

The Antiseptic

A MONTHLY MEDICAL JOURNAL

Edited by

DR. U. RAMA RAU & U. KRISHNA RAU, M.B., B.S.

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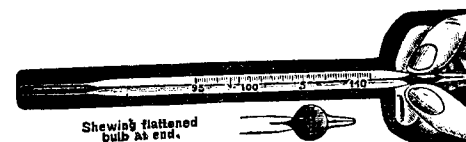
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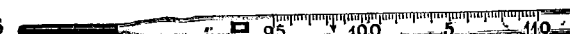
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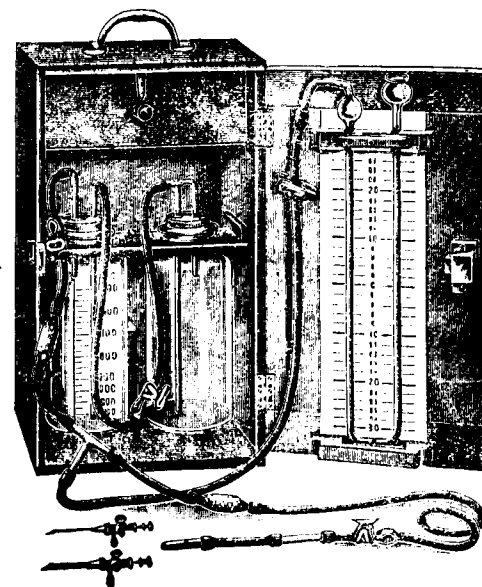
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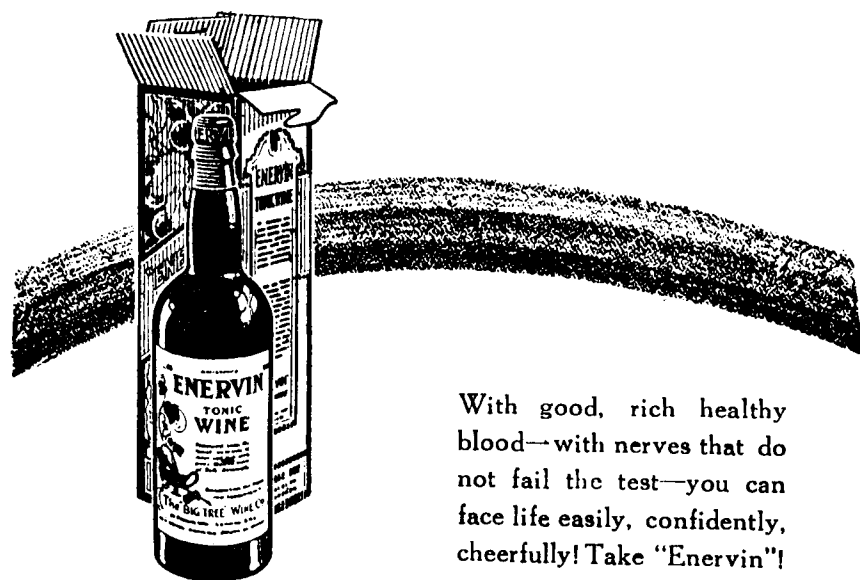
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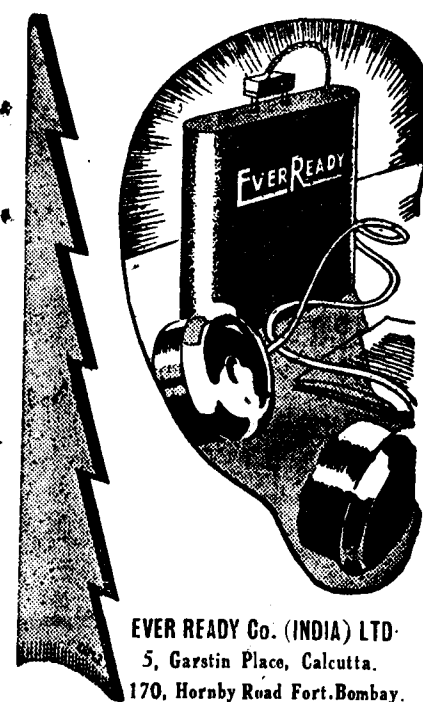
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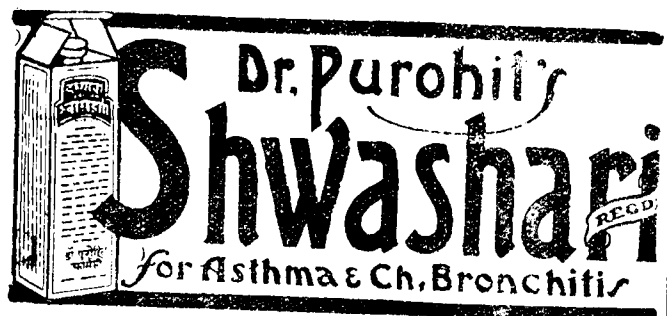
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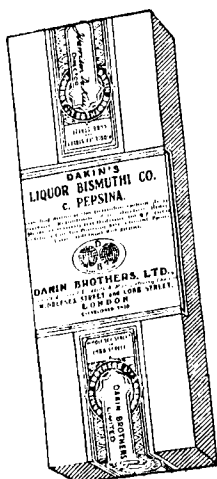
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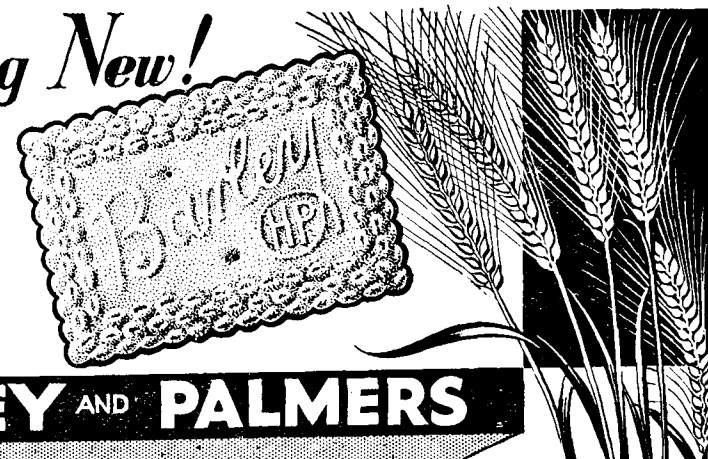
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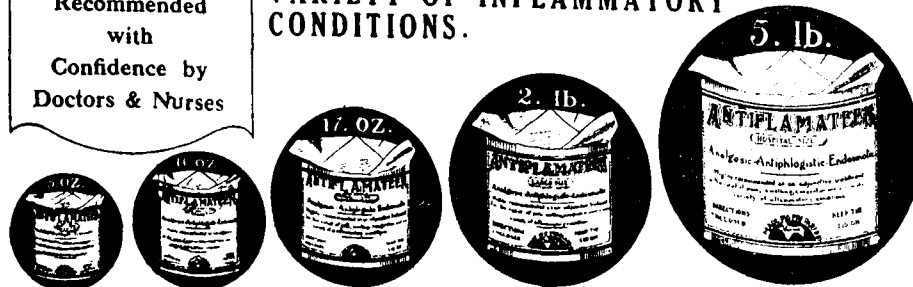
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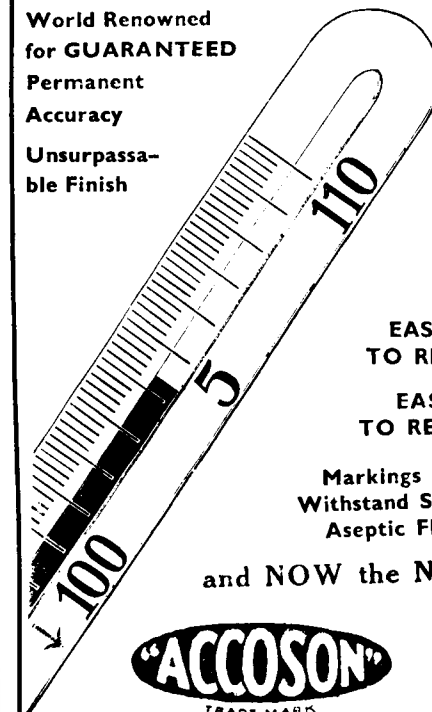
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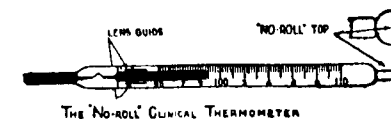
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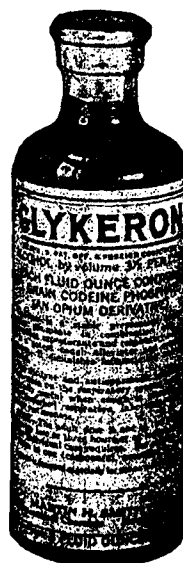
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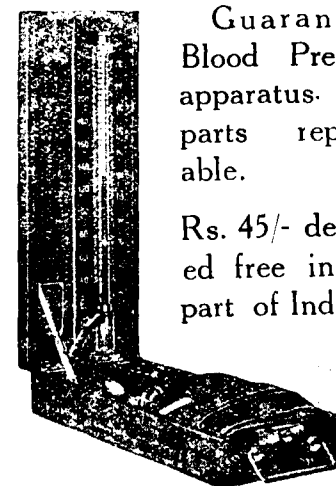
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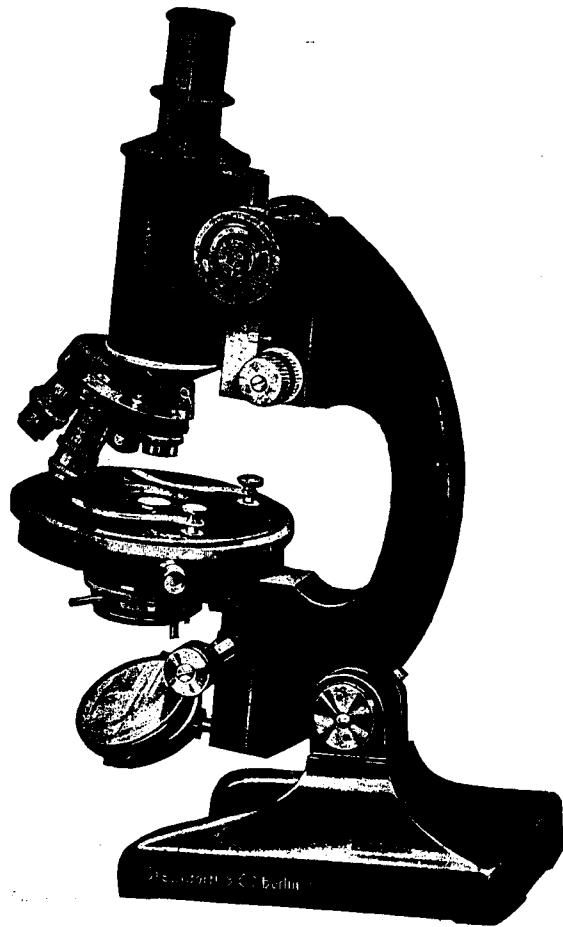
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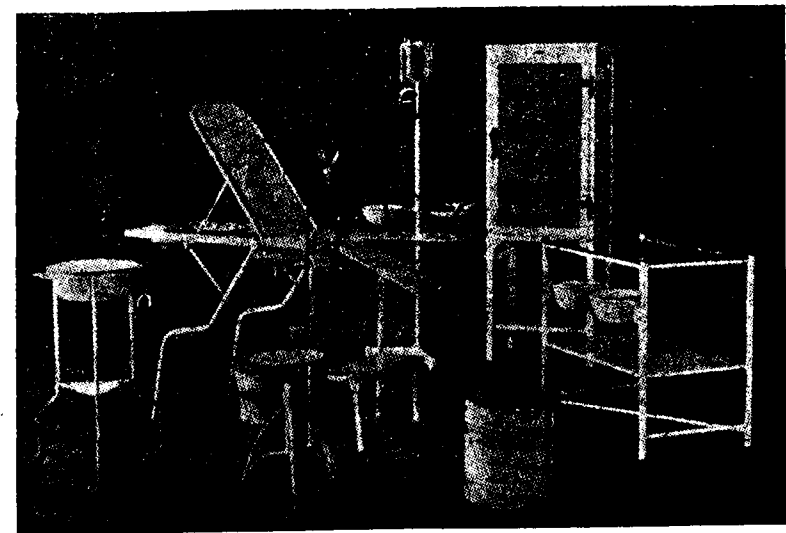
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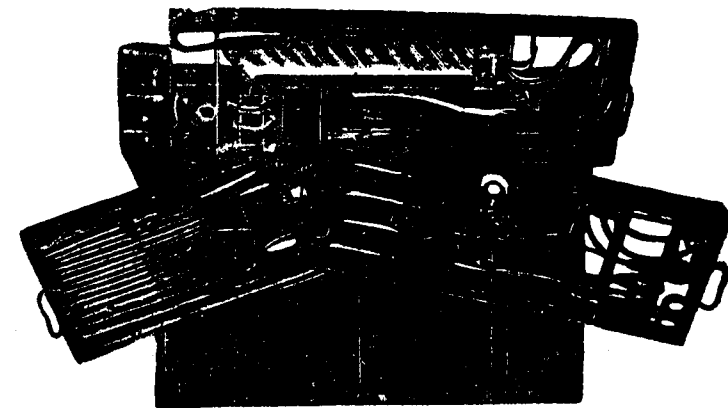
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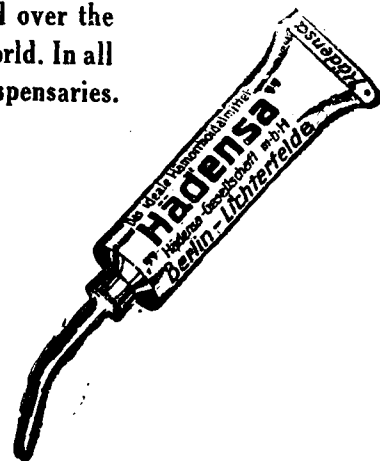
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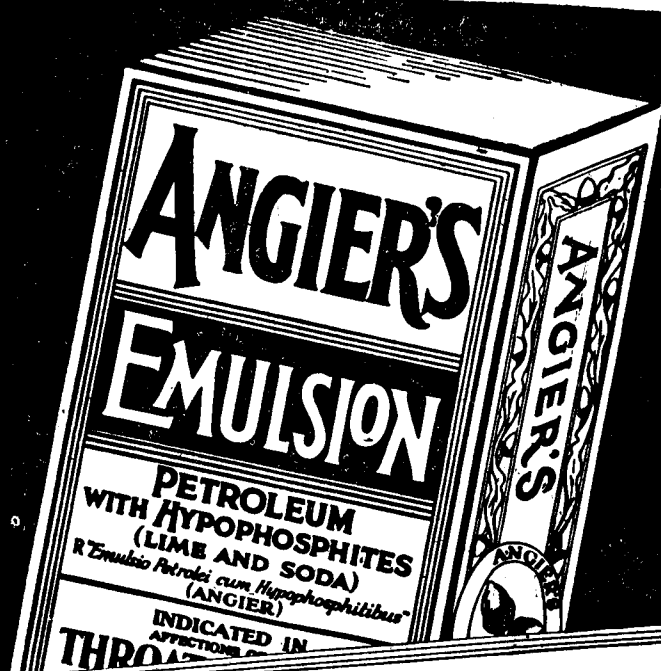
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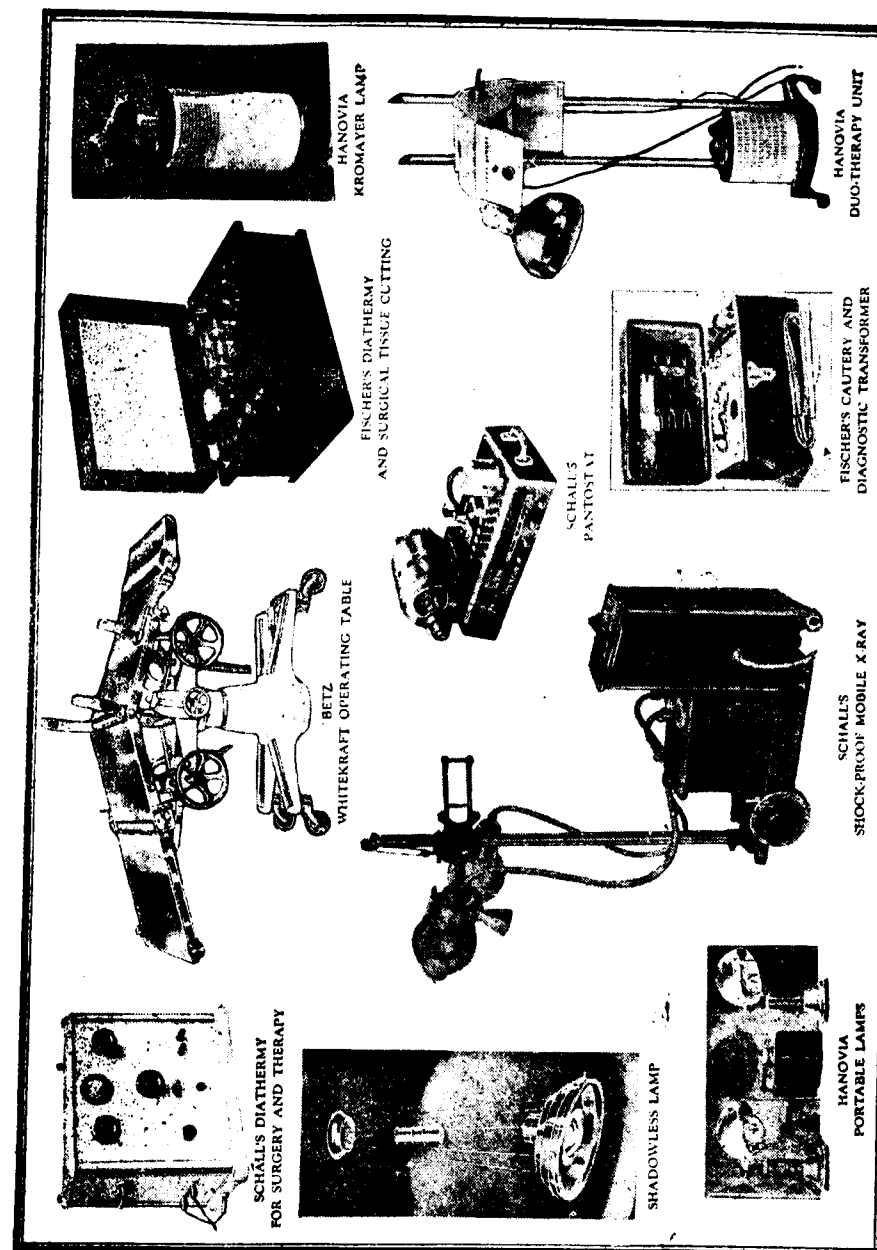
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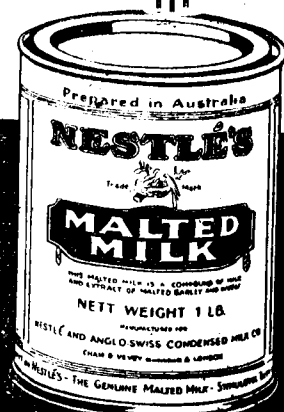
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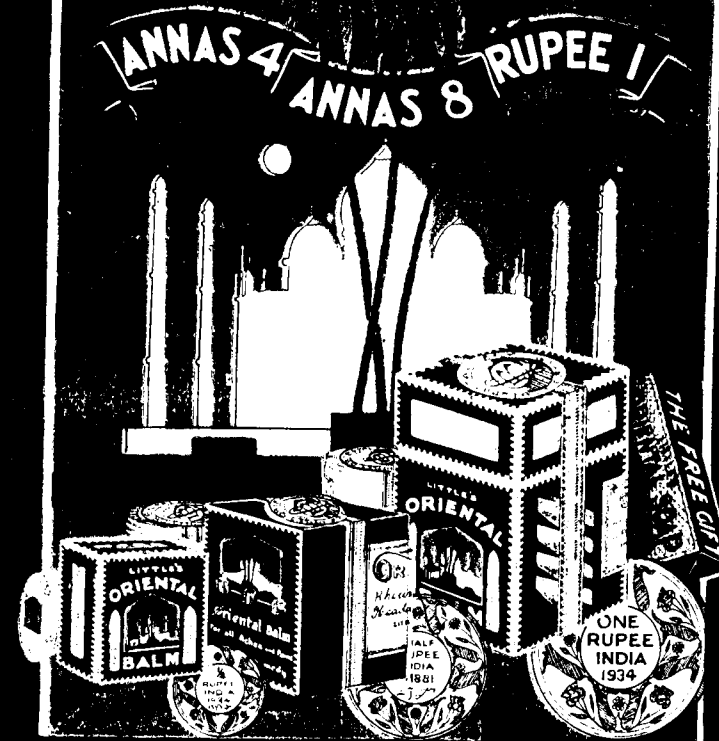
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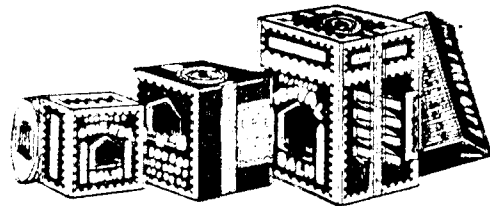
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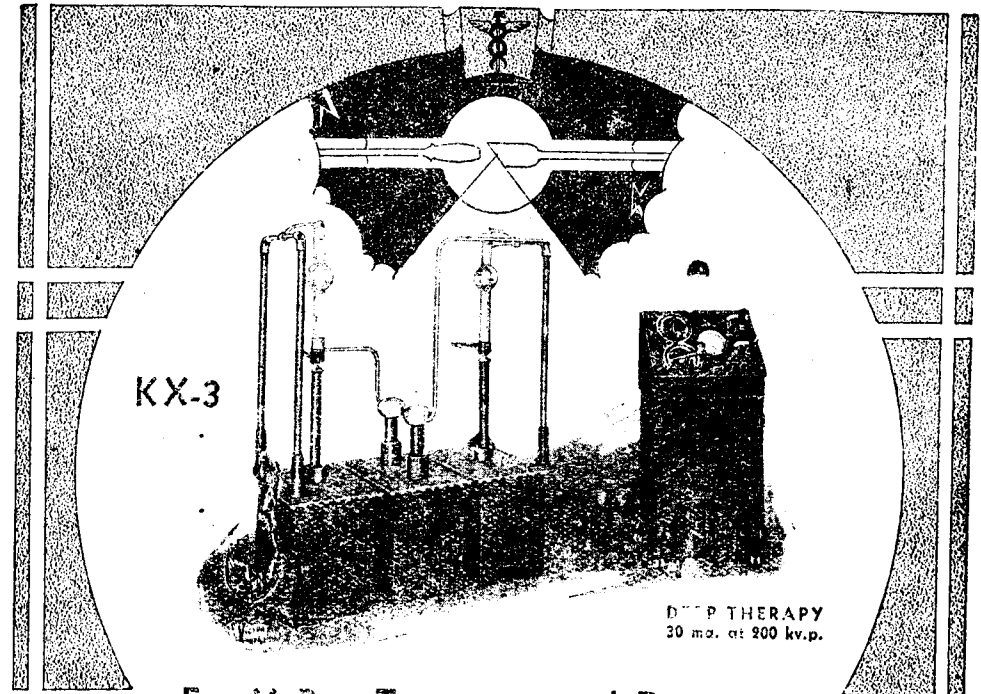
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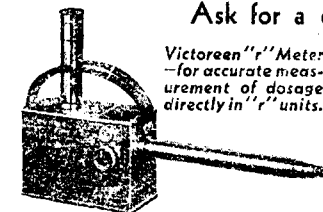
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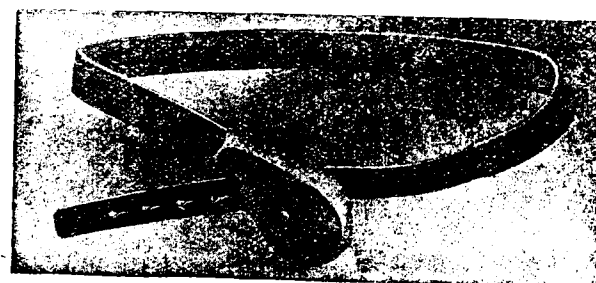
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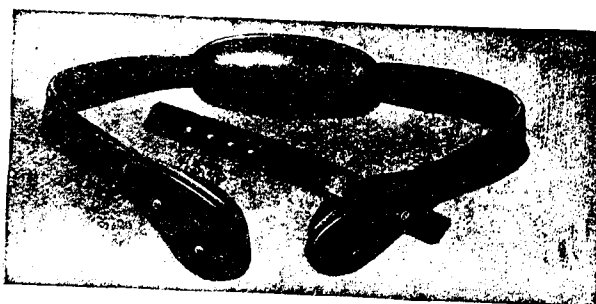
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INGUINAL HERNIA AND HERNIOPLASTY

