

so may alter the constitution of their idioplasm.* Nor do we lack clear evidence that this actually occurs. Thus Mairet and Combemale have subjected male dogs to chronic or acute alcoholic intoxication and found that the young exhibited various arrests of development and were predisposed to epileptoid seizures. The only explanation is that the alcohol had caused deterioration of the spermatozoa so that these conjugating with a healthy ovum nevertheless gave origin to impaired individuals. Nor are clear cases wanting of similar degenerations occurring in the progeny of alcoholic fathers and healthy mothers in man. The most clear example, however, of this order was afforded years ago by Constantin Paul in connection with the effects of lead poisoning. Studying a series of cases in which the father alone in the course of his work became the subject of plumbism, he was able to obtain the history of 32 pregnancies. Of these 12 resulted in the death of the fetus before term; 20 children were born alive, of which 8 died during the first year, 4 died during the second year, 5 died during the third year, and 1 died later. Two alone were found to be alive, one of these aged twenty.

The only conclusion from these observations is that substances which lead to intoxication in the parental system tend also to act deleteriously upon the germ cells. It will be seen that the individual offspring itself is in one respect the direct victim of the intoxication; inherited plumbism is lead poisoning acting initially upon one or both of the conjugating germ cells, but it must be clearly understood that the mental and other disturbances showing themselves after birth are not the results of the action of lead salts still circulating in the system and that thus the lesions of inherited plumbism are of a totally different order from those of acquired plumbism. These are one and all lesions of incomplete development.

3 A. *Neuropathies.* The effects of parental intoxications are peculiarly liable to show themselves in the form of conditions of imperfect organization of the nervous system. This is in accordance with the principle or law, the action of which we repeatedly see in evidence: the law, namely, that characters which are the last to be acquired by the individual, the race, or the species are those which are the first to be lost. Thus in the evolution of man, one of the last acquisitions—that acquirement whereby man is distinguished from all other animals—has been that of the higher mental organization. Another has been the development of relative insusceptibility to sundry infectious diseases. Thus the individual whose full development is arrested is particularly liable to manifest a greater or less degree of mental instability, the development of the higher nervous centres being incomplete; or again to manifest a peculiar susceptibility to the infections liable to attack human beings. Thus nervous instability of various orders may be regarded as an evidence of familial degeneration and as due to a condition acquired by one or other parent or to inheritance from previous ancestors, and noticeably may be accompanied by other stigmata of incomplete development and degeneration, liability to contract infectious disease, etc.

3 B. *Increased Susceptibility toward Infectious Diseases.* What I have stated regarding neuropathies may be stated *mutatis mutandis* with regard to infections. That is, the toxins of one or other infection circulating through the system, becoming absorbed by the germ cells and acting chemically upon the idioplasm, may result in the weakening of that idioplasm. As a matter of fact Gheorghiu has pointed out the frequency with which one notes the history of parental infection in cases of various monstrous developments.

It may be and it has been urged that here we are not dealing with the inheritance of conditions acquired by

*I say *may* because it has to be remembered, as pointed out by Meyer, that the cellular absorption of toxic substances is of two orders: (I.), absorption into the cell without resulting chemical combination (as in the case of the alkaloids), the absorbed substance interfering with metabolic processes, and (II.) absorption with chemical combination, the toxic substance becoming fixed, e. g., tetanus toxin in the cells of nerve tissue.

the parent, that the toxic substances produced these results not by their action on the body cells, but by direct action on the germ cells, and that so the inheritance is blastogenic, not somatogenic. We are quite prepared to grant that these inheritances are of blastogenic origin, nevertheless they are of individual acquirement. The individual consists of body plasm and germ plasm, and whether the defect tell primarily or secondarily upon the germ plasm of the individual we here have examples of conditions acquired by the individual transmitted to the offspring. We have to admit, that is, that the environment of the germ cells when present in the parental organism is capable of bringing about modifications in the germinal idioplasm, and so of producing variations in succeeding generations.

4. *Intoxications, Infections, and Diatheses; Direct Inheritance.* From the above considerations it will be seen that we have reduced the possibilities of the direct inheritance of acquired conditions to a relatively narrow field. We have not, however, completely removed all of them; certain conditions remain to be discussed. Is it possible, for example, that these various parental intoxications, telling as they do in one or other direction upon the parental constitution, tell also in a differential manner upon the germ cells, so that alcoholic inheritance differs from inherited plumbism, and these again from the indirect inheritances of syphilis and tuberculosis? *A priori* if these toxic substances, circulating in the parental system and gaining entrance into the germ cells, forming different combinations with the idioplasmic molecules, combine with, that is, or alter different side chains, we must conclude that it is possible. As a matter of fact, children of syphilitic parentage not the victims of intra-uterine syphilis, tend to show parasymphilitic lesions which are of a somewhat different order from the paratuberculous lesions of the progeny of tuberculous parentage. Here, however, it has to be admitted that fuller statistics are necessary regarding the lesions of those whose fathers and mothers respectively have suffered intoxication; for in maternal intoxication of any order, placental absorption of the circulating toxic substance must tell upon the fetal existence. Nevertheless, taking the germ cells as, from their potentialities, apparently representative of the body cells in general, the abundant studies upon immunity made during recent years give us the strongest grounds for believing that the different toxins act specifically and affect the idioplasm in one particular direction. At this point it will be seen that our theory falls into line with Ehrlich's side-chain theory of immunity.

