to normal diet. In severe cases only is it necessary to resort to sour milk and bananas, perhaps also curd as a transitory food. At home the infant may be given rice-water and zwieback with bananas after the pure banana-days, and then full diet. For older children the bananas can be mixed with well grated apples free of skin and core. Failures are due to allowing even the smallest quantities of gruel, flour, zwieback, and the

like, to be added during the 3 or 4 days when an exclusive raw fruit diet has to be maintained.

AUTOHAEMOTHERAPY IN PSORIASIS AND HERPES ZOSTER.

In two cases of psoriasis and seven of herpes zoster, C. E. Barksdale (*Virginia Med. Monthly*, 64, Oct 1937, p. 378) withdrew 6 or 8 c.c. of blood from the cubital vein and injected it immediately into the gluteal muscle before it had time to clot. The two cases of psoriasis responded well to a routine consisting of autohaemotherapy, anthralin ointment locally and a diet rich in milk and butter.

Autohaemotherapy not only shortens the duration of pain of herpes zoster, but also shortens the course of the disease. It is as good as any other form of therapy that has been used in the past, if not a specific, in the treatment of herpes zoster. The majority of patients gave a history of the absence of milk or butter from their diet. Patients with psoriasis were advised to partake of a diet rich in milk and butter, supplemented by carotene in oil or cod-liver oil, the local application of anthralin ointment, which is chrysarobin derivative, ultra-violet radiation and autohaemotherapy.

("Medical World".)

GUAIACOL INTRAVENOUSLY IN LUNG ABSCESS.

Nammack and Tiber (*J. A. M. A.*, July 31, 1937, p. 330) report on the successful results obtained by the intravenous use of guaiacol in 20 cases of acute and chronic lung abscess.

A solution of from 5 to 10 grains of guaiacol, 2 c.c. of ethyl alcohol and 18 c.c. of water containing $2\frac{1}{2}$ grains of sodium iodide is employed every third day. The fever soon subsides, the cough is relieved, the sputum gets less in amount and loses its foul odour, until finally the patient is cured both clinically and roentgenologically. There were only four deaths in this series—one due to malignant condition of the lung, another to traumatic subdural haemorrhage and two due to recurrence of the abscess in persons who refused treatment for their infected gums.

The authors maintain that eradication of all foci of infection about the mouth, nose and throat and moderate restriction of all activities during this course of treatment are absolutely essential.

for success. As surgery cannot be tried before six to twelve weeks have elapsed, and as guaiacol treatment produces such pronounced effects without any unfavourable reactions, the latter is certainly worthy of trial in cases of acute and chronic lung abscess.

("Medical World")

Medical News & Notes.

THE TINNEVELLY DISTRICT MEDICAL ASSOCIATION, PALAMCOTTAH.

(Tinnevelly Branch of the Indian Medical Association).

The monthly meeting of the Tinnevelly District Medical Association was held at the Subramanya Karayalar Public Library, Palamcottah, on Saturday the 24th of September 1938. Members numbering 58, including two women doctors were present on the occasion. Sri K. P. Yegneswara Sarma, M.A., M.L.A., was present at the meeting on special invitation. After Tea and sweet music the meeting began under the Presidentship of Lieut. Col. T. S. Shastry, I.M.S. The Secretary and Treasurer read out the Minutes of "President's function" of the Association held on the 21st of August 1938, which were passed. He also read the minutes of the Special meeting held to meet Lieut. Col. H. E. Shortt, M.B., I.M.S., the Acting Surgeon-General with the Government of Madras, which were passed.

The Secretary read the letter received from the Honorary Secretary, Indian Medical Association, Calcutta, regarding the inclusion of the Licentiates within the purview of the Indian Medical Council Act, Maintenance of the register, etc. Sri. K. P. Yegneswara Sarma, M.A. M.L.A., promised to bring the subject before the next Legislative Assembly meeting.

The Secretary, read the letter received from the Honorary Secretary, The Indian Medical Association, Calcutta, regarding the fees for examining candidates for Life Insurance Companies. After discussion by the members the following resolution of Dr. L. Mahadevan, M.B.B.S., D.O.M.S. was passed unanimously, having been duly recorded:—

"It is hereby solemnly resolved that no member of the Tinnevelly Branch of the Indian Medical Association shall examine any case for Life Insurance unless he is paid a fee of Rs. 10 (as minimum, and of Rs. 16 if the Blood Pressure is to be taken) (exclusive of any expenses of travel if it is outside his consulting room) for the medical examination, whatever the amount of Insurance may be".

It was also resolved to enlist the co-operation of the neighbouring District Medical Associations in this very important matter, and then to finally decide it at another meeting.

The following resolutions of the Managing Committee were discussed:—

1. Amending Rule No. 19 as follows:—

To have the power to alter the constitution at any one of its meetings after previous notice has been given where not less than one half of the number of members on the roll are present and three-fourths of the number present are in favour of the proposed change in the constitution.

New Rules:—

2. Members whose subscription is in arrears for six months shall cease to be members, but they continue to be liable to pay the arrears.
3. Members who have lost their membership under the above Bye-law shall not be re-admitted unless they pay all the arrears due from them.
4. A non-residential membership shall be instituted as follows:

"It is resolved that the Association may institute a non-residential membership for the benefit of those members of our Association who get transferred or who have to move out of our District in the course of the Journal year (1st October to 30th September following) after having paid their subscription for the year.

 - (2) This non-residential membership applies only to such members as are residing and working in a District which has not branch of the Indian Medical Association.
 - (3) It is proposed that the Annual subscription for the non-residential members should be Rupee one for the membership plus a Journal subscription of Rupees three per annum for the Journal. But since he has already paid Rupee one towards the Journal he should pay only Rupees Two for it for the current year.
 - (4) Before he leaves the District he should pay all the subscription and other dues due to the Association; and when he applies for non-residential membership he should also pay in advance the subscription of Rupees three for the current year.
 - (5) For the next year onwards he should pay Rupees Four in advance. If it is not paid in advance his name shall be removed from the list.
 - (6) His application should be accepted by the Managing Committee and he should be enrolled as a non-residential member, before he gets the benefit of it, and

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