BY

DR. N. MANGESH RAO, M.B., C.M., F.R.C.S., (E.),

Surgeon, Govt., General Hospital, and Lecturer in Principles and Practice of Surgery, Medical College, Madras.

Historical:—Hernia is as old as the human race. It was known to Egyptians as early as 3000 B.C. and was treated, by bandaging. The first operation was done by Celsus in the first century A.D., of ligating and excising the sac. The popular name of "rupture" for this disease was apparently due to the view expressed by Galen that hernia was due to a rupture of peritoneum.

A greater advance in the knowledge of this condition took place in the 18th century through the work of Littre, Richter, Campor and Gimbernaut and in the 19th century Cooper and Scarpa added to the knowledge of the surgical anatomy of the part.

Czerny introduced the method of high ligation of sac through the external abdominal ring with closure of the pillars snugly round the cord by sutures. This became the standard operation till the twentieth century. But recurrence was 30% or more after this operation. Opening of external oblique for high ligation was introduced by Lucas Champoniere in 1883. Bassini introduced his method of hernioplasty in 1882-84. This has now become the standard method of treatment. But recurrences have induced surgeons to modify the methods in various ways. In 1887

Halstead placed the cord subcutaneously with plastic suturing of the muscles of the canal behind the cord. It was given up but has been reintroduced with modifications for repair of direct hernia. In 1895 E. W. Andrews introduced a modification of Bassini operation called the "Inbrication or lap-method". Coley and Ferguson introduced the method of suturing the conjoined tendon to inguinal ligament in front of the cord. Halstead performed a similar operation in which he reduced the size of the cord.

Mac Arthur in 1901 suggested the use of live fascial sutures, using strips of fascia from the aponeurosis of external oblique. Gallie and LeMesurier introduced the method of using strips of fascia lata for the repair of the inguinal canal in 1921. This method has become most useful in the repair of large and long standing oblique hernia, direct and recurrent herniae and has displaced the filigree operation.

1. **The truss.**—In 1306 Gordon introduced the iron truss and in 1628 Le Quin introduced the steel truss. The English cross-body truss was introduced in 1806. Since then several modifications and improvements have been introduced.

Three chief types of hernia are noticed in connection with inguinal canal:—the oblique, the direct and interstitial herniae.

The oblique inguinal hernia:—In this the sac emerges through the internal abdominal ring lateral to the deep epigastric artery and lies in front of the cord and is covered by all the coverings of the cord. After traversing the canal it emerges through the external ring and descends downwards into the scrotum. According to the length and position of the sac in relation to the inguinal canal and neck of scrotum three clinical degrees can be recognised.

Bubonocoele or incomplete hernia in which the hernia is entirely within the canal. The complete type in which the hernia just emerges through the external ring. The Inguino-scrotal type in which the hernia has passed down into the scrotum.

2. **Direct Hernia:**—In this the sac, always an acquired one emerges through the inner part of the inguinal canal medial to the deep epigastric vessels. It stretches out the transversalis fascia and conjoined tendon in front of it. In about 10% the sac may emerge through gaps in the transversalis fascia. The sac always lies behind the cord. After emerging through the external ring, it seldom or less frequently becomes scrotal, but more often rises towards the pubic region.

3. **Interstitial hernia:**—In certain cases of oblique inguinal herniae there is an irregularity of the sac due to the development of a lateral diverticulum or a complete congenital displacement of the entire sac into the planes of the abdominal wall. They are usually seen in arrested descent of testis or its maldevelopment. These herniae are rare.

4. **Sliding hernia:**

Frequency:—In males 95% of all herniae are of inguinal type and of these 70 to 80% are oblique and 30—20% are of direct type. In women 45 to 50% of herniae are inguinal (Watson), 98% of which are oblique and only 2% of direct type. The sex ratio for the oblique type is 10 males to 1 female. Keith says that 4.4 % of male children have oblique herniae in the first year of life and two-thirds of these are cured spontaneously or by a simple truss (Bull). About 1—2% of male adults in the third decade (20—30) suffer from oblique herniae.

It is more common on the right side than on the left in the ratio of 3 : 2. This is probably due to later descent of testis and later closure of processus vaginalis in the male and the canal of Nuck in the female. In the male oblique inguinal hernia shows a tendency to become bilateral in 25% of sufferers and in women about 20%.

Etiological factors:—1. *The persistence of a patent funicular process* as a whole or in the proximal part in males and the canal of Nuck in women provides a potential hernial sac. Keith found a patent funicular process in 30% of infants up to four months after birth and in 12 % of adults. Russell found it in 10% of adults. In women the canal of Nuck was found open in 10% (Nobbe). Sachs found a patent funicular process in 29% of male adults and a patent canal of Nuck in 25% of adult women. It is generally accepted now that 90 % of oblique herniae have congenital defect in the obliteration of the funicular process. In most cases developing in young adult life there is usually a tiny sac which in course of time enlarges at the expense of abdominal peritoneum.

Though the patency of this process is about equal in both the sexes, the ratio of incidence of hernia is 10 males to 1 female. Hence other factors must influence this condition. These are:—(1) The processus-vaginalis is wider and longer in males than in females. (2) Both the rings and inguinal canal are much wider in men owing to the descent of testis. (3) The drag on the patent funicular process is in opposite directions in the two sexes. In the male the increase in size, weight, and mobility of the

dependent testis exerts a pull on the patent process in an inward and forward direction. In women this process is adherent to the round ligament which is subjected to a backward pull towards the pelvis by the uterus. (Hitzrot). The incidence of oblique inguinal hernia in the two sexes is more even before puberty.

2. *Inadequacy of inguinal sphincter surrounding the cord.*—According to Keith, the conjoined tendon acts like a shutter coming down against the fixed inguinal ligament and closes the canal snugly round the cord during abdominal strain. Hence a weakness in this tendon either congenital or acquired permits a weak point for the development of a hernia. Relaxation of the tendon occurs when the thigh is slightly flexed or in stooping postures. Under these conditions repeated sudden increased intra-abdominal tension is liable to force out an enlarging cone-like process of peritoneum into the canal. The presence of any small cone of peritoneum at the proximal end of canal (a congenital sac) favours formation of hernia. Conjoined tendon may be weakened by starvation, wasting diseases, fatty degeneration or atrophied by a previous operation. Damage to its nerve supply as after a MacBurney or Davis' incision may also weaken the tendon. A greater development of cremaster muscle in the male is at the expense of conjoint tendon.

3. Presence of subserous lipomata in the inguinal canal is liable to drag down a cone like process of peritoneum. These are often seen in stout individuals. Weakness of the shutter-action is also caused by growth of these lipomata.

4. Increased intra-abdominal pressure would force its contents through gaps in the abdominal wall or points of weakness. A pre-existing sac or weakness of inguinal canal or both would at once favour the formation of a hernia. This increased intra-abdominal pressure may be brought about by chronic cough, straining in chronic constipation and urinary obstruction, by intra-abdominal tumours, or by increase of intra-abdominal fat, etc.

5. *Heredity*:—According to Eccles 25 % of cases give a family history of hernia. This probably consists of an inherited weakness of the conjoined tendon and an incomplete obliteration of the funicular process.

6. *Occupations* subjecting one to severe abdominal strains in stooping posture as in stokers, coal-heavers, porters, etc., are liable to cause hernia. In the stooping position the conjoined tendon gets slightly relaxed and hence cannot completely close the canal and if the intra-abdominal pressure is increased

repeatedly, a gradually increasing peritoneal cone is liable to be forced through the internal ring.

7. *Age*:—In the first two decades of life hernia is usually of the indirect type and due to presence of a patent congenital sac. In the third decade new hernia formation is not common. But from fourth decade onwards the incidence increases and is 50 % more common than in the first decades. This is partly due to weakening of the conjoined tendon from age, increase of fat, subserous lipomata and partly due to causes, that induce repeated increased intra-abdominal pressure such as urethral strictures, enlarged prostate, chronic constipation, etc.

Direct Hernia

In this the sac presents through the inner part of inguinal canal and neck of sac lies medial to the deep epigastric vessels. It stretches the transversalis fascia and conjoined tendon or more often slips under a poorly formed conjoined tendon and finally projects through the external ring but shows little tendency to descend into the scrotum. In about 10 % of cases the hernia may push through gaps in the transversalis fascia. 25 to 30 % of herniae belong to this type and 98 % of all direct herniae occur in males. They occur from fourth decade onwards in life. 50 % of recurrent herniae belong to this type and are usually due to atrophy of conjoined tendon. 60 % of direct herniae are liable to be bilateral sooner or later. They are prone to recurrence in 30—50 % of cases after operation.

Anatomy:—Hesselbach's triangle is bounded laterally by deep epigastric vessels, medially by the lateral edge of rectus abdominis for a distance of 3 to 5 centimeters in people with poorly formed conjoined tendon and below by the inguinal ligament. Where the conjoined tendon is well formed, its arching fibres cover the inner two-thirds of the space. But where it is poorly-formed, only the transversalis fascia and extra-peritoneal fat intervene between the peritoneum and the aponeurosis of external oblique. The obliterated hypogastric artery crosses this space and may be found stretched over the sac. More often a direct hernia projects at the lower inner angle of this space, near the margin of the rectus. Where the conjoined tendon is atrophied by the existence of a large hernia or destroyed by a previous operation, the gap near the outer edge of rectus may be as wide as 7 cms., the lower fibres of internal oblique and transversalis being inserted into the rectal sheath 7 c. ms. above the pubic crest. The floor of this triangular space is formed by transversalis fascia which is

well formed in the upper and medial part but thins out below. In the upper part it is reinforced by arching fibres passing from medial to lateral side. The lower central part of the floor is weakest and it is here that hernia first begins to bulge out.

1. **Causes:**—A congenital or acquired deficiency of conjoined tendon and the presence of a large external ring favour the production of a direct hernia. A well formed cremasteric muscle is always at the expense of the conjoined tendon, and the external ring has to be wider to accommodate for a thicker cord. The conjoined tendon is also damaged by previous operation and more than 50 % of recurrent herniae are of direct type owing to this damage.

2. Increase of both intra-abdominal and sub-peritoneal fat favours the formation of a direct hernia. According to Cloquet fatty masses project through gaps in transversalis fascia caused by blood vessels penetrating through it. The cord also is liable to be loaded with fat thereby widening the external ring.

This hernia first appears as a bulge in the inguinal triangle, stretches the transversalis fascia, then usually slips below the conjoined tendon. It next meets the aponeurosis of external oblique and projects upwards over the internal oblique. But if the external ring is large, it projects through it and ascends upwards showing little tendency to descend into the scrotum. The sac is globular or dome shaped and situated on the inner side of inguinal canal just above the pubic bone. Often the bladder is adherent to its inner wall and sometimes the colon on the outer side in herniae of left side. In about 25 % of these cases there is also a small indirect sac giving rise to a "pantaloön or saddle-back" sac made of two components, a small one lateral to the deep epigastric vessels and the larger sac medial to them. In a large sac the obliterated hypogastric artery is found stretched across the sac. In a few cases a large indirect sac may be accompanied by a small direct sac. In about 10 % of cases narrow necked sacs may project through gaps in the transversalis fascia. It is this type of direct sac that is liable to slip through the external ring early and is liable to incarceration and strangulation. It resembles very much an indirect hernia. Direct herniae rarely grow to large size and rarely get strangulated. Only about 2.5 % of strangulated herniae belong to this type. Patients with direct hernia often complain of indigestion and constipation but these are not relieved by operation. The bulge of a direct hernia subsides suddenly on recumbency and appears as suddenly on standing. The bulge appears on straining even in the recumbent

posture, but this does not occur in an indirect hernia till the internal ring has become wide. The expansile impulse is more evident and communicated to the entire sac at once in a direct hernia. If the little finger is introduced well into the canal and with pulp directed to the floor, one may appreciate the laxness of the floor and deficiency of conjoined tendon in direct herniae. On coughing the impulse is felt by the pulp in the floor in direct type and by the tip of the finger in the indirect type.

Interstitial Herniae

These occur with retained testis or undescended testis. There is an absence of cremaster and the abdominal rings may be superimposed. The external abdominal ring may be partially obliterated. They are very liable to strangulation and the lateral sacs may be missed during a radical operation. This type should be suspected when the testis is absent from scrotum and a bulge is noticed lateral to or above the internal ring. According to frequency the following varieties may occur.

- (1) A patent processus vaginalis is present in the scrotum with lateral diverticulum lying between.
 - (a) the peritoneum and transversalis fascia (Pro-peritoneal).
 - (b) sac lying between internal and external oblique muscles. Sac between transversalis and internal oblique muscles is rare. This type is called inter-parietal.
 - (c) Sac lying between transversalis fascia and muscles.
 - (d) Sac lying between the peritoneum and iliac fascia-retro-peritoneal.

2. Processus vaginalis absent from the scrotum and sac is found between external oblique and the skin (pre-parietal), or between the peritoneum and iliac fascia. Often these lateral diverticula may be missed unless looked for. However these types are rare

Sliding or Sacless Herniæ

The caecum on the right and the sigmoid on the left slide down preceded by the iliac peritoneum. They commonly occur as direct and less commonly as indirect hernia. At first a small sac may be present but as the hernia enlarges the peritoneum is displaced upwards and bowel slides down behind it. Finally a small sac is found high up on the anterior aspect, the bowel forming the

posterior and inferior wall. These most often occur in people with visceroptosis. Some are due to congenital deficiency in the endoabdominal fascia which fixes the different organs to the posterior abdominal wall. In some, elongation of their mesentery may be a cause. These herniae are liable to become irreducible and may get incarcerated. They are a difficult problem for operative treatment. They are rare.

Treatment of Oblique Hernia.—In infants and children under 18 months, 65% of herniae are cured spontaneously or by the use of a simple truss. Hence any exciting cause as phymosis, anal stenosis etc., should be dealt with and a truss tried. After the age of 10 years, hernia cannot be cured by trusses and requires operative treatment for cure. Herniae occurring with undescended testis should always be treated for both conditions by operation as early as possible. After the age of 60, operative treatment except for strangulation or obstruction is inadvisable. These should be prescribed trusses. Long standing large inguino-scrotal herniae are best left alone. These have such large openings that strangulation is rare. They are generally irreducible from adhesions and the abdominal cavity is unable to accommodate all the hernial contents and respiratory difficulty and distension are likely to follow if operation is done.