In short, the facts gained from the study of immunity point to three possibilities:

(a) Where through disease or through introduction of toxins the cells of the body become immunized to one special infection, those cells are so modified that they now produce substances antagonizing the toxins of that infection. We might therefore expect specific parental immunity to be accompanied by specific immunity of the germ cells and a condition of relative immunity of the offspring.

(b) When through disease immunity is not attained, but on the contrary the parental tissues—as in progressive tuberculosis—become progressively weakened and susceptible to the deleterious action of the toxin, the germinal idioplasm may also be weakened and an offspring be developed more susceptible to the particular infection.

(c) The idioplasm of the germ cells, being relatively undifferentiated while within the reproductive glands, might not in this state possess side chains capable of being acted upon by the circulating toxins; so that while the parental organism in general is affected thereby in one or other direction, the germ cells may be uninfluenced, and the offspring in consequence may present neither increased susceptibility nor increased powers of resistance to the specific disease.

Here it will be seen that we are in a region of hypothesis pure and simple; indeed, our researches into the in-

heritance of specific morbid constitutional states have not been sufficient to indicate positively which, if any, of these possibilities accords with the ascertained facts. Even granting that the first of these possibilities is correct, it has to be noted that according to the principle already mentioned—that conditions last acquired are those soonest lost,—we should expect that acquired immunity of the germ cells, if obtained, would tend to be of slight duration, not strongly impressed. Hence for the development of acquired immunity as of acquired susceptibility, not a single act of immunization of the parent but repeated immunizations through several generations might be requisite before any marked and permanent influence showed itself. As a matter of fact, the various attempts to confer immunity by immunizing the male parent have in the main given negative results. Gley and Charrin alone have detected such; Ehrlich strenuously denies that it is obtainable.

Nevertheless I think it is useful to call attention to these possibilities in order that more exact studies be made which shall prove or disprove them. Thus far the importance of determining the part played by acquired disease, more especially in the father, in the development of morbid constitutional states of the offspring has not been sufficiently recognized. It has not been grasped by our profession that in the study of these states we have a more subtle means of determining this question of the inheritance of acquired states than can possibly present itself to the morphologists, dependent as they are upon the more extensive gross anatomical changes before they can determine whether any alteration has been impressed upon the offspring.

We have, as I have pointed out, definite evidence that constitutional disturbance in the parent affects the germ plasm, and having this, it is for us sedulously to collect all the evidence which presents itself in order that we may determine the limitations of this affection of the germinal idioplasm within the parental organism.*

The views here enunciated are, I know, contrary to the generally expressed opinions of morphologists. It is well to point out once again that the conception of the existence of micelli, ids, gemmules, etc., can only lead to the view that acquired characters, not being able to tell on these ancestral bodies, cannot possibly be inherited. Save in the matter of spontaneous variation I do not suggest that visible anatomical changes can be expected in the course of one generation; but this physico-chemical theory, by realizing the possibility of progressive modification in the constitution of the idioplasm while within the parental organism, is fitted to explain not only what we observe in connection with the effects of disease, but also the development of familial and racial characters, and to aid materially our comprehension of the nature of evolution in general.

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*More especially I would call attention to the valuable material in the possession of the great American insurance companies and the possibility of gaining information as to the effects of acquired disease of the parent upon the offspring by employing the mathematical methods elaborated by Carl Pearson (*vide article on Evolution*); if they can give mathematical expression to the influence of, say, acquired parental tuberculosis, upon the life period of the offspring, we shall make a most material advance.

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HERMOPHENOL, mercury phenol-disulphonate of sodium, is a white, amorphous powder soluble in five to seven parts of water. It is said to contain forty per cent. of mercury, and to be strongly bactericidal without caustic effect on the tissues. It can stand a heat of 120° C. without decomposition. Bérard uses it as a disinfectant for the hands, and in three-per-cent. solution for ophthalmia neonatorum, and Reynes injects 4 c.c. (3 i.) of a half-per-cent. solution, every two or three days, for syphilis.

