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 fœtus 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 acquirements—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 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 parasyphilitic 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 fœtal 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 inheritance 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. *J. George Adami.*

Bibliographical References.

Adami: New York Med. Journ., June 1st, 1901.
Chantemesse and Podwyssotsky: Des Processus généraux, Paris, 1901.
Charrin: Influences des Toxines sur la Déscendance. Comptes rend. de l'Acad. des Sciences, July 29th, 1895.
Charrin and Gley: *Ibid.*, October 4th, 1895, and La Semaine méd., 1895, No. 55.
Darwin: Origin of Species. Variation of Plants and Animals in Domestication.
Driesch: Zeitschr. f. wissensch. Zoologie, liii., 1892; lv., 1893; and Arch. f. Entwickelungsmechanik, 1900, pp. 361 and 411.
Ehrlich: (On the Side-Chain Theory of Immunity) Croonian Lecture; Proceedings Royal Soc., London, 1900.
Fournier, E.: Stigmates Dystrophiques de l'Hérédo-syphilis, Paris, 1898.
Galton, F.: Hereditary Genius, 1869.
Gheorghiu: L'Obstétrique, January, 1900, p. 63.
Legendre: Article on Heredity, Bouchard's Pathologie Générale, vol. i., Paris, 1897.
Mairet and Combemale: Influence Dégénérative de l'Alcool sur la Déscendance. Comptes rend. Acad. de Méd., March 5th, 1888.
Morgan, T. H.: Anat. Anzeiger, x., 1895, p. 19.
Naegeli, C.: Mechanisch-physiolog. Theorie der Abstammung, Munich, 1884.
Nussbaum, M.: Ueber Vererbung, Bonn, 1888.
Paul, Constantin: Arch. gén. de Méd., xv., 1860, p. 513.
Sutton, Bland: Introduction to General Pathology, London.
Vincent: Ann. de l'Inst. Pasteur, xii., 1898, p. 785.
Weismann: On Heredity. Authorized Translation by Poulton Schönland and Shipley, 2 vols., Oxford, 1889–91.
Wilson: The Cell in Development and Inheritance, New York, Macmillan, 1898, and Journ. of Morphology, viii., 1893, p. 579.

* 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.

* 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.

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. (℥ 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 introsusception of the intestine into one of the peritoneal fossæ. 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 fœtal 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 herniæ 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 herniæ: 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 herniæ.

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 fasciæ 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, cæcum, 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.