After puberty an oblique inguinal hernia is best treated by operation. It is always liable to strangulation and efficient surgical aid may not be available on the spot. This liability is greater in manual workers. If left alone, it is certain to increase in size and the conjoint tendon undergoes atrophy from pressure and then an elaborate hernioplasty would be required. "A stitch in time saves nine" is a true saying in this connection.

The following important principles should be observed in the operative treatment.

- (1) A free exposure of inguinal canal by slitting the external oblique and the pubic attachments of conjoint tendon and inguinal ligament should be well defined.
- (2) The sac should be freed right up its neck and the peritoneum all round it and especially on the medial aspect of the canal should be tightened by twisting the sac before its ligation and excision.
- (3) Where the conjoint tendon is poorly formed or atrophied an adequate plastic repair should be done.
- (4) Avoid leaving any weak points in the floor of the triangle, especially at its medial end.

- (5) So that the union of fascia with muscle may be firm, it is essential to free them well of all areolar tissue by gauze dissection.
- (6) In such union no tension should occur and hence fascial darning after the method of Gallie should be resorted to if the conjoint tendon cannot be brought down to inguinal ligament without tension.
- (7) Since at least 21 days would be required for firm union to take place, catgut should be chromicized or efficiently tanned and size 1 should be used. Linen thread or silk may be used as knots are fine, but "shooting" of sutures is liable to take place. Kangaroo tendon is too thick for the purpose and if used should be of fine quality. Living fascia is best and should be autoplasmic, the ideal one is fascia lata. I must mention here that a doubt has been cast about the union of muscle fibres with elastic tissue, on the basis of animal experiments. But other Surgeons affirm that firm fibrous union can take place, if muscles and fasciae are completely denuded of all areolar tissue. But in this recurrences though small—4—5 % do occur. Gallie reports excellent results without recurrence by the use of fascia lata suture. These living fasciae are not absorbed and do not stretch and remain as a permanent support for the weakened canal.

In young adults and children where the sac is of congenital nature and hernia is a small one and conjoint tendon is adequate it is enough if the sac is removed after high ligation and tightening of peritoneum. If conjoint tendon is slightly weak, then a suture at the medial end between the conjoint tendon and insertion of inguinal ligament is enough. The transversalis fascia has to be tightened in some by pleating, if found lax. The internal ring should be closed by suture snugly round the cord, if found to be large. Similarly the external ring should be snugly closed round the cord.

In complete oblique herniae of some standing Andrew's modification of Bassini should be done. If the cord is found to be large the cremaster muscle should be excised in addition.

The Ferguson-Coley Operation:—In this the conjoint tendon is sutured to the inguinal ligament in front of the cord. This operation is indicated in herniae with undescended testis of

children, in complete herniæ of children and in oblique herniæ of women.

Downes' modification of Andrews' operation:—This operation is of use in large oblique herniæ and some of the direct herniæ. It is also indicated for the pantaloon type of sac. In this the cremaster is excised and size of the cord is reduced. The fibrous part of the transversalis fascia is brought down and sutured to inguinal ligament and the internal ring is also snugly closed round the cord. The conjoined tendon is sutured to inguinal ligament. Then over-lapping of the two leaves of external oblique is done in front of the cord on the lateral side and behind the cord on the medial side. For recurrent hernia and large direct hernia Gallie's method should be employed. In this, strips of fascia lata are used in the form of basket work between the inguinal ligament and conjoined tendon and the rest of operation is as in Andrew's method.

I might stress here the point that the operation has to vary according to conditions found in the canal after it has been adequately exposed. There is no set method that could cure hernia. It is necessary for one to note if any exciting causes are present in such cases as phimosis, stricture, etc., and remove these causes before the operation is undertaken. The whole aim should be to reduce the chance of recurrence for the sake of the patient as well as the Surgeon's reputation.

Recurrence:—Andrews says that Bassini's operation gives 25 % recurrences, 2.5 % only for fascial repairs. Gallie says that with his method, he has had no recurrence. Max Page—his recurrences for 5 years were 11.5 % in the Metropolitan Police force.

Indications for prevention of recurrence :

- (1) Early and adequate operation before the conjoined tendon gets atrophied.
- (2) Use of fascial transplants as sutures for large herniæ with poor conjoined tendon.
- (3) Avoid tension when suturing the conjoined tendon to inguinal ligament.
- (4) Suture material used should not be absorbed in less than three weeks.
- (5) Absolute asepsis and dryness of wound, all bleeding points being ligatured.
- (6) Sufficiently prolonged convalescence—3 weeks in bed, 3 weeks on a couch. Return to work only after three months. Lifting weights and straining to be avoided for six months.

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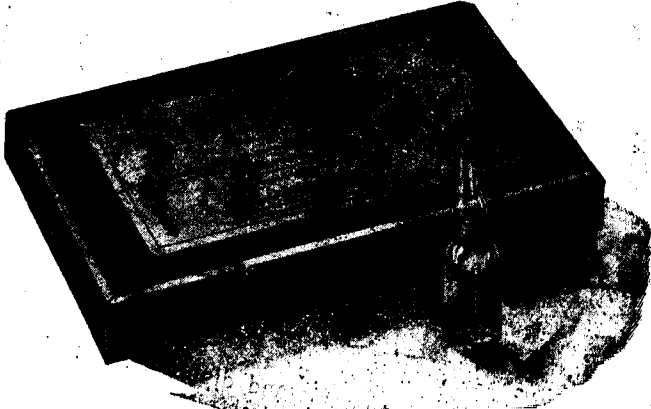
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MODERN VIEWS ON CHRONIC LOBAR AND INTERSTITIAL MASTITIS OR CYSTIC MASTITIS

BY

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Histology of Breast:—The acini and ducts are lined by a layer of epithelium that becomes continuous with the epithelium covering the nipple and skin. The terminations of ducts are lined by transitional epithelium which becomes squamous as it reaches the epidermis. The ducts and ampullae are lined by a single layer of columnar epithelium which ends abruptly at the beginning of acini. The acini are lined by several layers of cuboidal epithelium lying on a single layer of unstriated muscle fibres that are continued round the ducts. Outside this is a layer of fine connective tissue with a fine fibrillous matrix with a few cells scattered in it. This sub-epithelial connective tissue layer is continued round the ducts also. Outside this the ducts are surrounded by "lamina elastica" which usually terminates abruptly at the acini. Occasionally some acini or a lobule may be surrounded by a single layer of elastica. At the outer end this elastica becomes continuous with that of the skin. Outside this the larger and medium sized ducts have a coat of unstriated muscle fibres which may end in a delicate sphincter under the nipple and surrounding the ducts. Outside this, surrounding the ducts and acini, is a layer of loose connective tissue "the peri-acinous and peri-canalicular connective tissue" quite different from the supporting connective tissue of the breast which is more fibrous and coarse. This periacinous and peri-canalicular connective tissue increases in amount during normal breast activity. It is the source of fibro-adenomas in young adults. It is coarser than sup-epithelial connective tissue, and may be absent in senile breasts and in virgin breasts after forty. Outside this is a dense coarsely fibrous supporting connective tissue which separates the lobules from one another. This layer is surrounded

by fat of varying amounts upto two and a half centimeters thickness. This fat is intersected by ligamenta suspensoria, which arise from the supporting connective tissue and after branching are inserted into the connective tissue of the skin.

Physiological hyperplasia of breasts.—In the new-born columns of epithelium may be seen dipping into the subcutaneous fat to form the future ducts and acini and also an increase of periacino-canalicular connective tissue. The epithelium may show desquamative hyperplasia of epithelium resembling that of chronic mastitis of later life. Similar activity on a larger scale is seen at puberty with desquamative epithelial hyperplasia of ducts and acini and a hyperplasia of periacino-canalicular connective tissue. During menstruation slight hyperplasias are also noticed. During pregnancy a greater hyperplasia of epithelium and connective tissue is seen. The epithelial one may be of desquamative type. During lactation desquamative hyperplasia is very active consisting of budding and acini-formation at the expense of periacino-canalicular connective tissue. But if the breasts are not suckled increase of connective tissue with diminution of acini occurs.

Involution of breast.—After lactation is over, the breast resumes a state of rest but never returns to the normal of a virgin breast. In some parts the atrophy of epithelium and connective tissue is so great that it may resemble a virgin breast and in other parts the connective tissue may remain hyperplastic and in some others a mild desquamative epithelial hyperplasia may be seen.

Chronic Mastitis.—Both these conditions of chronic and lobular mastitis are the result of desquamative epithelial hyperplasia. Chronic lobular mastitis now called Mazoplasia by Lenthal Cheatle, is more a physiological process than an inflammatory one and is distinct from the cystic mastitis which is a pathological condition. Desquamative epithelial hyperplasia ends in shedding of the superficial cells which die in the process of shedding and remain as a debris in the acini and ducts. In mazoplasia no other change is seen. But in cystic mastitis an additional change of cyst-formation occurs and hence named Cystiphorous Desquamative Hyperplasia by Lenthal Cheatle. Further changes of neoplastic type may occur in this type. Hence the importance of separating these two conditions.

Chronic lobular mastitis or Mazoplasia:—In this desquamation of epithelial cells occurs in the terminal ducts and acini with hyperplasia of epithelium and peri-acino-canalicular connective tissue. Often the new formation of ducts and acini may occur. The shed epithelium may distend the ducts and acini giving rise to diffuse

pain and if there be little or no subcutaneous fat, a generalized fine nodularity is felt on palpation. In some cases a slight diffuse hyperplasia of elastica and in others a slight diffuse or localized hyperplasia of subepithelial connective tissue may also occur. This biological change does not end in cyst-formation, papilloma or carcinoma. But occasionally fibro-adenoma may form. Mazoplasia is almost universally present in varying degrees in all breasts of women that have borne children and to some extent in all normal breasts till menopause. The worst cases are seen in married women. Ovarian hypo-function is usual and is associated with excessive corpus luteum stimulation with hypo-ovarian function. Epithelial proliferation is more marked during proliferation of corpus luteum in the premenstrual stage. Mazoplasia may sometimes be seen in men between the ages of 30 to 45, the breasts showing slight enlargement with pain. This type resembles that found in infants at birth and changes seen at puberty. The epithelial layers may be two or three, the desquamated cells are desiccated, small and irregular in shape and stain poorly.

The age period varies but the highest incidence is between 30 to 40. It disappears after menopause.

Signs and symptoms:—Diffuse aching pain in the whole breast on one or both sides, becoming worse during menstruation and increased by excessive use of arms or jarring of breasts, is the chief complaint. Pain may subside spontaneously on the advent of pregnancy. In others it may be intermittent till menopause is reached. Ovarian hypo-function is shown by short periods with scanty flow. Neither retraction of nipple, nor change in the contour of breasts is seen. Breasts never become fixed to pectoral muscle, nor is there any spontaneous intermittent discharge from the nipple. But artificial suction occasionally gives a greenish fluid. On palpation a diffuse fine nodularity may be felt in breasts devoid of fat. The whole of the breast gland may be felt uniformly solid, or more pronounced in a particular area but without any defined margins. The posterior surface is smoother and free from nodules. The fine nodularity of the anterior surface is due to hyperplasia of peri-acino-canalicular connective tissue extensions into bases of suspensory ligaments. A coarser nodularity may be felt in breasts that have large amounts of fat especially in pendulous breasts and is due to subcutaneous fat being isolated into lobules by the suspensory ligaments becoming denser in pendulous breasts. A coarse nodularity in thin people with atrophic breasts is usually due to a diffuse

carcinoma spreading into bases of suspensory ligaments. This is often accompanied by puckering of skin, retraction of nipple and enlargement of axillary glands. These are never seen in *mezoplasia*. A diffuse increased density of the gland is present to transillumination.

The pain is always generalized and breast is tender. It may be unilateral or bilateral. Intermittent pain and variations in size are seen in normal breasts due to menstruation or emotional states.

Prognosis:—*Mazoplasia* is common and may be present for several years without any change. The only tumour that may occasionally form is fibro-adenoma. It never gives rise to cysts, papilloma or carcinoma. If these be present they are really independent formations with no connection with *mazoplasia*.

Treatment:—Local treatment consists of support to the breast without pressure and in bad cases the arm should be put in a sling. Radiant heat and diathermy are useful. Constitutional treatment consists of correction of any pathological conditions in pelvic viscera. Pregnancy cures the condition. Sexual habits should be inquired into and corrected. Ovarian residue consisting of follicular and interstitial substances should be given grs. 3—5 thrice a day during the latter half of menstrual cycle.

Cystiphorous Desquamative Hyperplasia of Cheate.—Chronic interstitial mastitis, Reclus disease, chronic cystic mastitis of Schimmelbusch and senile hypertrophy of Bloodgood, all of these belong to the same condition, now more appropriately called by the above name by Sir Lenthal Cheate.

This is a more serious condition usually starting about the age of 25 as a desquamative epithelial hyperplasia ending in the formation of multiple cysts during the age period of 25 to 35. It may show no further change in good many, but in others may progress to the second stage of benign neoplasia at the age period of 35 to 45. It may stop at this or in some progress on to carcinoma at the age period of 45 to 55. 20 % of carcinomata have an origin like this.

Cystiphorous Stage:—This condition was first described by Reclus in 1883 and consists of a generalized condition of multiple cysts of both breasts simultaneously or consecutively. In this stage there is hyperplasia of epithelium of the smaller ducts and acini with desquamation of dead cells. The condition may affect the whole breast or only one duct with its branches and acini. The change affects most often the terminal ducts and acini, sometimes the smaller ducts alone may be involved. But the

changes seen in the ducts differ from those occurring in the acini.

In the Ducts:—The changes are usually limited to the smaller and terminal ducts; though the larger ducts may show distension due to retained collection of fluid. In the small terminal ducts epithelial hyperplasia begins by the cells becoming long and feathery containing lightly staining elongated nuclei and clear cytoplasm. Among these cells and in the lumen may be seen colostrum-like cells of varying sizes and in all stages of development. These are formed only from duct epithelium and are large, rounded and appear swollen. Cytoplasm is abundant and clear with a small nucleus in the centre. Being desquamated cells they stain poorly. The tips of the feathery cells may occasionally coalesce enclosing the colostrum-like cells in their meshes.

In the Acini:—The cells become more cuboidal but stain poorly being pale in appearance and hence called "Pale Cells". The nuclei are hypo-chromatic and central, the cytoplasm is homogeneous and clear. The pale cells form two or three layers from which columns of three or four cells project into the lumen. Desquamation occurs from the superficial layers of these columns or lining wall of acini. These desquamated cells are irregular with poorly staining nuclei and other signs of degeneration. As the cysts enlarge, the walls separating adjacent cysts disappear resulting in larger composite cysts. These large cysts are lined by degenerated flattened epithelium which may even be absent in places when distension is great, leaving the underlying connective tissue bare. There are the blue domed cysts of Bloodgood. The cut surface of such a breast may show some large cysts, some smaller ones and innumerable tiny cysts, many of which are microscopic and dilated patent ducts. Small papillomata may often be seen in the acini and some may show a fibrillous core indicating that the second stage of neoplastic change is setting in. The subepithelial connective tissue usually shows a slight diffuse or localized hyperplasia. Both the pericanalicular and periacinous connective tissue show a slight hyperplasia with or without lymphocytic infiltration. The elastica may show in places a compact or loose hyperplasia and in others it may be normal or absent.

Mechanism of Cyst-formation: Cysts are due to obstruction with some inflammation as in Galactoceles, or desquamative hyperplasia causing a mechanical distension of acini and ducts, or a combination of these two-obstruction with desquamative hyperplasia.

The fact that no obstruction is seen in the ducts but only

convolutions and that the acini are not dilated before changes in the smaller ducts have taken place goes against obstruction theory. The dilatation of larger ducts is only a mechanical distension without any epithelial hyperplasia. The fluid in the cysts is not inflammatory as it does neither coagulate nor form fibrin. It is thin and clear, sometimes thick and greenish, sometimes creamy white suggestive of a secretory origin. Stagnation of contents occurs and this leads to hyperplasia of connective tissue. Mere stagnation may induce no other change. But if the causative agent persists in the fluid it induces after sometime neoplastic changes. If decomposition of the contents also occurs, the onset of neoplastic change is earlier. Stagnation is more common in the terminal ducts and acini and neoplastic change also is more common here. As soon as this change has set in, the second stage of the disease occurs, *viz.*, Benign Epithelial Neoplasia. Epithelial proliferation now gives rise to viable cells capable of multiplying. It is accompanied by hyperplasia of connective tissue resulting in papillomata and cystadenomata of Schimmelbush's disease. It is benign at first and may remain so for several years. The epithelium of larger cysts is so flattened and degenerated that it rarely responds to this irritation. But the epithelium of the tiny microscopic cysts is liable to the change by the continued constant irritation. The change is chiefly noticed in the terminal ducts and acini at the age period of 35 to 45. The cells become more viable, nuclei become chromatic and cytoplasm stains normally. Desquamation ceases and papillomata with a core of fibrous tissue or sessile overgrowths of epithelial cells alone occur. In the terminal ducts, papillomata of the former type predominate though both types are present. The character of cells of the sessile type approaches more towards malignant cells. The epithelium of villous papillomata is columnar and regularly arranged over a core of connective tissue. It may remain so for a long time or it may grow rapidly and fill up the cyst completely. Then the cells become more cuboidal in shape. Sessile type are more common in the acini.

Third stage is a change into malignancy occurring in the age period 45 to 55. The change to malignancy may be mild. It is shown by the cells becoming irregular in shape and size and the nuclei becoming hyperchromatic. The arrangement of cells also becomes irregular. Mitotic figures are not common. The cells have not invaded the tissues as yet and axillary glands are not enlarged as yet. This type occurs where villous papillomata predominate.

In the more malignant types, frank anaplasia is present as shown by the complete loss of arrangement: cells become polyhedral in shape and mitotic figures are common. This change is seen over larger areas and cells may show mucoid degeneration. Axillary glands are found enlarged early in this type. This type supervenes, where sessile type of papillomata predominates. The spread is multicentric and the diffusion is wider than in the first type.

Carcinoma arising in large cysts:—In a cystiphorous breast, it would be impossible to say that carcinoma is not present in a large cyst by clinical examination alone. Biopsy in the operation theatre is necessary, frozen sections being examined microscopically. The carcinoma may arise from pre-existing benign neoplasms in a large cyst or it may start in a small duct, communicating with a large cyst. These neoplastic changes are more common in the terminal ducts of cystic breasts, and the large cysts rarely show neoplasms. Hence, in the majority of cases, carcinoma arises from the smaller ducts.

Bloodgood considers that chronic cystic mastitis is not a precancerous condition. In a small percentage, there may be a discharge from the nipple, intermittent retraction of the nipple is diagnostic, but occurs only in 10% of cases. To palpation, a shotty condition of the breast with increased firmness of the breast tissue, is evident. The edge of the breast is smooth and sharply defined, not unlike the sharp edge of the liver. When raised away from the chest wall, it curves and feels like a saucer with thickened edge to palpation. It may be more advanced in one breast than in the other, the latter feeling only shotty and the former like a saucer. The pathological change appears in the upper outer quadrant of both breasts, next encroaches on the upper and inner quadrant, the lower and inner quadrant being the last to be involved. There is no atrophy of subcutaneous fat or dimpling of skin over a fully developed zone. The nipple is usually free, 10 % may show intermittent retraction and others may have a congenital retraction, of one or both nipples. Less than 3 % show unilateral retraction of nipple so characteristic of cancer.

Two types:—In one, large single or multiple cysts predominate in gross appearance, while in the other there are minute cysts or dilated ducts or papillary cyst adenomatous areas. The large cysts include single blue-domed cysts, multiple blue-domed cysts of galactocoele type. These cystic tumours give rise to "Lumpy breasts" in contra-distinction to "shotty breasts", Chronic

cystic mastitis of the diffuse non-encepsulated cystic-adenomatous type is not a pre-cancerous condition.