W. A. Bastedo.

HERNIA.—This term, derived from the Greek *ἔρνος* (a spout), is employed for denoting the protrusion of a viscus from the cavity in which it is normally contained. Although it may be used to denote the escape of organs from other cavities, yet when unqualified it signifies, as does the vulgar term "rupture," a protrusion from the abdominal cavity. As usually employed it implies a pouching of the containing wall forming a continuous sac for the displaced contents. An escape of the viscera through a wound in the wall is better termed a "protrusion." The term "internal hernia" is employed to denote either the protrusion of the abdominal contents into the thoracic cavity—*i. e.*, *diaphragmatic hernia*—or the intromission of the intestine into one of the peritoneal fossae. The latter condition is, however, more properly classified as one of the forms of intestinal obstruction.

A hernia may occur at one of the openings of the abdominal wall normally present in fetal life, which has not become closed at the time of birth, and is then termed a *congenital* hernia; *i. e.*, a congenital inguinal hernia is one in which the abdominal contents push their way into the unobliterated processus vaginalis. All herniae developed subsequently to birth are termed *acquired*. They occur at the points where the abdominal wall is relatively weak. Hence arises the anatomical classification of herniae: inguinal, femoral, umbilical, lumbar, diaphragmatic, obturator, etc. The parts constituting a hernia are, first, the sac and, second, the contents. The sac consists of peritoneum continuous with the parietal peritoneum of the abdominal cavity. The coverings of the sac vary with the anatomical variety of the hernia, and are as a rule fascial with the exception of the skin. The shape of the sac depends upon the nature of the opening in the abdominal wall and the degree of restraint afforded by the fascial coverings. If the opening be circular with more or less sharp margins the sac as a rule is globular, as in umbilical and femoral herniae.

In inguinal hernia the sac is elongated, owing to the fact that the fascial coverings are tubular. If the hernia is incomplete the sac is sausage-shaped. On the other hand, it is pyriform in shape when the hernia becomes scrotal. Bands of the investing fasciae may constrict the sac in such a manner as to produce hour-glass forms; or from the same cause diverticula may result, the pressure being unequally exerted. The parts of a sac are termed the mouth, the neck, and the fundus or body. The contents of the sac usually consist of small intestine or omentum, or both; more rarely they consist of the colon, caecum, or appendix, sigmoid flexure, bladder, or uterine adnexa. When the contents consist of intestine alone the hernia is called an *enterocele*; when of omentum alone an *epiplocele*; when of both, an *enteroepiplocele*. Fluid may be found in the sac either free or encysted by adhesions—*hydrocele of the hernial sac*.

ETIOLOGY.—Causes predisposing to hernia are:

1st. *Heredity.*—Weaknesses in the abdominal wall predisposing to hernia are undoubtedly inherited.

Macready¹ estimates that twenty-five per cent. of patients with hernia give an ancestral history.

2d. *Sex.*—About six males suffer from hernia to one female. This is to be explained both by the much greater frequency of inguinal hernia in males, due to the necessary enlargement of the inguinal canal, and by the nature of their occupation.

3d. *Age.*—Except in the period of infancy, when the influence of congenital weakness upon the development of hernia is most marked, the greater number of herniæ occur in the active period of life, between fifteen and fifty.

4th. *Elongation of the Mesentery*, although generally given as an etiological factor in the production of hernia, is rather a concomitant result—with hernia—of causes such as weakness and laxity of the abdominal wall.

Exciting Causes of Hernia are: 1st. Occupations necessitating violent muscular efforts. 2d. Strains, as lifting, coughing, sneezing, and parturition. 3d. Conditions producing stretching of the abdominal wall, as pregnancy, ascites, obesity. 4th. Atrophic changes, as senility. 5th. Traumatism resulting in the replacement of normal by cicatricial tissue, or in the destruction of nerves, thus causing paralysis and atrophy of muscle.

CLINICAL VARIETIES OF HERNIA.

The clinical classification of herniæ is based upon the condition of the contents and the relation of the contents to the sac. Thus we have: (a) Reducible; (b) irreducible; (c) inflamed; (d) obstructed; and (e) strangulated herniæ.

(a) **REDUCIBLE HERNIA.**—A reducible hernia is one in which the entire contents can be wholly reduced to the abdominal cavity, without recourse to a cutting operation. Reduction is often effected by posture, but may be accomplished only by taxis. It is the most common class of hernia.

Symptoms.—The earliest subjective symptom in an acquired hernia is usually pain (ninety per cent. of cases).

In a hernia caused by a strain the pain may be sharp and accompanied by a sense of something giving way. Usually it is dull and dragging and, after a varying period of time, is replaced by a sense of fulness which is marked on standing or straining and disappears in the reclining position. Large herniæ are not infrequently accompanied by gastric or intestinal symptoms, which are probably reflex in character.

The objective symptoms depend upon the size of the hernia and the nature of its contents. A small hernia exhibits a tumor as a rule only on standing or coughing, and the latter disappears when the patient lies down. A large hernia is often not spontaneously reducible; but it increases in size in the erect position or on coughing, and if inguinal it may then be seen to descend into the scrotum or labium.

In both small and large herniæ a characteristic impulse is felt on coughing. In an enterocele there may be a gurgling felt or heard either at the moment when the bowel descends into the sac or on reduction of the hernia, and at the same time the tumor is more or less tympanitic. An epiplocele is dull or flat and feels more irregular and does not give so marked an impulse.

Diagnosis.—See Anatomical Varieties.

Treatment.—For taxis, see Strangulated Hernia. For operative and mechanical measures, see under Anatomical Varieties; also under Mechanical Treatment.

(b) **IRREDUCIBLE HERNIA.**—This term is applied to a hernia in which the contents, although practically normal in condition, cannot be wholly returned to the abdominal cavity. The chief causes of irreducibility are, first, adhesions of omentum to the sac or between the contents, or thickening of the imprisoned omentum (according to Macready the contents consist of omentum in ninety per cent. of cases); secondly, the formation of part of the sac by the gut itself (more fully described under cæcal and sigmoid herniæ); and, thirdly, the bulk of the hernia may

be so great that the contents cannot be received into the abdominal cavity.

Irreducible hernia is more frequently observed in middle life and in the umbilical variety. It is uncommon in children. It may be permanent or transitory. It is said that it seldom persists throughout life.

Symptoms.—The symptoms depend upon whether the hernia is large or small and upon the nature of the contents. Small epiploceles usually give no especial symptoms, while larger ones, from dragging upon the omentum, are liable to cause digestive disturbances and are prone to inflammation. In irreducible enterocele the condition is more serious. The imprisoned bowel is apt to become obstructed, which condition may be evanescent or may persist, and furthermore strangulation may supervene. Hence the symptoms vary from occasional attacks of colicky pain and flatulence, with more or less constipation, to those of complete obstruction and strangulation.

Treatment.—Herniæ which have only recently become irreducible are, as a rule, more or less inflamed or at least congested; so that rest in bed and the application of cold with moderate pressure are indicated. Prolonged efforts at reduction under such circumstances are more than likely to aggravate the condition, and hence considerable judgment has to be exercised in the employment of taxis. In old cases or those in which the above treatment does not suffice, operation is indicated. The contraindications to operation do not exist to the same extent as in reducible hernia, since the condition *per se* is more dangerous. In cases in which operation is distinctly contraindicated, as in the aged or diseased, or in which the hernia is so large that the return of the contents to the abdomen would not be feasible, the employment of some device for supporting the hernia and preventing its increase is indicated.

For umbilical hernia some form of abdominal support is useful. In large scrotal herniæ a stout canvas bag, supported from the shoulders, has been found advantageous.

The employment of special trusses, as the "hinged-cup truss" for inguinal hernia, can be recommended only when operation is out of the question, although Macready claims sixty-eight per cent. of reductions by its use. But even then a cure of the hernia is seldom attained.

(c) **INFLAMED HERNIA.**—When a local peritonitis occurs in the hernial sac the hernia is said to be inflamed. It is usually the result of trauma, either from a truss or from prolonged taxis, but it also occurs from disturbance of the contents as from obstruction or possibly enteritis. A strangulated hernia always becomes inflamed if the condition persists for any length of time. Irreducible herniæ are most prone to inflammation, especially the femoral and umbilical varieties. The process results in the production of lymph and fresh adhesions. The sac may become distended with fluid, and obstruction and strangulation may supervene.

Symptoms.—A sense of increased fulness, pain, and tenderness are present. There is usually a slight rise of temperature. There may be vomiting and constipation. If obstruction or strangulation occurs the symptoms of this condition supervene. On palpation there are tenderness and local heat, and the mass may be tense or semi-fluctuating. Inflamed omentum usually feels hard and nodular.

Treatment.—Rest in the recumbent position and the application of heat or cold. Enemata, but not cathartics, should be used. The pain may be relieved by anodynes.

(d) **OBSTRUCTED HERNIA.**—An obstructed or incarcerated hernia is one in which the passage of the contents of the intestine is inhibited, but in which the circulation of blood is not interfered with.

Obstruction is more common in irreducible than in reducible herniæ. As it is usually due to constipation, it occurs more often in herniæ containing the large intestine. Hence we find it more often in umbilical herniæ and in large inguinal herniæ in which the contents are the sigmoid flexure or cæcum. As large herniæ are more

common and as there is a lack of intestinal tone in the elderly, obstruction occurs most frequently during the period from middle life on.

The determining causes are usually neglected constipation, and the ingestion of indigestible food or food that leaves a large residue.