Clinical signs and Diagnosis

1. **Single cysts.**—When large, are firm and tense, with smooth and sharp-defined walls, and are freely movable. Fluctuation may be elicited. In a fat breast, a deep-seated cyst may be difficult to diagnose from a solid tumour. But if the contents are clear, the cyst is translucent to illumination. These are never adherent to the surrounding structures unless inflamed by infection or trauma. The contents of an uncomplicated cyst are clear straw-coloured fluid. Nipple is not retracted, nor axillary glands are enlarged. But cysts with benign or malignant neoplasms may contain blood or serosanguinous fluid which, on aspiration and staining, may show malignant cells. There may be no discharge from the nipple unless malignant change be present. During the early stages, the pain is due to distention of tissues, but this subsides when the cyst has become clinically evident. But, either the pain or the later lump, makes the patient consult the doctor. A single cyst is a rare occurrence and may reach the size of a Tangerine orange.

2. **Multiple cysts.**—In the early stages, a multinodularity due to small, round, smooth, slippery swellings that are elastic to palpation is noticed. The sizes of these cysts vary, a few being larger and palpable, others small and indistinct. In the later stages, the contour of the breast becomes irregular from these cysts. The breast shows areas of translucency but no opacities. This condition is much rarer than mazoplasia. It may be bilateral simultaneously or consecutively. There is never any discharge from the nipple but it may be covered by a layer of dried serum. Mazoplasia is usually present also with this condition. But mazoplasia may exist alone to a greater or less degree in breasts of women from 30 years to menopause without any other change. Mazoplasia is universal and is only a physiological remnant of a previous normal activity.

3. **Localised nodularity** may be due to a localized collection of cysts by the cystiphorous condition affecting a single duct, its branches and acini. When a main duct and its branches are involved, the clinical condition is noticed as a triangular nodular area with its base at the periphery of the breast. If the condition remains only as a cystiphorous condition, large cysts may form and dominate the picture. But if neoplastic change is occurring there is no tendency for formation of large cysts. Benign

neoplasia is more common from 35 to 45 years and malignant change in these may occur at the age period of 45 to 55.

A single cyst, a localised collection of cysts, or multiple cysts may exist for several years in a breast without any further pathological change. But neoplastic change may set in at any time usually after 35, without any definite signs and symptoms. But with advance of neoplastic disease to a precancerous or cancerous condition, serous, sero-sanguinous or bloody discharge appears at the nipple and breast shows areas of opacities to transillumination and finally axillary glands enlarge. As 20 % of carcinomata arise from such cystic breasts with neoplastic change, these cases should be kept under observation. Cysts should be aspirated and contents stained and examined for malignant cell. Biopsy and examination of frozen sections should be done in the theatre.

Treatment :—For a single cyst, a wide wedge section is done, rest of the breast carefully examined and a frozen section is immediately examined. If no neoplasia be present, wound is closed with a drain. Electro-surgical knife should be used for this.

Simple multiple cysts involving the whole breast :—Amputation of the affected breast with or without clearing the axilla but without removal of pectoral muscles. Such breast should be examined microscopically by serial sections.

For cases of localised nodularity of early cystiphorous breast :—These small cysts showing opacities on transillumination are to be considered dangerous. An exploratory operation and an immediate examination of a frozen section to exclude fat-necrosis, fibro-adenomata, xanthoma and early tubercle is done. If only a benign neoplastic change is present, amputation of breast with or without clearance of axilla is done. But if a pre-cancerous or cancerous change is observed, a radical amputation should be done and later prophylactic deep X-ray therapy.

Conclusion :—What was formerly known as chronic lobar and lobular mastitis is divisible into two distinct conditions. Both are desquamative epithelial hyperplasias. The one which is simple and called Mazoplasia is more universal and is really the remains of a physiological activity. The other called Cystiphorous hyperplasia results in multiple cysts of breast and is a distinct pathological condition. But this condition may progress towards benign neoplasia and finally end in malignant neoplasia or cancer. Hence this condition should be kept under observation and treated accordingly.

Those who are interested in these conditions may refer for

details to "Tumours of the Breast" by Sir Lenthal Cheate and Max Cutler.

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ACUTE APPENDICITIS

BY

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Anatomy.—The appendix is a blind tubular diverticulum opening into the postero-medial aspect of caecum about two inches below the ileo-caecal opening. In the infant it forms the terminal part of caecum and may persist as such and is called "foetal type". Usually the growth and saculation of caecum displaces it to the former position. After middle age it begins to atrophy and in the aged it may be represented by a fibrous cord. Its function is unknown and the body suffers in no way by its removal. But its surgical importance is great as it is responsible for a large number of abdominal catastrophies. Its length varies from 1 to 9 inches, average being 3—4 inches and about $\frac{1}{4}$ inch in diameter. Its opening into the caecum is often guarded by a fold of mucosa called "Valve of Gerlach". Its lumen is narrow. It is well supplied with lymphoid tissue especially so in children and is known as the "abdominal tonsil". Owing to its length and congenital peritoneal folds and bands it is liable to twists and kinks giving it different shapes, viz., U. S. J. C. etc., Excessive mobility of caecum and a long appendix favour torsion and kinking.

Positions.—It occupies several positions. It is retro-caecal in 64 %, sub-caecal in 2 %, hanging over pelvic brim in 32 % and medial to caecum in about 2%. It may be misplaced by non-descent of caecum and take up a sub-hepatic or sub-pyloric position. It may be retro-caecal and extra-peritoneal (loin position.) It may be in the left iliac fossa due to complete transposition of viscera or non-rotation of intestines. If the caecum and ascending colon have a mesentery the appendix may be found anywhere in the abdomen—in the left side, in the true pelvis or in hernial sacs inguinal and femoral.

Blood and Lymphatic Supply.—The appendicular artery is a branch of ileo-caecal and passes under the terminal part of ileum, into the meso-appendix. In addition to this, there is a second fold of peritoneum "the ileo-caecal fold" from the anterior surface of terminal part of ileum to the base of appendix, which more often than not contains a blood vessel. The lymph is drained by 3 to 4 lymphatics into the ileo-caecal glands. There appears to be a rather intimate connection between the lymphatics of appendix, pylorus and duodenum.

Incidence.—Appendicitis seems to be more common in civilised communities due to habits and diet. It appears to be less common in vegetarians than in meat eaters. The disease is rare in infancy but becomes increasingly more frequent with advancing age reaching the maximum in the third decade of life. Then it begins to decrease and in old age is as rare as in infancy. It is about twice as common in men as women. However no age is exempt from this affection.

Etiology.—Dietetic errors, chronic constipation, gastrointestinal disorders may favour its onset. Parasites as whip and thread worms may give rise to ulceration of its mucosa and to infection. It may also be secondarily involved from inflammations of caecum, tubes etc. Faecoliths which are fairly common in constipated people may also give rise to ulceration and infection. A large majority of acute cases are due to partial or total obstruction in its lumen and infection. The obstruction may be due to bands and adhesions, congenital or acquired, producing kinks and twists in the appendix leading to stagnation and decomposition of contents which leads to a spreading infection. Impaction of faecoliths especially in the presence of strictures or kinks ends in a similar way. A primary infection of the lymphoid tissue is followed by swelling of mucosa and sub-mucosa and purulent exudate which leads to distension. The presence of fibrous strictures in the proximal part of appendix as a result of a previous attack is fairly common. Occasionally a primary attack of acute appendicitis is due to a primary bacterial infection without any obstruction.

Pathology.—Professor Aschoff considers that infection is entirely enterogenous. A few hold that hæmatogenous infection may also take place. The organisms found are Gram-positive cocci and bacilli. Cultural studies have shown that 70% of acute cases are due to intestinal streptococci, 17%, due to pneumococci. Owing to richness of lymphoid tissue a small primary focus of infection is liable to spread. The virulency of organisms is increased by

stasis and decomposition as a result of obstruction, more so in the presence of protein food material. Primary bacterial infection without obstruction may occasionally occur. This gives rise to a catarrhal inflammation of the mucosa and sub-mucosa which become swollen and hyperaemic and the lumen becomes filled with a mixture of purulent exudate and secretion. In the absence of obstruction the exudate escapes into caecum and the attack may subside. But as desquamation of epithelium with ulceration would occur, such attacks leave behind diffuse or localised fibrosis. Localised fibrosis is more common especially in the proximal part of appendix and ends in strictures. Diffuse type ends in obliteration of lumen. Occasionally the virulence of organisms may be severe enough for inflammation to spread to the peritoneal coat and infect general peritoneal cavity. But rupture or gangrene in the absence of obstruction is rare in this type. If obstruction be present the inflammatory changes may vary from a mild superficial catarrh to total gangrene of appendix.

In the mildest case, the mucosa becomes swollen and hyperaemic and the lumen filled with secretion and purulent exudate. If obstruction is slight, it may be overcome and contents escape into the caecum and inflammation may subside. But such repeated attacks only increase the stenosis. This is seen in recurrent appendicitis. Partial obstructions result in retention and inspissation of mucous and faecal matter into concretions or faecoliths, varying in sizes from a cherry stone to a date-stone. These often show laminated structure. These faecoliths may produce ulceration or complete obstruction by impaction into a stricture. Faecoliths have been found in 25 % of acute cases. Obstruction is liable to produce small diverticula liable to rupture in acute obstructive cases. A total obstruction with no infection may end in a mucocele or if infection has been mild end in a pyocele or empyema of appendix.

Acute appendicitis can be divided into

- (1) acute inflammatory type
- (2) acute obstructive type
- (3) a combination of obstruction and inflammations simultaneously or one supervening on the other.

Acute inflammatory type:—In the primary type an acute catarrhal inflammation occurs. In the mildest case the mucosa becomes congested and proliferation of epithelium occurs and the attack subsides. This type is liable to be missed. If the inflammation is more severe it involves the submucous layer also. The mucosa shows ulcerative destruction and the lumen is filled with a purulent

exudate full of desquamated cells. The attack may subside under treatment but leaves behind a damaged appendix with strictures and liable to relapses, formation of stercoliths, pyocele mucocele or diverticula. If the infection is more virulent, it spreads to the peritoneal coat. A protective plastic exudate binds the omentum and loops of intestines round the appendix. The attack is thus localised into an abscess. The abscess may become definite and point in some place, sometimes it may burst into the general peritoneal cavity or the fluid portion of abscess become absorbed leaving a mass of adhesions round a damaged appendix. One may find sometimes a small cheesy encapsulated abscess inside the adhesions. In children the omentum is poorly formed and in the aged it is often atrophied and in both defensive powers are poor and hence infection in them is liable to spread into general peritonitis. Administration of purgatives is a serious danger in these cases as it produces violent peristalsis and is liable to spread the infection to a fatal issue. If there be kinks and twists in the appendix owing to congenital or acquired adhesions, the lumen becomes distended with purulent exudate and perforation or gangrene is liable to occur and flood the general peritoneal cavity with virulent organisms. The medial position of appendix in such cases is very dangerous as the upper half of peritoneal cavity gets infected.

Acute obstructive type.—The obstruction is often the result of strictures in the proximal third of the appendix, a legacy of previous inflammatory attack. In some it is due to adhesions foetal or inflammatory, producing kinks and twists in the appendix. The outlet being obstructed, retention and putrefaction of contents leads to distension, and pulsion diverticula may occur at weakened spots in the wall. In some inspissation leads to formation of concretions. Attempts to empty the contents give rise to colics. If contents escape into caecum the colic slowly subsides to repeat itself again after some time. If a stercolith be present, it is liable to impact in the stricture and complete the obstruction. In this the organ becomes turgid and distended and either a perforation or gangrene may result. In meat eaters the contents, being richer in proteins, are liable to give rise to more distension from decomposition than where contents are carbohydrates. Where infective inflammation is added to obstruction the consequences are worse. Inflammatory oedema completes the obstruction and the contents also rapidly increase by the addition of purulent exudate. Hence perforation or gangrene occur earlier and the contents are also more virulent.

90% of fatal cases of acute appendicitis are the result of obstruction or obstruction with inflammation. (Wilkie)

Symptoms.—In the inflammatory type the symptoms are chiefly due to absorption of toxic products and a local peritonitis. The attack is ushered in with a dull aching pain round the umbilicus, becoming steadily worse and followed by nausea and occasionally vomiting. The pulse steadily rises in rate. Soon a rise of temperature to 99° to 100° F occurs and tongue becomes coated. Usually constipation is present. The pain soon settles down to the right iliac fossa. Tenderness and reflex rigidity are evident in the right iliac fossa. With onset of peritonitis, spontaneous rigidity becomes evident. The area of tenderness and rigidity increase with spread of peritonitis. The tongue becomes dirty, abdomen distended and rigid, respiration thoracic, pulse small and rapid and cheeks hollow and eyes sunken when general peritonitis is present. In the earlier stage pressure in other parts of abdomen elicits pain in the right iliac fossa. In retro-colic appendix pain is referred to lumbar region and in pelvic appendix it is referred to hypogastric region and tenderness elicited by rectal or vaginal examination. As pelvic peritoneum is not as sensitive as that of upper abdomen rigidity is not noticed in the earlier stages till inflammation has extended to upper abdomen. Diarrhoea may be present in retro-colic appendicitis. Dysenteric symptoms and frequency of micturition may be complained of, in a pelvic appendix. If sudden colicky pain with vomiting occurs in the earlier stages, prognosis becomes graver as this is due to obstruction, and rupture or gangrene threatens. But if they occur later in the disease when adhesions have formed they are not so serious. In the former general peritonitis may result and in the latter only an abscess.

B. Acute obstructive type.—In this the patient is suddenly seized with cramping pain (often at night) in the lower epigastric region or round the umbilicus invariably accompanied by vomiting or retching. He may writhe in pain for a few minutes to an hour. At this time he has a clean tongue, normal pulse-rate and normal temperature but has an anxious look. There is definite tenderness in the appendicular region. A distended appendix may be felt but most often not. Reflex rigidity is slight or absent. The rigidity of an inflamed appendix is absent. The pain may abate gradually to repeat again as an intermittent colic. If the contents have emptied into the caecum the colic may appear after some weeks or months, otherwise it may repeat in a few minutes or hours but in the latter type there is always a dull ache

in the interval. If a purgative has been given in such cases, as so often happens, the pain reappears and is liable to end in gangrene or perforation. Any sudden cessation of this pain with relief is a danger signal of rupture or gangrene of appendix. This is a dangerous stage, as the patient is certain to refuse an operation that is urgently indicated. After some time pain, rising pulse-rate, fever, rigidity in the right iliac fossa and tenderness set in. Tongue becomes coated and dry. Next signs of advancing peritonitis become evident. Perforation is usually absent in the first twenty-four hours if a purgative has not been given.

Localisation of pain, tenderness and rigidity become evident with onset of peritonitis. Tenderness on deep pressure may be present with a distended appendix. The more frequent the appendicular colic, the more rapid the onset of gangrene or perforation. Hyperaesthesia over the region of appendix may be sometimes present if appendix is distended. According to Wilkie, retention of protein matter and its decomposition are the principal factors in the distension-gangrene of obstructive type, but with carbohydrate contents, distension is less severe and more likely to lead to an empyema of appendix. If the lumen be empty, a mucocoele may result from impermeable strictures. *The obstructive types, with symptoms resembling an acute internal strangulation,* are the most serious cases. If operated within 24 hours, 50 % would show patches of gangrene or be found tensely distended and angry-looking, 15 % may show local peritonitis and 3 % general peritonitis. (Hawe). Sudden recurring abdominal colic, with vomiting but without rise of temperature and pulse-rate, is a warning to withhold any purgative, as this would certainly end in an abdominal catastrophe in most cases.

Mixed type.—In this, rise of temperature and pulse is added to colicky pain and vomiting. Gangrene and perforation occur earlier, owing to increased distension by the exudate and inflammation of the wall.

Later symptoms.—After a period of 4 to 24 hours, local pain appears usually in the right iliac fossa and is constant and accompanied by tenderness and rigidity and pulse-rate rises with temperature, varying from 99° to 101° F and tongue, coated and dry. Later still, signs of advancing peritonitis may become evident as shown by abdominal distension and rigidity with thoracic type of respiration.

Peritonitis may be localised or general.

Localised peritonitis or appendicular abscess.—When peritonitis becomes localised, an indefinite swelling begins to form at first and

after about four days, a definite mass becomes palpable. With this, the pulse-rate and temperature show improvement, tongue becomes moist and general improvement begins to show itself. A definite leucocytosis of 15 thousand and over occurs. In a pelvic appendix, dysuria with frequency, rectal tenesmus with passage of mucus may occur at this time. In retro-colic and retro-caecal appendix, diarrhoea may occur and flexion of thigh due to inflammation spreading to the sheath of ilio-psoas and tenderness, most marked in the lumbar region, are present. The temperature in retro-peritoneal appendicitis will be higher from infection of cellular tissue. A varying amount of purulent exudate forms in this mass. This may become absorbed or work its way to the surface or into a hollow viscus as colon, bladder, rectum, etc., But such localization is rare in children and poor in the aged.

Spreading peritonitis.—Occurs if localization fails. It is often seen in children, in the aged, and in the obstructive type with gangrene or perforation, owing to virulency of infecting material. The pulse-rate rises with a falling temperature, regurgitant vomiting becomes more frequent, tongue becomes dry, brown and cracked, breath is foul, cheeks and eyes sunken. The area of rigidity and tenderness steadily increase, distension becomes more and more marked. Finally, hiccough, indicative of sub-phrenic peritonitis, sets in.

Vomiting.—Is slight in simple inflammatory type, more severe in the obstructive type. It reappears as a regurgitant vomiting with onset of spreading peritonitis.

Temperature.—Begins to come down with improvement in pulse-rate and general condition on localization of infection. But a fall in temperature with a rising pulse-rate is indicative of spreading peritonitis. Temperature varies up to 101° F in adults, and may rise to 102° or 103° F in children. When abscess has formed chills or slight rigors with rises to 101° F may occur. Temperature is higher in infection of retro-peritoneal connective tissue.

Leucocytosis.—Occurs if resistance is good and is best seen in localization into an abscess. A leucocytosis over 15 thousand of polymorpho-nuclear type indicates an appendicular abscess.

Acute Appendicitis in children.—Inflammatory type is more common but localization is less likely and often incomplete and apt to break down again. The younger the child, the more serious the prognosis. The appendix is either pelvic or retro-caecal in type. Since it has thinner walls and is richer in lymphoid tissue,

inflammation spreads more rapidly to the peritoneal surface and liability to rupture or gangrene is greater. Localization is poor, owing to poorly developed omentum, want of acquired immunity to infections and poor resistance. Administration of purgative increases the danger of rupture and gangrene. Early diagnosis is essential but is difficult for want of a coherent history, lack of the usual sequence of symptoms and the frequent atypical character of disease, as diarrhoea, dysuria, chills etc., often mislead. Moynihan advises immediate operation in suspected cases with history of a purgative having been administered. Rigidity is often absent owing to appendix being pelvic or retro-caecal. A rectal examination though difficult gives more information than in adults. It is often simulated by intestinal colic, pneumonia, pleurisy, pneumococcal peritonitis and ileo-caecal adenitis. In children operation should be done as early as possible without waiting for localization.

In the aged.—Owing to poor resistance, poor vascular supply to appendix from arterio-sclerotic changes and fibrosis of appendix, the obstructive type ending in gangrene is more frequent. Localization is poor in them as omentum is often atrophic and resistance to infection is poor, and spreading peritonitis and thrombophlebitis are frequent. Symptoms are often atypical—Indigestion, sudden abdominal pain, erratic vomiting or nausea and a uniform soft distension of abdomen are present. Temperature may be normal or subnormal and leucocytosis is absent or poor. Chills are of serious import—gangrene, mesenteric thrombosis or pyelephlebitis. Operation should be done at once.