Symptoms.—The symptoms vary considerably, but are distinguished from those of strangulation by their gradual development. Constipation at first is not generally absolute. The tongue is coated, the breath is foul, and there is complete anorexia followed by vomiting, which is not usually persistent and seldom stercoraceous. There is colicky pain which may be severe, but there is no collapse. Distention is more or less marked. The hernia, if before reducible, becomes irreducible and increases in size. Impulse, however, is obtainable, which serves to eliminate strangulation. The tumor is often doughy and may be dull on percussion, although imprisoned gas may give a tympanitic note. The prognosis is usually good.

Treatment.—The emptying of the bowel is the first consideration, and this should be accomplished by high enemata and by massage of the hernia. Cathartics should not be given until the tumor has decreased in size.

If there be no response to treatment, and if strangulation be suspected, operative interference should be at once instituted, as delay may be fatal. Recurrence of obstruction may be to a large degree averted by proper attention to the food and bowels.

(e) **STRANGULATED HERNIA.**—A strangulated hernia is one containing intestine in which both the passage of bowel contents and the circulation of blood are interfered with. For the time being it is not spontaneously reducible.

This condition is rare in infancy and is most common in middle life and old age. It occurs more often in small than in large, and in old than in recent herniæ; although rarely a hernia may become strangulated on its first appearance. Narrowness of the neck of the sac conduces to it. Hence it is more common in femoral than in other varieties, and in irreducible herniæ when the neck is partially occluded by adherent omentum, as in some umbilical herniæ. According to Berger, the risk of an inguinal hernia undergoing accident is 2.16 per cent., of a femoral 9.02 per cent.

Determining Causes.—Among the more common causes may be mentioned straining, such as occurs in lifting, sneezing, coughing, etc., in all of which acts there is an increase in the intra-abdominal pressure. Consequently all conditions predisposing to such strain, as certain occupations, pulmonary disease, constipation, and obstruction to urination, are all predisposing causes. Trauma from ill-applied trusses, inflammation, and bands in the sac all may produce it. Volvulus of the contained loop may occur.

Mechanism.—Several theories have been advanced as to the manner in which the pressure is brought about and exerted upon the contained bowel. Undoubtedly the exact mechanism varies in different cases, and it is a combination of factors rather than any single one that produces the result. Among the various theories advanced, the following are the more important: 1st. Elasticity of the ring (Richter). 2d. Compression of the efferent by the distended afferent end of the loop (Lössen). 3d. Angulation of the distal end at the margin of the orifice (Scarpa). 4th. Fæcal impaction. 5th. Torsion or volvulus of the loop (de Roubaix). Whatever the cause it must first interfere with the return circulation, producing congestion which increases the bulk of the contained gut and thus augments the pressure. The condition thus tends always to increase itself.

Violent peristaltic efforts of the intestine to force its contents onward increase the pressure at the point of constriction. The mesentery becomes swollen with the intestine and at the same time is put upon the stretch by the excessive peristalsis. This stretching of the mesentery may produce interference with the circulation in the mesentery and gut on the abdominal side of the constriction, which accounts for the not infrequent circula-

tory disturbances observed proximal to the site of constriction. The latter may also be due to the nipping of the mesenteric vessels supplying gut which is not in the hernial sac. Furthermore, volvulus of intestine lying in the abdominal cavity may be caused by the anchoring of a loop in the hernial sac. The intestine in the sac then represents only a portion of that involved.

Anatomical Changes.—The strangulated intestine passes through stages which can be conveniently stated as (1st) congestion; (2d) inflammation; (3d) gangrene. The rapidity of sequence of these stages depends upon the degree to which the circulation is interfered with. As the return circulation is the first to be interfered with, the first change is an engorgement of the vessels. The walls of the intestine become oedematous and change in color, becoming dark red or purple, and black or grayish-black as gangrene supervenes.

The peritoneum loses its glistening appearance and becomes dull and granular as its vitality disappears. At the site of constriction, usually the neck of the sac, there is a deep furrow which is as a rule grayish in color. The intestine becomes cold, loses its power of contractility, and is distended with gas. As these changes progress there is an exudate of serum, later becoming bloody, into the sac; and a migration of pathogenic germs, notably the *B. coli communis* and varieties of streptococci and staphylococci, takes place through the walls of the gut. The local peritonitis in the sac may extend to the general cavity and produce a spreading peritonitis, or it may be shut off by adhesions. On the other hand, if the vitality of the bowel within the abdomen be impaired, from the causes already stated or from a progressive thrombosis of the vessels, peritonitis may be primarily instituted within the abdomen itself. If the strangulation be due to inflammation of an irreducible hernia, the sequence of events is slightly changed in that the stage of inflammation precedes that of congestion. When gangrene ensues it may occur locally in small patches at the seat of constriction or at the convexity of the loop, or the whole loop may slough. With the gangrene there is an escape of the contents of the bowel. The skin over the sac at first is not involved, but later the tissues covering the sac become infiltrated and oedematous. Finally, an abscess may form and burst and a spontaneous artificial anus result. This is more likely to be the outcome in cases of partial enterocele (Richter's hernia), when only a portion of the circumference of the gut is nipped, and explains the manner in which spontaneous cures may sometimes occur.

The extent of gangrene varies from a portion of the circumference of the bowel to several feet.

The period elapsing between the onset of strangulation and the occurrence of gangrene varies considerably, from four hours to several days, but is rarely less than twenty-four hours. The presence of omentum in the sac delays the onset of gangrene.

Symptoms.—The symptoms of a strangulated hernia are conveniently divided into the general and the local. The general symptoms are practically those of acute intestinal obstruction in contradistinction to those of an obstructed hernia, which are rather those of chronic intestinal obstruction. They are pain, vomiting, complete constipation, and collapse. To these may be added those of general peritonitis.

Pain is of two varieties: a severe colicky pain felt in the region of the umbilicus, which may be constant but more often remittent, and an aching pain at the hernial site. After the onset of gangrene the pain may disappear.

Vomiting is an important symptom. It is as a rule persistent from the moment of strangulation. The contents of the stomach are first expelled, then those of the upper part of the small intestine, and finally the vomitus becomes stercoraceous. It is not accompanied by marked nausea, and is remarkable on account of the quantities expelled. In later stages it becomes regurgitant and there may be hiccough. It may be absent in Richter's hernia, and it may cease on the advent of gan-

grene. Constipation is complete, no gas or feces being passed. However, the bowel may be emptied below the seat of strangulation, and if the strangulation is high up in the small intestine a considerable quantity of feces may be expelled. Also in Richter's hernia, constipation is not the rule and there may even be diarrhoea. The degree of abdominal distention is likewise dependent upon the site of strangulation. If this is located high up in the small intestine it is relatively slight, while if the large intestine is implicated it is great. It is at first accompanied by excessive peristalsis, but later the paralytic meteorism of peritonitis may be present.

Collapse is usually profound, although it may be delayed for several hours. The pulse becomes weak and thready, the temperature is subnormal, the extremities are cold and moist, and the facies abdominis is marked. When inflammation sets in, the temperature usually rises, but it seldom reaches a high level. The duration of life averages from five to seven days. In the aged the symptoms often are not marked, the course presenting an asthenic type in which some of the classic symptoms may be wholly absent. The symptoms at the site of the hernia are pain, irreducibility, loss of impulse, tenderness, dullness, and the absence of gurgling. The hernial protrusion may be so small as readily to escape notice, and it must be remembered that the absence of a history of hernia does not exclude, inasmuch as a hernia may become strangulated on its first appearance. The attention of the patient is usually attracted to the hernia by the presence of pain. The local pain may be masked, however, by the general abdominal pains.

The chief value of irreducibility as a sign of strangulation is in hernia that have previously been reducible. It may be absolute or relative. Tenseness, dullness, loss of impulse and the absence of gurgling are all due to the same causes, *i.e.*, fixation and swelling of the contents of the sac and the exudation of fluid within it.

In certain forms of strangulated hernia the characteristic symptoms may be masked or modified.

The following classification, slightly modified from that of Eccles,² will serve to indicate the sources of ambiguity.

I. Cases in which the peculiarities are dependent upon the contents of the sac.

1. Strangulation of a portion of the bowel.

(1) Partial enterocele (Richter's hernia).

(2) Strangulation of the vermiform appendix.

(3) Strangulation of Meckel's diverticulum (Litré's hernia).

2. Strangulation within the body of the sac.

(1) By bands, adhesions, apertures in omentum, etc.

(2) By kinking or volvulus.

II. Cases in which the peculiarities are dependent upon the sac.

1. Strangulation within a loculus or pouch of the sac.

In cases of strangulation of a portion of the bowel the general symptoms as a rule are not marked, while the local symptoms may be characteristic. On the other hand, the remainder of the classified cases present the usual general symptoms, while the local symptoms may be markedly modified. The differences in the local symptoms are due to the fact that the entire hernia is not necessarily irreducible, and there may be impulse, tympany, and gurgling at a portion of the protrusion.

In the following groups of cases errors of diagnosis as to the site of strangulation, or as to whether it is present or not, may arise (modified from Eccles²).

I. Cases with multiple hernia.

1. Two (or more) hernia, one irreducible and the other strangulated.

2. Two hernia, one concealing the other.

3. An inguinal and a femoral hernia on the same side, one strangulated and the other not.

II. Cases with irreducible hernia in which the obstruction or strangulation is within the abdomen and not at the site of the hernia.

In cases of multiple hernia the local symptoms as a rule are sufficient to determine the site of strangulation. In infants, inasmuch as they are unable to direct atten-

tion to the local condition and as they are frequently subject to attacks of vomiting and colic, the diagnosis may be delayed. In them continued vomiting should lead to a thorough examination of all hernial sites. The differential diagnosis lies chiefly between strangulated and obstructed or inflamed hernia. Other conditions which may simulate strangulated hernia are inguinal adenitis, acute epididymitis or orchitis, and severe colic, peritonitis, or the uncontrollable vomiting of pregnancy in conjunction with a hernia.