Acute Appendicitis during pregnancy is very serious as mortality is 50 %. Even in the presence of localization abortion is liable to occur about the 7th day when either a spreading peritonitis or infection of uterus may lead to a fatal issue. The abdominal pain and vomiting are liable to be ascribed to pregnancy and a pregnant uterus hinders an abdominal examination and detection of rigidity. B. Coli pyelitis simulates the early stages of appendicitis. Hence a case should be kept under observation and operated as soon as diagnosis has been made.

Treatment.—As it is impossible to predict what the end of an acute attack would be, it is necessary to operate within 24 hours of onset. It would not take more than 24 hours to make a definite diagnosis and mortality in this is no higher than in the "interval operation". Up to 48 hours very little localization occurs and operation should be done if the case is within this period. Mortality is no higher. In children, in the aged and in pregnancy

operation should be done as soon as diagnosis has been made whatever the interval be for reasons already stated. In obstructive type immediate operation is indicated especially so if a purgative has been given.

In adults after 48 hours either localization or spreading peritonitis is present. For the latter condition an immediate operation for draining the peritoneal cavity with removal of appendix if easily accessible, should be undertaken. But where localization is evident expectant treatment is carried out. Doubtful cases are treated expectantly without morphia and if no improvement occurs and pulse-rate rises above 100, it is advisable to operate. Localization may progress to abscess formation or end in resolution. For the latter an interval operation a couple of months later, is done. This gives time for things to settle down and some of the adhesions get absorbed. An abscess may burst into a hollow viscus or point at the abdominal wall. In the latter drainage is done and appendix removed, if found inside the abscess but no attempt is made to search for the appendix by breaking down any adhesions, for fear of infecting general peritoneal cavity. If at any time during the formation of the abscess leakage into general peritoneal cavity is evident by symptoms, an immediate drainage of the abscess by the shortest route should be done. Never drain an abscess through the general peritoneal cavity.

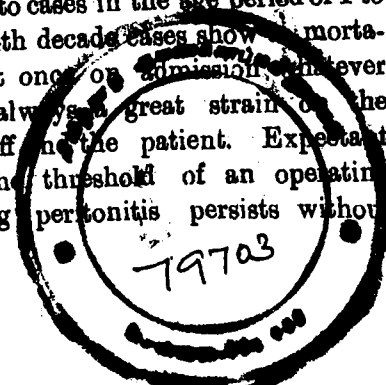
Expectant Treatment.—Where localization is evident when the case is first seen, where operation is not undertaken for other cogent reasons or has been refused, expectant treatment is undertaken. Complete rest in Fowler's position, no food by mouth except sips of glucose water to moisten the mouth and tongue, normal saline and 5% glucose solution is given liberally by other routes, by rectal drip method, subcutaneous method or by the continuous intravenous method at the rate of 8 c.c. per minute. Locally radiant heat or warm stupes of saturated solution of magnesium sulphate in glycerine are given. Injections of polyvalent antigas serum combined anti-streptococcic and anti-coli sera are of value. Food and drinks by mouth, aperients, pituitrin and enema should be strictly prohibited as these give rise to peristalsis of intestines preventing proper localization and aiding in diffusion of infection. The glucose administered not only provides an easily available food material but also prevents acidosis. I do not advocate morphia except when a patient has to be sent some distance over rough roads for an immediate operation and this fact should be intimated in writing to the Surgeon who would attend on him. Some advocate morphia in expectant treatment, a starting

dose of gr. $\frac{1}{2}$, with half that dose repeated every six hours till localization is definite and pain is bearable. But it is liable to mask symptoms of spread of peritonitis. A trained Nurse should be in charge of the case and a second-hourly chart of pulse, temperature, etc., should be maintained. At any moment an urgent operation may be indicated by a rising pulse-rate and spread of pain, rigidity and tenderness. With improvement in local and general condition as shown by lowered pulse rate, temperature and a clean tongue, small amounts of fruit juice, and peptonized milk are given. Later diluted milk, junket, jellies, etc., are added. Glycerine enema may be given after this if bowels do not open spontaneously.

Of cases seen in Adults after 48 hours period, 90% usually get localized and 10% may end in generalized peritonitis. Of the former 65% end in resolution and are advised an operation three months later, 25% end in abscess requiring drainage. If the appendix was not removed then these are advised its removal 4 to 6 months later. The other 10% require an immediate operation of at least drainage of peritoneal cavity with or without removal of appendix. The mortality in these is heavy about 20%.

According to Sherren-school, expectant treatment should be carried out if a case is seen 48 hours after onset, as surgical interference from the third to the 5th day inclusive is attended with a high mortality for in this period natural immunity has been exhausted and acquired immunity has not been established. But the opposite school with equal cogent reasons advocate immediate operation whatever the time period. Mortality for all cases operated at once is 5.8% but is reduced to 3.5% in the hands of experienced operators. H. C. W. Nuttall strongly advocates immediate operation. Atkinson Stoney of Dublin gives a mortality of 2.5% in immediate operations for all cases. Mortality is high at the extremes of life being 12% in infants, 7.9% in children and 42% in people over 60 but in adults it is only 2.16% lowest being in the third decade of life. (E. L. Keyes.)

H. C. W. Nuttall says that there is no evidence to prove that there is a negative phase in the period of 3rd to 5th day, and that high mortality in this period is due to cases in the age period of 1 to 20 and those over 50. Third and 4th decade cases show a mortality of 3% or less when operated at once on admission, whatever the day. Expectant treatment is always a great strain on the hospital Surgical and Nursing staff and the patient. Expectant treatment has to be carried on the threshold of an operating theatre as it were. If spreading peritonitis persists without



localization, patient has to be operated on at once as an emergency. He finds that it makes little difference in the mortality rate whether immediate operation is done or expectant treatment is carried out. He advises removal of appendix if it can be done easily but with a congenitally fixed caecum and appendix buried retro-caecally simple drainage is carried out. Muscle-split incision over the tenderest point or palpable mass is done. If there be no difficulty or danger remove the appendix. If abscess is present, do not attempt to separate dense adhesions. If the appendix is retrocaecal, close the peritoneum and drain the abscess intra-peritoneally. Removal of appendix may be done by direct method, retrograde or sometimes by removing the mucous tube only. If infection is mild mop the part dry and close without drainage. But if caecum is very inflamed and fair amount of massive peritoneal infection be present, drainage should be done, lumbar, suprapubic or both as required. The abdominal wound should be drained with rubber tissue. Where it is difficult to remove the appendix, simply drain the area and carry on with expectant treatment. Three weeks later remove the appendix. He gives the following figures of different surgeons that have performed immediate operation without any consideration of the day of onset.

Name.	Total	Recovered.	Died.	Mortality.	Reference.
Raw	619	608	16	2.58%	B. M. J. 1933; 1, 1930
Stoney	371	267	7	2.58%	Roy. Soc. Med. 1930
Black	340	326	14	4.1%	Do. 1930
Hawe	250	240	10	4%	Liverpool Med. Clin. 1934
Nuttall	531	527	4	0.75%	Brit. Jour. Surg. 1934
Total	2081	1978	64	3%	

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About 150 children were treated in the children's ward of the General Hospital under the care of my Chief, Col. Pandalai, I. M. S. in the year 1932. The following table shows the distribution and incidence of the various diseases:—

No.	Disease.	No.	Per-centage.	Remarks.
1	Fractures	14	9	both bones leg 6 Femur 8 Humerus 3 Clavicle 1 Mandible 1
2	Hernia	10	7	Inguinal 8 Umbilical 2 Inguinal & umbilical 1 One inguinal had phimosis
3	Tetanus	8	5	5 cured 3 died
4	Burns and scalps	7	5	6 cured 1 died
5	Tuberculosis of bones and joints	7	5	Spine 2 Hip 2 Knee 1 Rib 1 Calcaneus 1
6	Angioma	6	4	Lip 3 Cheek 1 Scalp 1 Thigh 1
7	Hare-lip and cleft palate	6	4	2 associated with cleft palate
8	Bilateral talipes equino varus	4	3	Tenotomy done in 2 prior to plaster casing
9	Imperforate anus	3	2	Stricture rectum dilated 1 Colostomy done in other 1
10	Acute appendicitis	3	2	Interval operation done
11	Cancerum Oris	3	2	2 died 1 discharged moribund
12	Empyema thorasis	2	1	Both did well with open drainage.
13	Prolapse rectum	2	1	Only palliative treatment done
14	Osteomyelitis	2	1	
15	One each of Hydrocephalus with meningocele, congenital dislocation hip-Epispadias etc.			

The miscellaneous group consisting of 70 children were

cases of pyæmic abscesses or other rare disease conditions or deformities.

In comparing these two tables one would be struck at the rarity of that extremely common condition Phimosis, in the hospital statistics which in the school comes to nearly 9%. The reason is that practically all cases of phimosis are done by the general practitioners and the out-patient departments of the Hospitals.

Nevertheless, it is an important common surgical condition which should merit our consideration this evening. It is one of those few but common conditions which will fall to the lot of a senior student working in the out-patients as well as the surgery of a general practitioner, especially in mofussil towns and rural hospitals and dispensaries. And among the other diseases which I am going to discuss briefly are Congenital Hernia, Hare-lip including cleft palate and Talipes Equino varus. I shall make passing references to Naevus, Burns and Tetanus especially with regard to their treatment. Fractures I shall leave to Dr. Raman Menon and another common condition, chronic otitis media, to the specialist concerned.

Naevus

Of the six cases of the Naevus treated 3 were on the lip, one on cheek, one on scalp and one on thigh. That on the scalp, a very large one was treated by excision in a really bloody operation. The one on the thigh was also excised. Two cases on the lip were treated with radium with very good results. Naevi are hæmangioma and are congenital flat, reddish areas seen on skin. These consist simply of group of dilated capillaries which lie in corium and are covered by a very thin layer of epithelium and surrounded by connective tissue. Microscopically the structure is mostly that of a cavernous angioma but portions may resemble the capillary form also. A capillary angioma occurring on the forehead or scalp may be infiltrated with fibrous tissue and may be clinically indistinguishable from a sebaceous cyst which is also very common in the same region. Recently I had a case on the right forehead just above the outer part of the supra-orbital margin. It was of the size of a lime. I, along with a number of students at the out-patient thought the tumour to be a sebaceous or dermoid cyst. On operation under Novocaine infiltration anaesthesia, the tumour was much more vascular than usual. The pathologist reported the specimen as capillary Angioma.

As regards treatment, the method of choice would be excision, if possible, and radium treatment otherwise. Naevus of lip may

After
Infections

After
Operations

In general Weakness
and Exhaustion

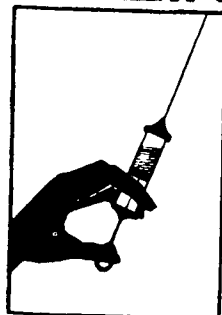
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SOME COMMON SURGICAL AFFECTIONS OF CHILDREN

BY

Capt. E. S. GOPALAN, M.B., B.S.,

Honorary Assistant Surgeon, Govt. General Hospital, Madras.

YOU may be surprised to learn that I found very great difficulty in coming to a definite conclusion as to what the common surgical affections of children are. How is one to decide the common surgical ailments of children? If I trust to my general impressions gathered in my private and hospital practice, I thought I would be lost in a large number of diseased conditions from a hydro-cephalus which is evidently a rare phenomenon to pyæmic abscesses which are very common both in private and hospital practices, but which are not dignified enough to merit our serious consideration here this evening. Moreover, there is another important difficulty which confronts me. The surgical diseases of children are shared by a number of specialists also. As I shall show you statistically, conditions of teeth are tackled by the dental surgeon, conditions of the throat are attended to by the oto-rhyno-laryngologist and traumatic injuries including fractures, which is a very important and vital part of the surgery of children, is handled by the orthopaedic surgeon. And to boot, acute emergencies in the newly born such as imperforate anus, ophthalmia neonatarum etc., are seen and managed at the Lying-in-hospitals. So after sorting off these important diseases to the specialists concerned, the poor general surgeon is left with a residue of diseases which comparatively are more difficult to treat. Which is easier to treat, say, a hypertrophied tonsil requiring tonsillectomy or a cleft palate?

I have had the privilege of being a medical inspector of a secondary school with a student population of more than one thousand. The following table shows the incidence of some of the surgical ailments in a population group which is expected to be healthy for all practical purposes:—

Year of Examination: 1927.

Total Number of Children: 1,079

No.	Disease	Number.	Percentage.
1	Caries teeth	20	2% roughly
2	Tonsils and adenoids	106	10%
3	Hydrocele	23	2%
4	Hernia	12	1%
5	Phimosis	98	9%

be treated by interstitial implantation of small strength radium needles. Flat naevi on the fore-head, cheek, etc., may be treated by specially prepared pads. One has to be extremely careful in treating children with radium especially on the face and head. The necrosis and reaction might be terrible and undreamt of. Very close and individual attention is necessary and the effect of Radiation watched from day to-day and from morning to evening. On the slightest sign of reaction, or irritation, the radium should be removed for application on a future occasion, if necessary. Recently a few months ago, a rapidly creeping Naevus of the scalp and face was admitted in the Hospital in a young child of a month or two. The surgeon rightly declined to take the responsibility of treating the case with radium in the Hospital. Such a case should be handled by one who can devote constant personal attention all the 24 hours of the day. Other methods of treatment which may be mentioned in passing are :—

- (1) Application of CO₂ snow,
- (2) Caustery-galvano or diathermy. Naevi which are not growing and which do not cause disfigurement or impairment of function may be left alone.

Burns

We were lucky with our figures for burns. Out of seven cases, six recovered and one died. My impression is that during this period we did not get very bad and extensive cases of burns amongst whom the mortality due to primary shock should be more.

The routine treatment was tanning of the skin with repeated sprays of 2½% sterile solution of freshly prepared tannic acid under a cradle covered with woollen blankets. An ordinary gas-filled electric bulb is placed under the blanket day and night. In all cases where there are a number of blebs and tags of skin, it would be very wise plan to give the child a little anæsthetic in the bed itself, and remove the blebs and tags of skin and then start the tannic acid treatment. There need be no hesitation in giving older children a small dose of morphine. The younger children may be dosed with bromides and paraldehyde by rectum. Shock should be treated in conscious children with plenty of fluids by mouth, and in the unconscious with continuous rectal glucose and saline (5% glucose in normal saline). Recently we have adopted a slightly different variation, in the spray solution. Instead of the 2½% solution, we use a 5% solution, and in addition we put trypanflavine or acriflavine at a strength of 1 in 1000. So far I

have used it in 2 cases and my impression is it controls the sepsis and tans the skin earlier, thus reducing the period of shock, which is due to the absorption of toxic products from the scalded or burnt skin. Another point which Col. Pandalai wants his assistants and house-surgeons to do is not to bother the patients with repeated dressings after the tannic acid treatment is over. The granulating wounds are dressed with plain gauze soaked in sterile liquid paraffin containing trypanflavine to the strength of 1 in 1000. The dressings need not be disturbed 3 or 4 days or till the patient begins to stink. It is not unusual for one to have noted in the Hospital to see a child disturb the whole hospital by-screaming at the time of dressing. No case of burns should be dressed when student or house-surgeon is in a hurry.

Another essential point in the treatment of burns is the prevention of deformities by suitable splints and in the final stages by recourse to skin grafting. Barring cases of suicidal burning which is uncommon in children, the two methods which can effectively prevent burns are: (1) prohibition of naked kerosine lights in house-holds, and (2) building the fire place at a height of at least 2 to 2½ feet above the kitchen floor level, so that children may not dip their hands in boiling materials and pull burning cinders from ground level fire places.

Tetanus

We were also particularly lucky with our tetanus cases. Out of cases we lost 2 cases and 5 cases were cured. So far as treatment is concerned there is nothing special except that heroic doses of concentrated anti-tetanic serum are being given, 30,000 units is perhaps a daily normal dose. Along with the serum we give bromides and chloral and purgatives with epsom salts. In older children, small doses of morphine may be freely given. Sitting at the Out-patient department, I see that prophylactic anti-tetanic serum is becoming very popular. I have seen cases dressed for injuries by corporation dispensaries and private practitioners going to the O.P. of the General Hospital specially what people call "Preventive injection". It is a good sign and if every case of street accident, minor, major, or trivial, gets a protecting dose of A. T. serum, the incidence of tetanus will be markedly reduced indeed.

Congenital Hernia

Of the 10 cases 8 were inguinal and 2 umbilical. One inguinal was associated with an umbilical hernia and another with phimosis.

One word about the conservative treatment of umbilical hernia

in babies below one year. In cases of small gaps, not exceeding the diameter of $\frac{1}{2}$ inch, I have found an improvised hard padding very successful. In fact, I have cured a number of cases in this manner. The principle is to prepare a circular metal sheet of copper of the diameter which exceeds the diameter of the hernial opening by at least 1 inch, enclose the circular copper sheet in sticking plaster all round with sticking side towards the metal, clean the umbilicus of the child with spirit, powder it, and place the padded circular metal directly over the aperture, so that the circumference of a sheet overlaps the circumference of the hernial opening very clearly and to an extent of $\frac{1}{2}$ inch on all sides. Fix the sheet very firmly with good Z—O plaster or leucoplast adhesive plaster applied in several strips in a radiating manner. Then apply plenty of cotton wool over the plaster and bandage firmly but not too tightly. When the outer dressings are soiled, the cotton may be changed but not the plaster and metal pad which should not be disturbed earlier than a week. In certain cases a copper half-an-anna coin or silver rupee may be used with advantage. One will be surprised to see the marked improvement this treatment has on some type of cases.

Inguinal hernia in children is due to the persistence and failure to obliterate the processus vaginalis. In the course of normal development, the processus vaginalis obliterates down to the testicle at or before birth. If it remains patent, abdominal contents may pass into it and the processus becomes the sac of a hernia. In order to understand the development of this hernia, it is necessary to consider briefly the descent of the testis and the formation of the processus vaginalis.

The description may be divided into

1. Anatomy of descent and
2. The Physiology of descent.

The anatomy of Descent.—The testes are situated in the abdominal cavity until nearly the end of the foetal life. They lie on the posterior abdominal wall at the level of the upper lumbar vertebrae, each being held in place by a fold of peritoneum—the mesorchium by which it is attached to the mesonephros. As growth proceeds so does the abdominal movement of the testes. At the third month they lie in the iliac fossa. In the seventh month, near the internal abdominal ring. By the eighth month they reach as a rule the scrotum.

Each testis acquires at an early stage an indirect attachment to the anterior abdominal wall by means of the gubernaculum. This structure develops within two folds of peritoneum—the

inguinal fold and the inguinal crest—which grow from the mesonephros and the anterior abdominal wall respectively and ultimately fuse. The gubernaculum reaches its full stage of development at the 5th or 6th month. The upper part is too thin and delicate and soon afterwards atrophies. The lower part is stout and rod-like and contains a central core of unstriated muscle fibres. It fixes the lower end of the testis and epididymis to the future scrotal wall.

As the testis descends to the internal abdominal ring two pouches of peritoneum are formed, one above it and one below it. The upper one plica-vascularis later conveys the testicular vessels. The lower surrounds the gubernaculum and passes downwards in front of it to its attachment to the scrotal skin. The process becomes the processus vaginalis.

Having arrived at the abdominal ring, the testis, following the course of the gubernaculum passes through the inguinal canal into the scrotum and at the same time the gubernaculum atrophies and finally disappears, little if any, trace of it being found after birth. As the testis descends into the scrotum, it invaginates the processus vaginalis. The upper part of the processus vaginalis becomes narrowed and soon after birth, its lumen is obliterated. The lower part of the processus vaginalis becomes the parietal wall of the tunica vaginalis which invests the testis.

The Physiology of Descent.—The older view attributed the entire descent to the activity of the gubernaculum, by active contraction of its muscular fibres. Later on the gubernaculum was denied any active part in the descent. The modern view lies between these two extremes. The migration is usually divided into two phases. In the first phase gubernaculum is held to have no active function. Movement of the testis is effected partly by the upward growth of the surrounding structures, partly by the degeneration of surrounding structures and partly by the degeneration of the cranial end of the testis and growth of the caudal end. The process is complete in three months. A stage of arrest follows. The second phase starting at the sixth month is probably actuated by a contraction and shrinkage of the gubernaculum, the testis being drawn into the scrotum through the inguinal canal.

There are three groups of hernia according to Hamilton Russel.

- (1) Process vaginalis, normal in shape.
 - (a) Total.
 - (b) Partial.

- (2) (a) Pro-peritoneal,
(b) Inter-muscular,
(c) Superficial inguinal.
- (3) Testicular portion, caught up in abdominal wall, Hernia-magia, etc.

You have to know the important clinical and fundamental fact that the sac is an all-important structure in inguinal hernia. It is the cause of the hernia and by its obliteration, cure is achieved. The hernia is generally reducible and with gentle pressure returns with a characteristic gurgle.

The only satisfactory treatment of hernia in children is by operation. The operation is simple and sure with a record virtually free from mortality. Treatment by truss is uncertain, unclean irksome and a source of irritation to a growing child. It is true that a truss can be made to control a hernia and if the hernia is perfectly controlled in the first few years of life, natural cure may result; but no exact prognosis can be given and the child is doomed to an uncertainty, which is indefensible. Under certain conditions, truss must be worn.

- (1) Infants under six months. Operation is better postponed until the child is definitely established.
- (2) Children whose general condition precludes operation.
- (3) Children suffering from alimentary, cardiac, respiratory or nervous disorders.

In all cases of hernia, it is first essential to examine the child carefully for any cause of straining—phimosis, stenosis of the urinary meatus, intestinal indigestion, worms, constipation and diarrhoea must be excluded or treated.

Operation is indicated :

- (1) In all healthy children over six months.
- (2) Earlier if no truss will control the hernia.
- (3) If the hernia has been irreducible.
- (4) For strangulation.

In the operative treatment, the two important points are :

- (1) to dissect out the sac completely.
- (2) not to interfere in any way with the normal anatomical position of the inguinal canal and external abdominal ring including the apo-neurosis of the external oblique. As a matter of fact Bassini's operation or its

modifications are uncalled for and might be causes for a recurring direct ventral hernia. The child should not be burdened with too many dressings. A thin layer of gauze painted with tincture benzoin Co. or Collodin is all that is necessary. After operation the child is kept in bed for a fortnight at least.

Hare-lip and cleft-palate

Hare-lip is more common than cleft-palate. It is sometimes associated with a partial or complete cleft-palate. I shall omit the embryological and developmental data for a separate lecture on a future occasion and content myself now with stating a few broad principles in the management of both types of defects. For hare-lip, the best time of operation is during the third month, for by then, the tissues are fairly strong, there is plenty of material to work on, the infant is strong enough to withstand the shock of the operation, and in the event of the size of the cleft or the condition of the mother having rendered bottle or spoon-feeding necessary, should be free from all gastro-intestinal disturbances.

When hare-lip is associated with cleft-palate the method in vogue in our Hospital is to do the hare-lip early and reserve the operation on the palate when the child is about three years. The main principles that underlie a successful and ornamental issue are (1) rectification of the maxillary arch (in unilateral clefts) or, replacement of the premaxilla in bilateral cleft, (2) free liberation of the lip, the cheek, the ala of the nose and occasionally of the columella from the underlying bone. (3) shaping the nostril, (4) paring the margins of the cleft or cutting a good sized flap or flaps. (5) accurate suturing of the raw surfaces. These operations must be undertaken very seriously and the surgeon undertaking such work must be an artist. Every detail of the operation must be meticulously gone through to ensure good cosmetic result.

For cleft-palate the best time of operation is about $2\frac{1}{2}$ to 3 years. To obtain the best phonetic result, the hard palate must be closed. The soft palate when sutured should be long, well formed, and so freely movable that it can shut off the post nasal space by making contact with the posterior wall of the pharynx, the teeth should be regular of good shape and normally placed, the nasal passages should be well-formed and whenever possible, the cleft should be closed before the child has learned to speak. The point of the whole operation is to produce an efficient and

movable soft palate. A cleft in the hard palate can at any time be closed by a suitable dental appliance. In our Hospital usually the Langenbeck type of operation is performed.

SOME COMMON SURGICAL AFFECTIONS OF CHILDREN

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Part II

Congenital Talipes Equino Varus

It is very commonly bilateral. All the four cases treated were bilateral. The following are the five elements in the composite club-foot deformity.

- (1) Plantar flexion at the ankle joint (Equinus)
- (2) Plantar Flexion of the fore-foot at the mid-tarsal joint (Cavus).
This movement adds to the general equinus of the whole foot and is always accompanied by adduction of the fore-foot.
- (3) Inversion of the whole foot excluding the astragalus at the sub astragaloid joint. (Varus). The foot rotates on an antero-posterior axis. Occasionally a still further degree of inversion occurs at the mid-tarsal joint.
- (4) Adductions or rotation inwards of the whole foot on the vertical axis of the leg taking place in sub-astragaloid joint. In this motion the front of the os calcis turns inwards under the head of the astragalus while the heel goes in the reverse direction, that is, approaching nearer to the external malleolus.
- (5) Adduction of the fore-foot on the back foot at the mid-tarsal joint. This is a further degree of inward rotation on a vertical axis and is the most striking element in the composite distortion. It produces the dislocation inwards of the scaphoid on the head of the astragalus.

Nothing in surgery responds so well to proper treatment as

club-foot. The time to start treatment is immediately after birth. According to the extent of the deformity and the age of the patient, the following are the principle methods of treatment:—

- (1) **Manipulation.**—When the baby is born, a light plaster may be applied which can be changed as frequently as possible.
- (2) **Forcible Correction.**—This is reserved for cases that resist gentler manipulation and for those cases which come to the surgeon after some months. The correction may be done by hand or a Thomas's wrench may be used. In some cases division of the plantar fascia or tenotomy of the tendoachilles may be useful. The foot and leg are put in light plaster. The plaster is changed and foot corrected every month followed by plaster.
- (3) After three or four years of age "Trethowan's Operation" of cutting appropriate ligaments, fascia and tendons is said to give very promising results.
- (4) The operations of the astragalectomy, excision of a wedge from the dorso-external aspect of the mid-tarsus enucleation of ossific centres, removal of cuboid, transverse division of the tarsus and phelp's division of all structures on the inner side of the foot have fallen into disuse as primary corrective procedures. An operation for the cure of club-foot that destroys bone and joints is the refuge of the destitute. Some such operations are sometimes necessary in inveterate cases and cases which have relapsed after other methods of treatment. Osteotomy of the neck of the astragalus and of the oscalsis half an inch behind its anterior end after which the whole foot is abducted and elevated, and stabilizing operations such as arthrodesis of the sub-astragaloid and mid-tarsal joints may be of value occasionally.

Phimosis

As I have shown in the first table it may be taken as granted that roughly one male child out of ten born is having the condition known as phimosis. The degree of phimosis may vary. My criterion of phimosis is irretractability of the prepuce over the glans penis. There are cases where you might forcibly retract the skin but occasionally para-phimosis may result from such a

proceeding. Phimosis then is that condition in which the preputial orifice is so small or the preputial tissues so inelastic as to prevent retraction of the prepuce over the glans. It is of two types, congenital or acquired and I am more concerned with the congenital type to-day as occurring in young children. There are many degrees of phimosis. On the one end I have operated on a child within 8 hours of its birth for a condition which is not described in text books and which I should prefer to name as 'imperforate preputial orifice' or 'congenital occlusion of the preputial orifice'. The child's nurse told me that the child did not and could not pass urine. I applied a clean white diaper and watched for nearly four hours; the diaper was not wetted. I made a careful examination with the use of a fine probe but no orifice could be seen but the glans could be felt alright. With the consent of the parents, I did circumcision without any anaesthesia. The baby was not at all troublesome, the child did very well afterwards and now it is about 1½ years old in excellent health.

The next variety, perhaps, the common variety is the condition which I should call "pin-hole preputial orifice". The child passes urine but in a peculiar way. If you closely question the mother, she may have observed that instead of the child merely wetting the diaper or clothes or projecting a jet of urine of a height of a foot or so, she might have noticed the child squirting urine to a height of 3 feet or 4 feet or even against a wall, cradle or mother's face. How do you account for this? It happens this way. When a child passes urine, urine readily comes out of the external urethral orifice and being unable to be thrown out because of the pin-hole preputial orifice, distends the space lined by inner layer of the prepuce which in some instances might hold even two to three ounces and then finally due to the force of the bladder and penile contractions urine comes out in a forcible narrow jet. The child takes a longer time to empty the bladder and you can observe the child muttering a low groan (straining) as when a habitually constipated man does when he attempts to pass a few scybalous masses after prolonged effort. The next variety is also common in which the preputial orifice is stenosed in different degrees but there is absolutely no question of a possibility of retracting the prepuce. Such cases do not attract the attention of the parents but are brought before the Surgeon when the boy grows up along with the growth of the glans penis. The next variety is what I should call the "Border line" variety.

In this condition, the prepuce is not ordinarily retractable but with force it can be retracted. There is no obstruction to the

passage of urine. Such cases are precursors of para phimosis in adolescents and young adults especially after coitus. Clinically the cases can be divided into obstructive and non-obstructive cases. In obstructive cases immediate operation is a necessity. Non-obstructive cases especially in small children may be left alone for some years. I have often found one remarkable thing about these "Border line cases" I have traced and followed these cases for a number of years successively. As age advances and with the increasing growth and size of the glans penis, these irretractable cases become gradually retractable and spontaneous cure of an original non-obstructive phimosis has taken place. Hence in some such cases, the Doctor will be well advised to follow the case for a number of years say till twelve years and even then if the prepuce does not easily retract, circumcision can be advised.

Development of the prepuce.—The prepuce makes its appearance in embryos of two to 2½ months. It develops as an ingrowth of the epithelial covering of the dorsum of the penis. The ingrowth is in the form of a circular ridge placed at right angles to the long axis of the penis. In its early stages it is known as "Glandar" lamella, and it definitely divides the penis into two portions, the shaft proximally and the glans distally. It is a solid process of epithelium extending into the mesenchymatous tissue of the penis and directed towards the urethra. It is C shaped with the open portion of the C directed ventrally. This open portion remains as much throughout the entire development of the glandar lamella and from the mesenchyme within it, the frenum of the penis takes origin.

With subsequent development, the point of origin of the glandar lamella from the epidermis of the penis is gradually shifted distalward. Its deeper free edge does not greatly alter its position, and as a consequence the direction of the glandar lamella is changed from transverse to longitudinal with the continued shifting of the attached edge, the glandar lamella approaches and then rolls into the external urethral orifice.

While these changes in position are taking place, the glandar lamella becomes thickened and the number of layers of epithelial cells is increased. The epithelial thickening is accentuated at various points by the development of spherical masses of cells, epithelial whorls or pearls. These vary in size, number, and distribution, certain embryos show a marked precocity in these respects. They are, however, constant and numerous in all the foetal stages.

In the pre-natal stages the glandar lamella undergoes a

longitudinal splitting throughout its entire extent. That the epithelial pearls are concerned in this splitting process, there can be no doubt. As they become larger, their centres undergo degeneration, resulting in the formation of cavities filled with cellular debris, these distended cavities become united as the splitting of the glandular lamella proceeds, and the presumption is that they either mark the course of the splitting or are themselves responsible for it by the coalescence of their cavities.

The splitting process is usually completed at birth. It is responsible for the division of the epithelium of the glandular lamella into two portions, an inner which forms the mucous membrane covering the glans and an outer which becomes the mucous or inner leaf of the prepuce. Incomplete division of these layers, however, may result in continued union after birth. For this reason, complete retraction of the prepuce should be performed by the Obstetrician at the time of birth. If this is neglected, it will still be found possible during childhood becoming gradually more difficult, until the adhesions become so fibrosed that splitting by traction is impossible. When the prepuce is retracted at birth or during early childhood, one frequently finds a deposit of cheesy appearing smegma more abundant in the coronal sulcus, the broken down cellular debris of the epithelial pearls.

Function of the prepuce

- (1) To protect the tender glans during the early years of life.
- (2) Later in coition to enhance the vigour of the ejaculatory orgasm by its friction over the corona.

Symptoms.--Children are brought to the Surgeon for three reasons :—

- (1) The parents see the ballooning of the preputial cavity during micturition as well as the narrow preputial orifice and then seek advice.
- (2) The child is discovered to have a phimosis during a routine medical examination as in Medical inspection of school children ; and,
- (3) For certain complications which indirectly lead a Doctor to the probable cause of the complications.

The following complications may be mentioned :—

- (1) Difficulty and pain in passing urine.

With a pin-point prepuce and as I have occasionally seen it happening, even an ant bite brings on œdema of the prepuce with pain and retention of urine.

- (2) Retention of urine, with its remote complications as hypertrophy of bladder, hydroureter and hydronephrosis. If retention is associated with sepsis you can have cystitis, pyelonephritis and pyonephrosis.
- (3) Incontinence of urine.
- (4) Retention of secretion or formation of calculi under the prepuce. About three years ago a patient was admitted with a hard indurated swelling inside an irretractable prepuce of the size of an orange. The preputial orifice was ulcerated, rigid and tunnel-like. Very offensive discharge was coming out and the patient's condition was intolerable. It was a dense hard and fixed mass. It was provisionally diagnosed as Carcinoma of the penis. On exploration and circumcision, the mass was a collection of phosphatic calculi, about 84 in number. The specimen is kept in the Pathological museum of the Medical college. The patient was an elderly adult and did well subsequently.
- (5) Inflammatory conditions :—Balano-posthitis, urethritis, cystitis.
- (6) Hernia due to straining.
- (7) Prolapse rectum due to straining.
- (8) Sexual excitement, masturbation and its effects.
- (9) Pruritis prepuce.
- (10) Difficulties in coitus (does not occur in children).
- (11) Para-phimosis and venereal complications.
- (12) Malignant diseases in adults
- (13) Neuro-psychosis, convulsions, etc.
- (14) Religious. A few months ago a poor Mohamedan child was circumcised in the O. P. department, because, his father admitted he was penniless and could not afford to spend for the ceremony enjoined.

It has been definitely established that untreated phimosis in youth is a precursor of epithelioma in old ages.

Prognosis.—is good. There should be no mortality with

ordinary precautions. There is only one contra-indication to operation, and that is *haemophilia*. It is only very rarely that cases are reported as lost, after an ordinary circumcision. As regards the time of operation, any time is good enough, provided child is fit to stand the anaesthetic.

Treatment.—Treatment is by circumcision.

History of the operation.—This operation has the unique reputation of being the most ancient, most frequently performed and the simplest of surgical operations. Its origin is unknown, the operation being traced far back into the dawn of history. Among the ancients it was enshrouded with mystery, or conducted with religious rites, a custom which has been handed down century after century with but little change to the present day. Among a section of the Mohamedans the circumcision ceremony is celebrated as a grand ceremony, much grander than the ear-boring ceremony of the Hindus. In the richer classes of Mohamedans thousands of rupees are spent on account of this ceremony and there are special moulvis who are considered experts in the art and technique of the ritual and the surgical operation following. About a decade ago, I went from Nagapatam to Nagore, which has a mosque of considerable importance. I saw a procession obstructing the narrow meandering road. The procession was about a furlong in length and in a coach in the middle of the procession, a magnificently decorated boy was seen. He had undergone the ceremony that day.

Medical history informs us that the earliest records prove that the operation existed among the Arabs before the advent of Mohamed. The Mohamedan Arabs claim that the custom originated with Ishmael, son of Abram, who was circumcised in his 13th year, the age of puberty. According to Marcus, the Arabic term 'Hatana' denoted both "to marry" and to "circumcise" so that an early connection between circumcision and the nuptial ceremony appears clearly proved. Ancient Egyptian carvings show definitely that circumcision was performed as early as 3500 B. C.

The Jews, it is believed became acquainted with the custom through the Arabians. Their early records show it as a quasi-religious rite, and, with but few exceptions, they have practised it universally. The old testament contains numerous references to the ceremony, the first being in Genesis, XVII—10 which addressed to Abraham reads as follows:

"This is my covenant, which Ye shall keep, between me and you, and thy seed after thee. Every male among you shall be

circumcised", the 8th day being set as the day for the rite. As religiously as this custom was practised by the early Jews, it fell into abeyance during their wandering in the desert, but was revived again by Joshua. Among the Jews at the present day, circumcision is always carried out on the 8th day, unless the day falls on the Sabbath or unless the health of the child does not permit it. The operation is performed by the 'Mohel' (operator) who is qualified only after he himself has performed this operation upon his first born son. The Rabbi when thus qualified is usually called for to perform the ceremony.

In the days of the Roman Empire, rigorous laws were passed forbidding the circumcising of Jewish proselytes. This was because the Jewish people still had numerous privileges which were denied the gentiles and many of the Romans were taking up the Jewish faith. When Flavius Clemens, a nephew of the Emperor Titan and Domitian, embraced Judaism, and was circumcised, he paid for the violation with his life. Later, the penalty for circumcision among the Jewish proselytes was modified, the violator being subject to confiscation of property and banishment. The death penalty was retained however for the surgeon performing the operation.

But circumcision has been practised for many years among people and races other than those of the civilized. It was known by the early Greeks to be practised by the Ethiopians, who it was believed did not learn it from the Egyptians. By numerous ethnologists it has been described as a custom among savage tribes of Africa, Australia, the Malay Archipelago, and the South Sea Islands before the advent of the White Man. With practically all of these tribes the operation was in the nature of an invitation to manhood or a pre-nuptial rite and was carried out with ceremonies, feasting and dancing, befitting the occasion.

Among Gentiles circumcision has never been practised universally nor made a religious or ceremonial rite. It is mentioned by many of the older surgeons and was practised as it is to-day for hygienic purposes and to facilitate normal sexual intercourse.

Circumcision destroys the extreme sensitiveness of the glans and prevents premature or too-rapid ejaculation. The sexual-intercourse is prolonged and the female partner is given sufficient time for an orgasm. Circumcision is thus believed to be an important factor in marital happiness, a fact borne out by the lower divorce rate among Jews.

Operation.—It is often said that the operation is quite easy.

It is easy but is not too easy. It should be performed with the seriousness befitting the antiquity and past and present religious associations. The results of the operation are vitally connected with the marital happiness of two souls, who will have to be partners in life. When the operation is carelessly performed either too much or too little of the redundant skin may be removed, poor approximation of the cut edges with an imperfect healing may result, large haematoma of the entire penis may occur due to imperfect control of bleeding vessels, infections may ensue owing to faulty technique, etc.

While it is true that these faults seldom prove serious, yet their effects are sufficient to be apparent throughout the life of the individual. *The ideal is attained when just enough of the fore skin is retained to cover the corona and yet is easily and fully retractable; when the frenum is neither too tight nor too loose during erection; when skin and mucous membrane are united at the edge of the stump, and neither overlaps nor is turned under when the cut-edge is straight and not irregular and when the wound heals without haematoma or infection.* The operation should be done with the care afforded, other plastic procedures and requires more time and judgment than is usually accredited to it.