Prognosis.—The prognosis in untreated cases is uniformly bad, with the exception of the exceedingly few cases that end in recovery with the spontaneous formation of an artificial anus. The mortality following taxis and that associated with herniotomy manifestly cannot be compared, inasmuch as the conditions found in many cases successfully operated upon would absolutely contraindicate the employment of taxis, and also because cases in which taxis proves successful would in all probability be successfully treated by operation.

The statistics of Frickhoffer¹ in regard to cases in which taxis has been apparently successful give in 308 cases of femoral hernia a mortality of 14.9 per cent., and in 518 cases of inguinal hernia one of 7.8 per cent. The statistics of herniotomy show a mortality that varies from 10 per cent. to 12 per cent. in cases in which the strangulation has existed twenty-four hours or less, to 50 per cent. in cases in which it has existed over three days. The risk of herniotomy when the gut is still returnable is comparatively slight. In gangrenous hernia the mortality depends largely upon the method of treatment of the intestine (see Treatment).

In a total of 354 cases of gangrenous hernia, collected by Gibson,³ 120 were fatal, a mortality of 34 per cent. Of these 354 cases, 96 were of the inguinal variety, with 25 deaths (mortality: 26 per cent.); 188 were of the femoral variety, with 69 deaths (mortality: 37 per cent.); 22 were of the umbilical and 2 of the ventral variety, with 16 deaths (mortality: 67 per cent.); and 7 belonged to the obturator variety, with 6 deaths (mortality: 86 per cent.). By these statistics it will be seen that the mortality of femoral hernia is greater than that of the inguinal form, and the mortality of umbilical hernia is greater than that of femoral hernia.

Treatment.—The treatment of strangulated hernia is by means either of *taxis* or of *herniotomy*.

Taxis is the reduction of the protrusion by manipulation. It is best performed with the aid of a general anesthetic, and the operator should be prepared to resort at once to herniotomy if his efforts at reduction fail. In inguinal and femoral hernia the parts are relaxed by flexing the thigh on the pelvis and rotating it in an inward direction. Placing the patient in a position of slight inversion, *i.e.*, with the head lower than the pelvis, is permissible.

The neck of the sac is then grasped with the left hand and gentle efforts are made with the right hand to press the contents back. Pressure should be exerted in the direction of the canal; upward, outward, and backward in inguinal and backward and upward in femoral hernia. The efforts should never be prolonged for more than five minutes and roughness is distinctly contraindicated.

Success is determined by the complete disappearance of the hernia, and by the fact that the surgeon can pass his finger through the ring.

Taxis is contraindicated in the following conditions:

1. When it has already failed.
2. When the superficial tissues are inflamed.
3. When there is reason to suppose that the intestine may be gangrenous.
4. When strangulation has existed for twenty-four hours.
5. When the hernia has been previously irreducible.
6. When there is great prostration.

The dangers of *taxis* are:

1. Injury or rupture of the intestine.
2. The return of gangrenous intestine to the abdominal cavity.

3. The transference of septic fluid from the sac into the general peritoneal cavity.

4. "Reduction *en masse*."

Reduction *en masse* is rare, is more frequent in inguinal hernia, and is practically the conversion of the hernia into a form of interstitial hernia; the sac and its contents being forced into a position between the transversalis fascia and the remainder of the abdominal wall, while the constriction still remains at the neck (Fig. 2622). The sac may be ruptured and its contents escape into its new position; or only part of the sac may be reduced.

In modern surgery taxis is resorted to with constantly diminishing frequency, most surgeons now performing herniotomy at once.

Herniotomy and kelotomy are the terms applied to the operation for the relief of strangulated hernia. This operation possesses the advantage over taxis of avoiding its dangers, of permitting inspection of the intestine, and of affording an opportunity for the radical cure of the hernia.

The steps in the operation are:

1. The exposure of the sac.
2. Opening of the sac and relief of the constriction.
3. Treatment of the contents.
4. Closure of the wound.

The sac is exposed by an incision similar to that which would be employed in an operation for radical cure. The incision should be made in such a manner as to expose well the neck of the sac, and it may have to be prolonged in order to afford room for resection of the bowel. The sac is recognized by its dark color and its gliding over the contents. It is opened by pinching it up with forceps so as to form a fold which can be incised without injury to the contents. When an opening has been made, the fluid which escapes will be clear and odorless, although it may be bloody, if the gut is sound; or it will be cloudy, purulent, or feculent, if the vitality of the gut has become impaired. The interior of the sac and the contents are then thoroughly washed with normal salt solution to lessen the chances of contamination and the constriction is sought for and divided.