Preparation.—A few days before the operation the child, if suspected, may be treated for worms. On the day of the operation, no solids are given and if the operation is done early morning, all food and drink can be completely withheld. But if the operation is to be done after 9 a.m. a cup of milk or coffee and some barley, sugar may be given at 6 a.m. The penis and scrotum should be thoroughly washed with soap and water, dried and painted with pure alcohol, alcohol picric or tincture of iodine. In children with very delicate skins, picric and iodine should be avoided.

Anaesthetic.—In very young babies no anaesthetic is necessary. In children upto 7 years, general anaesthesia, ether or C_2E_3 may be given. For older patients who are nervous, General is to be preferred. In other individuals local anaesthesia may be utilised. There are two methods of producing local anaesthesia:

- (1) Subcutaneous injection all around the root of the penis producing nerve-block.
- (2) By producing a cutaneous wheel at the level of the coronal sulcus. When the second method is employed,

it is necessary after the outer-leaf of the prepuce has been anaesthetized to retract the fore-skin and inject the inner leaf in a similar manner.

There are two methods of doing the operation:

- (1) The clamp method.
- (2) The dorsal slit method. Personally I am in favour of the latter method, as the clamp may not be suitable for all kinds of cases and also the second method gives a good training in flap cutting which is a valuable asset for a doctor. I shall describe the latter method only.

The muco-cutaneous junction of the prepuce is caught with Kocher's forceps in three places:

- (1) Posteriorly and,
- (2) Dorso-laterally. A moderate degree of forward tension is made on the prepuce through these forceps and the slit is made with scissors. The cut should go only as far as the intended line of circumcision. The incision is then made at right angles to it. First right and then left, following the angle of the corona but keeping a little in front of it. Not so much should be removed near the frenum. The redundant skin is removed, certain amount of trimming if necessary should be done, the bleeding points should be ligatured with fine catgut. The wound is sutured with fine catgut and at four quadrants at right angles to each other, the stitches are left long. A thin strip of vaseline gauze is applied over the suture line in between the long ends of the four-stitches and the long ends respectively tied over the gauze and then cut short.

The external urethral orifice is inspected and suitably dealt with if there is stenosis.

Dressings are applied and may be changed as often as necessary.

In elder children, post operative erection may be prevented by bromides.

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- (3) Choice—*System of Surgery*.
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SOME BASIC PRINCIPLES IN BONE PATHOLOGY

BY

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THE problem of bone growth, the nature of the so-called osteoblast and osteoclast, the function of the periosteum, the chemical constitution of the bone-salt and the factors determining its deposition in the frame-work of the skeleton all have attracted the attention of physiologists, anatomists, bio-chemists and surgeons.

In no branch of Medicine has so much confusion arisen than over the pathology of ossification, calcification and decalcification. This is the result of inaccurate observation and the linking up of pathological entities with pathological processes, which may or may not be present. For example, the decalcification of the femur, the result of an infection, is attributed to hyperaemia, while the decalcification of the femoral neck, the result of a fracture, is attributed to a lack of blood supply. One condition-decalcification-attributed to two exactly opposite pathological processes.

In this lecture, then, in an endeavour to clear away certain misconceptions in this controversial subject of bone pathology, I will deal with but a few basic principles—elementary facts. Principles which, though very elementary, will, I trust, help the student in his study of the pathology of bone diseases.

1. **The Periosteum.**—The first question which demands an answer is—what is the function of the periosteum? Is it osteogenic or is it not?

Macewen (1912) has definitely proved that periosteum is not osteogenic. He removed a strip of periosteum from a dog's radius, laid the periosteum over the muscles and closed the skin wound.

The periosteum became a fibrous cord, there was no formation of bone.

He also showed that in fracture of a bone from which the periosteum had been removed, the formation of a new bone (callus) was more abundant than under ordinary conditions. Excess formation of bone is the result of the absence, not of the presence of periosteum. When a portion of bone, deprived of its periosteum but retaining its osteogenic layer, is transplanted to another part, new bone will be formed from the cortex.

The function of periosteum then is to build and shape bone and to carry blood to bone; that it is a bone-forming membrane is not now accepted.

2. **The Bone cell or Osteoblast;** and

3. **The Mesenchyma.** Leriche and Pollicard (1926) treat bone as a connective tissue, which it is, and maintain that bone, fibrous tissue and cartilage are each the end result of development from a common mesoblastic stem. From this common mesoblastic stem, the mesenchyma of the embryo, develop all body cells, be they bone cells, cartilage cells or fibrous tissue cells. There is nothing specific about any of these cells, in fact, they are all of the same type. The osteoblast is not now regarded as a cell endowed with the specific power of laying down new bone.

A similar power to effect the deposition of calcareous salts is possessed at certain stages of development by the cells of cartilage and of fibrous tissue. Whether or not, inorganic salts will be deposited in a tissue, depends on the *local influence of Enzymes* and is under the *general control of Endocrines*.

Bone cannot become transformed into cartilage nor can fibrous tissue become transformed into cartilage or bone, unless a dedifferentiation of this tissue into primordial mesenchyma occurs. The dedifferentiation having taken place, then a redifferentiation can occur, according to requirements guided and sometimes misguided by the endocrine balance of the body, by hormonal influences and by other factors.

An example of this is seen in osteitis fibrosa cystica where, as a result of endocrine disturbance, associated with hyperparathyroidism, bone becomes dedifferentiated to re-appear as fibrous tissue. The same process is at work when in myositis ossificans progressiva the fibrous tissue of intermuscular septa become changed into bone; also when bone appears in a semilunar cartilage or in an intervertebral disc.

An osteoblast is simply a fibroblast enlarged on account of its activity and its osteoblastic power lies in its ability to absorb and facilitate the transference of calcium from the colloidal form in which it is present, in blood and lymph to the form in which it is present in bone.

4. **The Osteoblast.**—The osteoblast is also to be regarded simply as a foreign body giant cell, which helps to absorb dead bone by halisteresis and to remove dead bone as granules, which it does much less efficiently than does the serum effusion in which a sequestrum is bathed.

5. **The Proosseus substance of Leriche and Pollicard.**—This is

preosseus substance, pabulum, young connective tissue, call it what you will, must form between the fractured ends of the bone. The initial trauma will produce a hyperæmia but not until this hyperæmia subsides can calcium salts be redeposited in the young connective tissue.

Inadequate immobilisation means disturbance of this preosseus tissue, interruption in its continuity: the repeated traumata of slight movements will constantly add to the hyperæmia, which will mean more and more decalcification of the bone ends.

Finally when the hyperæmia subsides as a result of the fibrosis of healing, (the enzymes from the dead fibroblasts modifying cell activity and lowering circulation) then only can the local calcium excess be utilised, but to what good? There is no continuous mass of connective tissue; there is sclerosis in the bone across the concave ends. Bony union cannot now take place.

This is constantly seen in fractures of the carpal scaphoid and of the neck of the femur.

For years, this has been put down to an atrophy of bone, the result of an inadequate blood supply, through the ligamentum teres to the proximal fragment of the femoral neck.

The function of the ligamentum teres however has been exaggerated while that of the periosteum of the femoral neck has been overlooked, though text-books of Anatomy lay stress on the cervical ligaments, the retinacula, as bearers of the vascular supply to the neck of the femur. In a fracture of the femoral neck not only the proximal but the distal fragment also shows decalcification.

This is not an atrophy, the result of a lack of blood supply, which would be a slow and gradual manifestation but a rapid and early rarefaction, the result of a hyperæmia due to trauma, shortly to be increased by the relative hyperæmia of disuse. If the decalcification is greater in the proximal fragment, it is due to the additional blood supply to that fragment through the ligamentum teres.

Here, as in every case, a dedifferentiation of tissue to mesenchyma, followed by a re-differentiation to bone has taken place.

In the case of fractures into joint capsules, another factor is present which helps to account for non-union.

It is wellknown that synovial fluid is not favourable to the formation of bone.

In the case of a joint, neither the fibrinous exudate nor the local calcium excess can be maintained in close approximation to the fractured ends as is the case in fractures of bones embedded

in soft tissues. The fibrinous exudate and the calcium excess are lost as it were in the articular hæmosynovial effusion.

If, however, even after much decalcification of fragments has occurred, the ends can be brought into opposition before definite sclerosis has made union impossible, and if the lessening of the effusion makes utilization of the calcium possible then union can occur but at the cost of the femoral neck.

The Smith Petersen nail, by ensuring immobilization and the Winnett Orr treatment of compound fractures, by minimizing calcium loss, by minimizing the volume of discharge, have helped to solve the difficulty.

The bone graft.—When non-union is established then a bone graft operation does facilitate union—Why?

- (1) In the preparation of the bed for the graft the sclerosed ends of the fragment are removed and revascularization with mobilization of calcium salts is ensured.
- (2) The graft augments the local supply of calcium.
- (3) The accurate fitting of the graft assists in securing immobilization.

If an autogenous graft is employed, then live bone cells, fibroblasts will be added to help in the decalcification of the graft. The term graft as pointed out by Leriche and Policard is a misnomer. It is a transplant, not a graft. A graft grows; it is not absorbed and replaced. A transplant is always decalcified and its fibrous elements reduced to thin primordial condition before new bone forms. Radiography sometimes demonstrates that the transplant disappears entirely before there is evidence of new bone formation, but the two processes of absorption and formation of bone usually proceed synchronously. Within the bone and the transplant, lacunae, canaliculi, Haversian canals are widened by halisteresis—*i. e.*, the absorption of calcium by fibroblasts.

The calcium store of the transplant is utilized by continued surface erosion until it is entirely absorbed. It has never been within the circulation of its host and so there is no hyperæmia in the transplant, it has no circulation of its own. The transplant remains avascular and finally becomes nothing more than a sequestrum, save that a transplant becomes completely decalcified while a sequestrum tends to be surrounded by a vascular scar tissue the result of infection which limits decalcification, and the sequestrum has to be removed if not extruded. In a radiograph

a transplant will tend to become osteoporosed while a sequestrum becomes osteosclerosed.

The granulation tissue surrounding the transplant becomes fibrous tissue and takes the place and function of periosteum.

9. **The clinical signification of pathological ossification.**—Here we have to consider the formation of subperiosteal bone and heterotopic bone:—

- (1) The first group includes the ossifying sub-periosteal hæmatoma of the shaft of a long bone and also the condition known as traumatic myositis ossificans.
- (2) The second group includes the localized formation of bone in scars and tendons and also the generalised form of myositis ossificans progressiva, also the formation of bone in semilunar cartilages and in costal cartilages, scars, and so on.

A subperiosteal hæmatoma which ossifies is a traumatic osteoma. Myositis ossificans is simply due to a sub-periosteal hæmorrhage, which has spread along intermuscular fascial planes, because muscular efforts or the manipulations of treatment have ruptured periosteum, torn fibrous septa from their bony attachments, lacerated muscle sheets, and allowed blood to spread, and along the track of the hæmorrhage an osteoma forms. Traumatic osteoma then is the correct name for an osteoma, the result of contusion both where the hæmorrhage has been limited by the periosteum and where it has been allowed to spread by laceration of periosteum and muscle sheets. In the first instance, the bone tumour will be an oval mass with smooth rounded edges, in the second an irregular mass. It is from the bony attachments of these structures that the traumatic osteoma grows and the osteoma remains attached to bone, and follows inter-muscular planes.

A ruptured rectus abdominis at a tendinous inscription never forms a traumatic osteoma, because it is not in contact with a supply of calcium. The pathological process which produces a traumatic osteoma is just the same as stated above. Trauma results in hyperaemia, the adequate blood supply: the hyperaemia, brings about a decalcification, which provides the adequate supply of calcium—local calcium excess: formation of fibrinous exudate, fibrinous blood clot, dedifferentiation to primordeal mesenchyma; then follows the decrease of blood supply with deposition of the bone calcium salts and ossification. No traumatic osteoma is clinically apparent before three weeks and is not consolidated as

firm bone till about six weeks after the injury and its formation may be delayed even longer than that.

There is no difficulty in differentiating a traumatic from a biotrophic osteoma, a cancellous osteoma, so commonly found towards the distal end of the diaphysis of a long bone.

This cancellous osteoma is congenital, is an abnormal attribute of normal growth. It is a simple exostosis and in the recent state is capped with hyaline cartilage, not epiphysial cartilage. Such a biotrophic osteoma has some features in common with the biodystrophic deformity referred to as diaphysial aclasis, better metaphysial aclasis. The direction of these congenital osteomata is more or less constant and that these at one time marked the metaphysis is shown by the fact that their roots have been prolonged distally as the bone has increased in length.

The second group—the formation of heterotopic bone is a slightly different process.

An osteoma may form within muscle, quite apart from and unrelated to bone. In such a case there has been an injury to muscle with rupture of fibres; an intramuscular hæmorrhage with proliferation of fibroblasts and with a dedifferentiation of tissue to mesenchyme and with the decrease of hyperaemia redifferentiation to fibrous tissue—practically scar tissue. Contraction of cicatricial tissue then follows, with further devascularization and a condition of very low metabolism. Under such circumstances calcium is deposited in crystalline form from the percolating blood serum and lymph. These crystals are foreign bodies. They irritate, histamine is liberated, and causes some dilatation of such vessels as remain. Fibroblasts are proliferated, the tissue in which the calcium was embedded become soft and liquified—a dedifferentiation to mesenchyme has occurred and present in this small area we have a relative hyperaemia, a local calcium excess and a proliferation of fibroblasts the three factors required for the formation of new bone.

Exactly the same process is gone through when bone is formed in coeliotomy scars, in old T. B. lesions, in the choroid of the eye ball long after some panophthalmitis has brought about its collapse.

In old tuberculous caseous lymph glands calcification is common, and calcareous nodules will remain as such and usually do so, because the deposit of calcareous salts has not been followed by vascularization. Should vascularization occur, heterotopic bone will form.

The same identical pathological process explains the

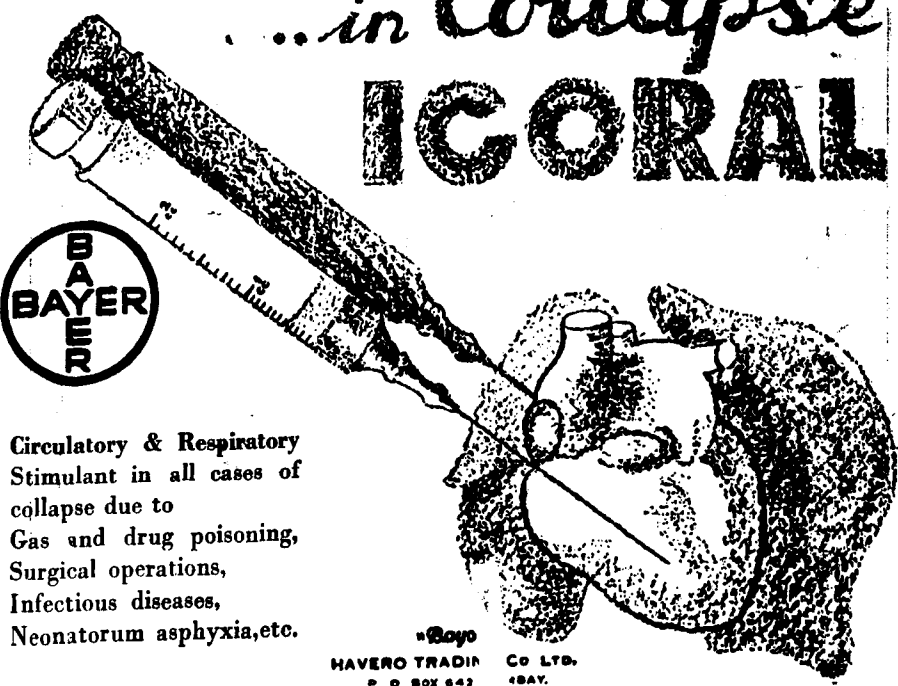
formation of bone in the perichondrium of costal cartilages, in the centre of synovial fringes, in loose bodies in joints, in semilunar cartilages, in intervertebral discs, in sacromata and in the condition known as fibrositis ossificans progressiva. Dedifferentiation to an embryonic stage, redifferentiation as fibrous tissue cartilage or bone.

If the student will grasp that there is a balance between calcium content and the vascularity of mesenchymatous tissues and remember that

- (1) Normal circulation means normal calcification,
- (2) Increased blood supply means decalcification,
- (3) Decreased blood supply means increased calcification,
- (4) Blood supply cut off means unchanged calcification,
- (5) Decreased blood supply in the presence of low vitality, low metabolism, means, pathological calcification.
- (6) And that an adequate blood supply plus a local calcium excess plus the presence of fibroblasts means the formation of bone,

He will have laid for himself a foundation upon which to build up his ideas of the pathological processes that give rise to the osteoporosis of tuberculous disease, the osteosclerosis of syphilis and to the many diseases blemished by an eponymous literature such as those we commonly recognise as Perthes, Schlatter, Kohler, Kienboch and others.

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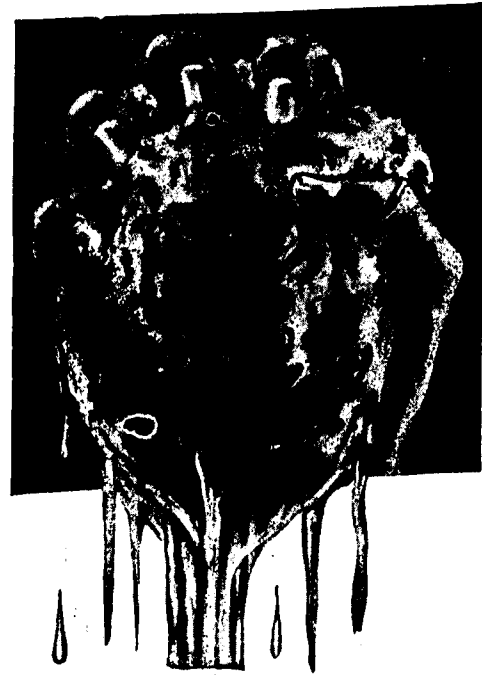
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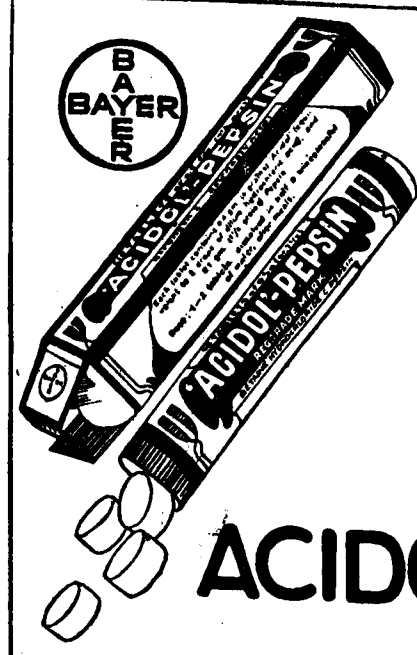
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EMPHYEMA

BY

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THE term empyema strictly means pus within a cavity but usage has limited its application to pus in the pleural cavity unless otherwise stated.

We shall first mention a few facts dealing with the physiology of the pleural cavities. The pressure in the potential cavity formed by the visceral and parietal layers of the pleura is termed the intra-thoracic or the intra-pleural pressure. The intra-pulmonic pressure, *i.e.*, inside the lungs and the bronchi is the same as the atmospheric pressure. On account of the elastic tissue which they contain, the lungs are constantly tending to collapse. Because of this property of the lungs, the intra-thoracic pressure under normal conditions is always a negative pressure, that is to say, it is less than the atmospheric pressure. This negative pressure varies greatly with a number of factors. It ranges from about 10 m.m. at the end of quiet inspiration to about 5 m.m. at the end of expiration. Even after the most forcible expiration, the intra-thoracic pressure is still negative.

The negative pressure of the thoracic cavity affects also the organs in the mediastinal space, the chief ones being the heart and the great blood vessels. The functions of these organs may be profoundly disturbed by an alteration in the intra-pleural pressure. It is only recently that this subject has been investigated, and its importance will be evident when we study the indications for a closed or open method of Empyema.

We shall first consider the changes which occur when a communication is made between the pleural cavity and the exterior. If the opening is relatively small, a small quantity of air enters the pleural cavity, and the lung just partially collapses. There is a demonstrable current of air through it with each inspiration and expiration.

If the opening in the chest is so large that respiration is not accompanied by any demonstrable changes in intrapleural pressure, that is, if the intra-pleural pressure is always that of an atmosphere, a complete collapse of the lung takes place.

If the mediastinum were a rigid structure, then even if one lung were completely collapsed, the other lung would be able to maintain life. The atmospheric pressure in the intrapleural

space, however, displaces the mediastinum to the opposite side and the vital capacity of it is further reduced by its encroachment on the opposite side. Displacement of the mediastinum, however, does not occur when it is fixed in position.

Mediastinal Flutter

This is the term used to indicate a movement of the mediastinum from one side to the other accompanying inspiratory and expiratory movements. This to and fro displacement of the mediastinum may produce a flapping sound which is both shocking to the patient and disconcerting to the operator. It is more commonly seen in traumatic conditions associated with great effort and anxiety on the part of the injured person than in operative conditions, when the patient is anaesthetized. The respirations are irregular and usually deep. Cyanosis and tachycardia develop. The blood pressure falls and the whole picture is alarming. Mediastinal flutter is accompanied by decreased tidal exchange, due to the lung on the intact side not being effectively aerated. It can be stopped by decreasing the size of the opening in the chest or by inflating the lungs with positive pressure. The importance of mediastinal flutter becomes apparent when we contemplate the open method of drainage of the pleural cavity. One must be sure that there is fixity of the mediastinum before opening the pleural cavity and allowing free communication between it and the exterior.

Aetiology:—Men are affected more frequently than women, and children, more often than adults. About 45 % of all empyemas occur in children under 10.

Empyema frequently develops as a complication of such conditions as lobar pneumonia, broncho-pneumonia, measles and other exanthemata, tonsillitis, pharyngitis, influenza and other infectious processes. There seems to be a definite relationship between the incidence of acute upper respiratory diseases and the incidence of empyema. This does not mean that pneumonia does not enter into the history of individual cases of empyema, for this is almost invariably the case. The conclusion is that whether or not a given pneumonia is to be complicated by empyema depends to some extent upon the character of the preceding infection of the nose or throat, caused perhaps by organisms with an affinity for the pleural tissues. The infecting organisms found in empyema include the pneumococcus, the streptococcus especially the haemolytic strains, the staphylococcus, more rarely the tubercle bacillus, Friedlander's bacillus, coli bacillus, typhoid bacillus,

streptothrix, several amoebic organisms and entamoeba histolytica. In many cases there is a mixed infection, several types of organism being obtained at the first aspiration. In the great majority of cases, the pneumococcus or the streptococcus haemolyticus is the organism found.

Pathology.—There are three possible routes by which organisms may reach the pleura :

- (1) Direct infection of the pleural cavity through penetrating wounds of the chest and particularly in connection with retained foreign bodies.
- (2) The organisms may reach the pleural cavity by way of the alveoli of the lungs during or after an attack of lobar pneumonia. About 75 % of cases follow pleuro-pneumonia. In such cases the sub-pleural tissues of the lungs become oedematous and the sub-pleural lymphatics contain numerous organisms. The pleural cavity is at first the seat of a serous effusion which may become progressively more purulent. The pneumococcus is encountered more often in these cases and the empyema may not be a prominent feature until after the crisis.

It has been established by a necropsy study of cases of acute empyema that died during the war, that the infecting organisms may reach the pleura by way of the interstitial tissues of the lung to which they gain access usually through some defect in the lining of the bronchi. Here, the sub-pleural tissue becomes involved and copious exudation occurs into the pleural cavity. The fluid is at first serous but may be loaded with organisms and becomes progressively more purulent. At first a layer of fibrin which soon becomes organised covers the pleura. The streptococcus haemolyticus is often encountered in this type of case. In some cases the infection is tuberculous and associated with a tuberculous lung underneath.

- (3) Acute Empyema arising by the blood-stream route is less common and very few patients show positive blood cultures until just before death except in cases of pneumococcal lobar pneumonia. Empyema is often seen following streptococcal septicæmia, and when it occurs it is but a manifestation of a wide-spread infection involving other serous cavities such as the

pericardium and peritoneum. A pyogenic process in the upper abdomen may rupture through the diaphragm and may cause an acute empyema. A sub-diaphragmatic abscess may be secondary to gastric or duodenal ulcer, or may be to a liver abscess which more often ruptures into the lung than through one of its bronchi.

Morbid Anatomy:—In streptococcal empyema, the liquid is at first a thin greenish brown liquid which becomes sero-fibrinous and later purulent. In a pneumococcal empyema the fluid is usually richly cellular and may contain masses of fibrin and there is more lymph formation. As a result of the irritation of the pleural membrane and the deposit of fibrin, the pleura may become greatly thickened; this is especially the case in chronic cases. The empyema may be general or localized by adhesions between the lung and the chest-wall, or between the lung and the pericardium, or between the lobes of the lung (inter-lobar empyema).

Symptoms:—The onset may be insidious, especially when empyema complicates a severe illness like lobar pneumonia or broncho-pneumonia or follows a simple pleurisy. In these cases, the maintenance of the fever, the absence of a crisis and the persistence of the physical signs in the chest and of constitutional symptoms suggest the presence of suppurative complications. The clinical course varies somewhat with the type of the infecting organism. In pneumococcus cases, except in children, the empyema usually occurs late in the course of the pneumonia. Streptococcus pneumonia and empyema are usually secondary to acute upper respiratory disease and frequently complicate an acute infectious disease such as influenza or measles. The empyema may occur early or may be associated with or be secondary to a broncho-pneumonia. In those cases of early onset, there is pain in the chest, fever, nausea and vomiting. Where the empyema occurs in the presence of a wide-spread disease of the lung, either lobar pneumonia or broncho-pneumonia, the vital capacity, already diminished by the infiltration of the lung may be further affected by the encroachment of pleural fluid. The respirations therefore become rapid and shallow and cyanosis is often marked. Signs of severe toxæmia are present with high fever and rapid weak pulse. There is often a constant cough—reflex in origin—which tends greatly to aggravate the pain.

If left untreated, an empyema sooner or later will burst through the chest-wall and form a localized abscess under the

skin. This usually occurs in front of the chest through the second or fifth intercostal spaces, but is sometimes seen in the axilla where it resembles an axillary abscess. When the pus is outside the chest wall, and underneath the skin, it may track far and wide before finally bursting through the skin. This condition is called "Empyema Necessitatis", and when situated on the left side of the chest the fluid may be felt to pulsate owing to the transmitted pulsation of the heart (Pulsating Empyema). Sometimes a neglected empyema may burst through a bronchus and the pus be coughed up. It has been stated that spontaneous cure has occasionally followed this accident, more especially in case of interlobar empyemata, but some doubt whether these cases are not really abscesses of the lung rather than empyema.

An empyema does not burst through the abdomen. It will be noted in passing, that suppuration underneath the diaphragm has a tendency to invade its upper surface also, but suppuration above the diaphragm never extends downwards, possibly it is due to the negative pressure on the upper surface of the diaphragm exerting a sort of suction action and possibly also it is due to the flow in the lymphatic channels, being directed from the under-surface of the diaphragm to its upper surface.

The physical signs are those of fluid within the chest. It may be quite evident in certain cases but in the presence of massive pneumonia, the diagnosis of empyema upon physical signs alone may be very difficult or even impossible. Roughly the physical signs are impaired respiratory movement on the affected side, the intercostal spaces may be flattened, or even bulging. The heart will be displaced towards the sound side.

On percussion, there is marked dullness over the lower portion of the chest to varying height, while at the apex of the lung and below the clavicle Skodaic resonance may be present.

On auscultation, breath sounds are usually absent, though in children they may persist in a big empyema and even be tubular in character. Loss of vocal fremitus and marked aegophony are also important signs. Dyspnoea, rise of temperature and pulse rate and leucocytosis are nearly always present.

Complications.—The complications of empyema are particularly serious and materially influence the prognosis. Bilateral pneumonia, bilateral empyema, pericarditis, peritonitis, mediastinal infections, are the common complications.

Diagnosis:—The diagnosis should always be confirmed by puncture with an exploring needle and syringe, for by no other means can the condition be distinguished with certainty from a

serous effusion. A considerable amount of information can be obtained from a study of the aspirated fluid and the infecting organism should always be ascertained before an operation, as it may influence greatly the nature and scope of the procedure to be undertaken and also the prognosis.

A simple effusion should show positive signs of subsidence after a fortnight. The presence of rigors or continuous fever, progressive emaciation and constitutional disturbance unaccounted for by some primary lung disease indicates that the effusion is an empyema. A large peri-cardial effusion producing dullness across the left base and signs of lung compression are recognised by the disappearance of the cardiac impulse, by muffling of the heart sounds, by dullness in the right fifth intercostal space and by the character of the radiographic shadow.

Empyema can be diagnosed from a sub-diaphragmatic abscess by the difference in level of the dulness. This, with the patient sitting up, is higher in the axilla than at the sternal border in the former condition and lower in the latter. X-ray has proved to be of valuable diagnostic aid provided the patient is not too ill to be shifted into the upright posture.

Latent Empyemas are divisible into two groups :

- (1) In the one it is recognised only during systematic examination, sometimes only at autopsy. These cases occur mostly in children and there is usually a previous history of an acute febrile illness since recovery from which the patient has not been as well as before.
- (2) The other group is that most often seen by the Surgeon, and includes those cases in which the Empyema, often latent as regards the symptoms, perforates the chest wall. Diagnosis has to be made from caries of the rib, abscess associated with caries of the spine, gumma and actinomycosis. The diagnosis can in the first three conditions be determined by X-ray examination. In Actinomycosis, pain is usually a more prominent feature, while the character of the pus when aspirated or discharging from a sinus is characteristic.

Character of the exudate.—A clear straw-coloured fluid with few cells—and those largely mononuclear—may indicate the so-called Idiopathic pleurisy which is usually of tuberculous origin.

In the early stages of streptococcal infection of the pleura,

the fluid is thin and watery, greenish or brownish in colour, and without large fibrin clots. Microscopic examination shows a varying number of poly-morphonuclear leucocytes. Many in various stages of disintegration and the presence of streptococci often in enormous numbers may be demonstrated.

The pus from *pneumococcus Empyema* is thick and yellowish-green in colour with fibrin masses which often plug the aspirating needle or drainage tube.

Prognosis.—The prognosis in acute empyema is largely dependent upon the prognosis of the underlying pneumonia but experience has demonstrated that this may be greatly influenced by the type of treatment employed and especially in the streptococcal cases. Stating generally, the prognosis in pneumococcal cases is good, especially if they are small and unilateral. In streptococcal cases it is not so good. A tuberculous empyema always carries a bad prognosis.

Formerly, when open drainage was instituted, for every case of empyema, whether streptococcal or pneumococcal, it was found that the pneumococcal cases did well, whereas there was a high percentage of mortality in the streptococcal cases. As we shall see presently, it is from this observation that the mode of treatment has been altered in the two different kinds of cases. The prognosis also depends on whether the empyema is a typical synpneumonic one or not. If it is concurrent with the pneumonic process in the lung, the prognosis is not so good.

Treatment.—Most of the cases are seen by the surgeon very late. Especially in the Hospital one sees cases in whom the pus has been in existence for at least two weeks and in one case that I saw recently even for three weeks. The patients are already in a highly toxic condition, running a high temperature. There should be no delay in these cases and surgical treatment, whether it is repeated aspiration, closed drainage or open drainage, should be adopted at once.

The later operation is undertaken, the more will the lung be bound down and fibrosed and the thicker the pleura will be found to be. As a result the prognosis both as regards recovery and rapidity of healing will be best if operation is undertaken early.

Generally speaking, there are two important points in the consideration of the treatment of an Empyema :

- (1) The nature of the operation performed may depend on the organism present;
- (2) It is not sufficient to apply a routine treatment to all

cases, consisting in removing a portion of a rib and inserting a tube. This used to be performed formerly and was followed by a large number of persistent sinuses.

As regards the organism causing the empyema in a particular case the rule to be followed is:—

A streptococcal empyema must not be opened until there is frank pus. In a pneumococcal infection, the lung is usually fixed by adhesions by the time it is diagnosed and there is little risk in opening and draining the pus. In a streptococcal empyema until pus has formed, the lung is not fixed and opening the pleural cavity exposes the patient to all the risks of an open pneumothorax. As was pointed out in dealing with the physiology of the pleural cavity, in the absence of stabilizing adhesions, the mediastinum suffers definite displacement from one side to the other with differences in pressure in the pleural cavities. This mediastinal flutter as it has been called renders cardiac action very difficult and cumbersome and this certainly should be avoided in a patient who is already suffering from severe toxæmia and in whom perhaps the lung also is in a state of pneumonic consolidation.

In such cases it is imperative that one should institute a type of treatment which will throw as little additional burden upon the cardio-respiratory physiology as possible. Either of two procedures can be adopted:—

- (1) Repeated aspiration ;
- (2) Air-tight continuous suction-drainage.

1. *Repeated aspiration* is indicated only in those cases which are gravely ill and in very small children, say under 5 years of age. Very few cases are cured by this procedure. In the majority of them subsequent operation is necessary but when performed later, is better tolerated by the patient. The object in repeated aspiration is to withdraw the pus without producing a pneumothorax. It diminishes toxæmia and improves the general condition of the patient.

Sometimes by aspiration, early pneumococcal cases, especially in children, clear up after one or two aspirations. In a very large collection where the patient is very ill, aspiration should first be done and open operation only later. In cases of bilateral Empyemata, it is wiser to aspirate both sides to commence with and do the open operation on the side that is affected more, at a later time.

In Empyemata due to the streptococcus, repeated aspiration should be done. The results of treatment by repeated aspirations are far better than those of early drainage. The pus is usually thin and watery and aspiration may be persisted in until it becomes thicker and more creamy. When this occurs open drainage can be instituted. When there is a pneumonic process of the underlying lung at the same time as the empyema—sometimes also known as the syn-pneumonic type of empyema, aspiration should be adopted at first and open drainage only later on. Aspiration can be accompanied by oxygen replacement in these cases.

2. *Intercostal drainage of empyema.* Air tight continuous suction drainage can be simply performed by introducing a catheter through a short intercostal incision under local anaesthesia. For this purpose a special trochar and cannula with a side-arm is employed. Through this the catheter may be introduced without allowing air to enter the chest. An air-tight dressing is then applied. The catheter is attached to a long tube filled with water and the lower end of the tube is placed beneath the surface of water in a large bottle beneath the patient's bed. A continuous syphon is established. In these cases a self-retaining DePezzer's catheter can be used. Only about 2" of tube should lie within the cavity. The intercostal method of drainage is advocated in cases of children. The child is kept in position in the bed, so that the wound is over a hole in the mattress.

Instead of one catheter, two DePezzer's can be inserted and then the cavity can be irrigated. Care should be taken in irrigating cases of acute empyema. Several cases of pleural shock have been recorded. The cause of shock is probably excessive pressure with lack of free exit for the fluid. The tube should not be kept longer than necessary. The tubes should be removed if the cavity does not hold more than one ounce of fluid. The first sign of gradual diminution of the cavity is usually the decrease of the sucking in of air on inspection through the wound when this is temporarily left open during the dressing.

Disadvantage of Closed Drainage.—(1) Incomplete emptying of the cavity, (2) Difficulty of ascertaining occurrence and amount of negative pressure in the cavity. (3) Difficulty of getting an air-tight junction between the chest wall and the drainage tube, (4) Difficulty of cleaning the tube when it is blocked (5) Impossibility of free irrigation of the cavity, (6) Difficulty of keeping the tube long enough to empty the cavity, but not so long that it impedes healing.

Good Points: It has saved many lives.

- (1) Trivial operation under local anaesthesia
- (2) Control of Rate of evacuation of pus
- (3) Avoidance of large inrush of Air.

3. Drainage by resection of a rib or open method of drainage.—

This can be performed in most adults who are moderately fit. This consists in resecting a portion of a rib, opening the pleura, letting out all pus, inspecting the pleural cavity thoroughly and removing all solid fibrinous masses. This is followed by drainage of the pleural cavity. Some surgeons however advocate that the pleura like other serous membranes has a considerable power of resisting infection when properly treated and they perform primary suture without drainage. They claim that in about 25% of cases primary healing by first intention is obtained but this method of treatment has not come into general use.

After the pus is let out, the pleural cavity is drained by a short but large tube and this tube must not be long enough to press on the diaphragm or the lung. The portion of the rib to be resected should be as far back and as low down as possible, care being taken to avoid the scapula—the incision in the skin may be vertical or horizontal, parallel to the line of the rib. The drainage should be continued until the discharge becomes thin and serous. After the operation every effort should be made to encourage the lung to expand and is best accomplished by blowing water from one Wolf's bottle to another through a tube.

If the pleura is very much thickened the cavity is irrigated with Carrel-Dakin solution which dissolves the fibrin in addition to its antiseptic power.

Treatment of localised empyema.—Relatively rare—about 3 % of empyema, *viz.*, apical, interlobar, basal and diaphragmatic types. More commonly it is seen in the lower part of the chest posteriorly.

Treatment.—Similar to above—but great care must be taken to make the incision directly over the collection of pus and not to break down the adhesions and infect the rest of the pleural space. If left for a long time, they sometimes rupture into a bronchus. Drainage should be provided for before this occurs.

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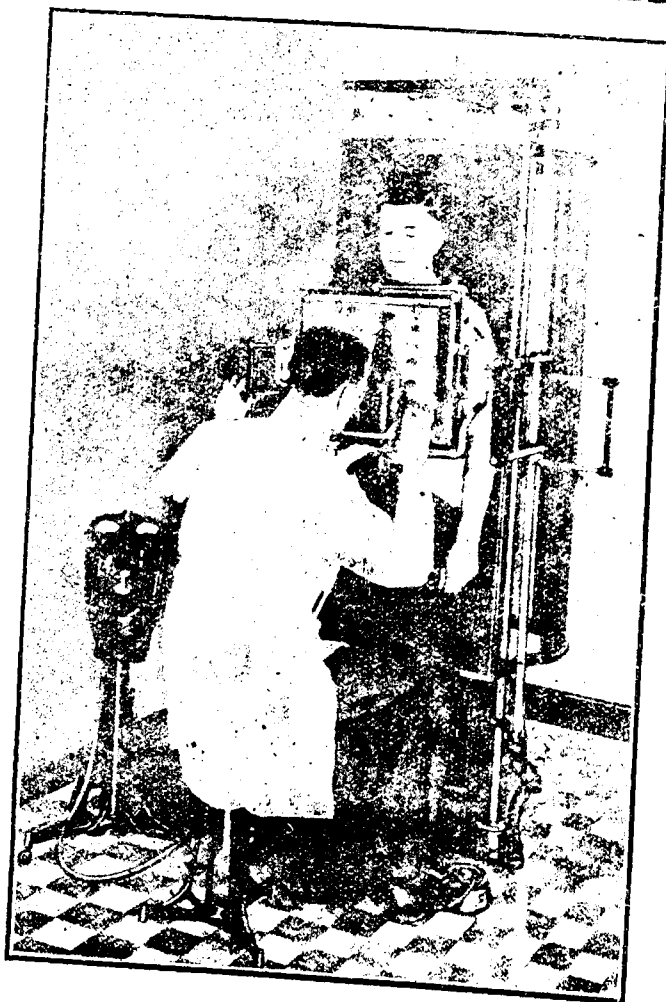
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