Inasmuch as the constriction is usually caused by bands of fascia which lie outside the sac, division is best done from without, and not from within, as was formerly taught by the old school. It may be necessary, however, in some femoral hernia to divide it from within, which is done by cutting upward with the herniotome or blunt-pointed bistoury or by use of a director. The constriction having been freely divided the gut must be gently drawn out to expose the efferent and afferent portions, especially the latter, in order to observe their condition, since changes in their nutrition may have occurred for a considerable distance within the abdomen. If the condition of the gut is satisfactory it is returned to the abdomen and the wound is closed as in the operation for radical cure of a hernia of the same variety.

The gut is returned if its peritoneal coat is smooth and glistening, if its color is not black or gray, even though it may be dark red or purple, if the fluid in the sac is odorless and clear, and especially if it has retained the power of contractility in all its parts. It must not be returned if the peritoneum has a granular aspect, if the fluid is cloudy and foul, or if the gut is cold and flabby and has lost its contractility. If there is doubt, the constriction having been freely relieved, the bowel may be surrounded with compresses wet in warm normal salt solution, for several minutes, and its condition again noted. In cases of exceptional doubt it may be thus left for twenty-four hours. Very small areas of gangrene

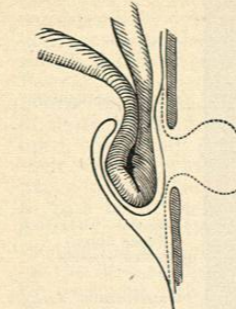


FIG. 2622.

may be excised and the edges of the resulting perforation turned in with Lembert sutures.

If the gut is not in a returnable condition, there are two methods of treatment applicable: 1. Resection and immediate reunion. 2. Formation of an artificial anus. In the performance of the latter the gut is either fixed by a few sutures to the margins of the wound and opened to relieve the obstruction, or it is resected and the ends are sutured to the wound.

In the choice between resection and immediate reunion, and the formation of an artificial anus, the operator must take into consideration the condition of the patient and his own skill as an intestinal surgeon. According to Hofmeister,³ the decision to form an artificial anus is tantamount to the expression that the case is otherwise hopeless.

As the following statistics in regard to gangrenous hernia show, the treatment by primary resection and immediate reunion is the most successful.

Compiler or operator.	ARTIFICIAL ANUS.			PRIMARY RESECTION AND REUNION.		
	Cases.	Deaths.	Mortality, per cent.	Cases.	Deaths.	Mortality, per cent.
Zeidler ⁴ (1892).....	287	213	74.22	289	142	49.13
Mikulicz ⁵	94	72	76.6	68	32	47.1
Hofmeister ⁶ (1900).....	167	101	60.5	214	99	46.0
Petersen (up to 1898)....	17	10	57.0	16	8	50.0
Petersen (since 1898)....	5	5	100.0	12	1	8.3
Petersen ⁷ (total).....	22	15	68.2	28	9	32.14
Gibson ⁸ (1900).....	101	53	52.5	226	58	25.0

Of these series Gibson's are the most comprehensive and argue strongly in favor of resection and reunion. His statistics in regard to the formation of an artificial anus with and without resection of the gangrenous intestine show a very slight advantage in favor of resection. In twenty cases collected by him in which the gangrenous portion was invaginated, three patients died—a mortality of fourteen per cent.

The large mortality following the formation of an artificial anus is due not only to the immediate condition, but also to asthenia if the anus is situated high up in the tract, and to the subsequent measures taken to cure the artificial anus. The mortality of the operation for relief of artificial anus following herniotomy is in the neighborhood of twenty-five per cent.

The chief considerations to be observed in regard to the treatment of gangrenous hernia are: the use of local anesthesia if feasible; the employment of a large wound (hernio-laparotomy), enabling the operator to return the intestine easily and visibly, thus avoiding kinking; the emptying of the intestine so far as possible; wide resection, the amount removed depending largely on the condition of the mucous membrane; open treatment of the wound by tamponade when peritonitis is present, or if the condition of the returned bowel is still suspicious.

For the technique of intestinal resection the reader is referred to special articles on that subject. A fatal outcome in herniotomy is due to one or more of the following causes: 1. Shock. 2. Return of damaged intestine (insufficient resection). 3. Extension of an existing peritonitis, or one due to leakage (faulty suture). 4. Kinking or twisting of the returned loop. 5. Exhaustion (artificial anus).

The after-treatment does not differ from that employed after other operations upon the intestinal tract. For the first few days the bowels are best opened by enemata.

ANATOMICAL VARIETIES OF HERNIA.

INGUINAL HERNIA.—This term includes all hernia into or through the inguinal canal.

Anatomy.—For the detailed anatomy of this region the reader is referred to special treatises. There are, however, certain considerations which are not generally made clear, and which have considerable bearing espe-