may look like a boy of ten or twelve. With this there is often an infantile condition of the genitalia and the hair on the pubes and other parts is slow in appearing. The characteristic lesion of syphilis in the placenta is endarteritis, which leads to extensive destruction of the vascular area, with degeneration, atrophy, and fibrosis of the region involved. The placental circulation must in such cases be greatly interfered with. In syphilis, however, other factors enter. We have to admit that the presence of so serious a disease in the parent, usually the father, must have a tendency to lessen the vitality of the germ cell, to say nothing of the presence of the germ of the disease (the existence of which on analogy we can hardly deny) in the fertilized ovum, with all that this implies.

Hitherto we have been discussing what might be called *primary* or *essential* dwarfism (microsomia) in which there predominating element is a deficiency in the vegetative energy of the cells. The affected persons are in fact normal individuals save in the one particular of size and weight. Besides this, however, we have to recognize another class of cases that clearly belong to a different category. I refer to instances of *secondary* or *symptomatic* dwarfism. Here, while the height of the body is below the normal, the weight is not so strikingly diminished as in the true dwarfs. The main feature is, however, that in addition to general hypoplasia, there are evidences of pathological changes in the tissues, particularly in the bones, leading to asymmetry, deformities, fractures, or malformations. The defects are structural as well as nutritional. Just at this point it may be remarked that the exact nature of these cases presents one of the most difficult problems in etiology. In former times many cases of congenital dwarfism of this type were put down to syphilis, rickets, or cretinism. It was soon found, however, that there were some important points of distinction between certain of the cases. The typical features of cretinism were not always present, nor did the disease, if rickets, conform to the picture of the ordinary post-natal affection. These considerations have led investigators to recognize several distinct types, which are by some regarded as separate entities. We have, therefore, to consider in this connection rachitis, cretinism, osteopsathyrosis, chondrodystrophia fœtalis, and osteogenesis imperfecta, affections that agree in this that with more or less stunting of the body there are structural modifications of the bones of an obviously pathological nature. The amount of confusion that has involved the subject is sufficiently indicated by the number of names that have been proposed for the condition—fœtal rachitis, pseudorachitism, cretinoid dysplasia, chondritis fœtalis, micromelia chondromalacica, achondroplasia, chondrodystrophia fœtalis. A decided step in advance has been made through the researches of Kaufmann ("Untersuchungen über die sogen. fœtale Rachitis," Berlin, 1892, u. Ziegler's "Beiträge," 1893, xiii., S. 32–64) who introduced the name chondrodystrophia fœtalis. Here the type of dwarfism is micromelic and the lesions are usually symmetrical. The body is plump, the micromelia rhizomelic, the head large, and the hands often show the so-called "trident" deformity. There are, however, notable differences in the length, curvature, and consistence of the bones of the extremities, and in the configuration of the skull. Kaufmann, consequently, recognizes two groups: one, in which there is a distinct cretinoid appearance of the skull and face, namely, a deeply sunken nose, prominent eyelids and lips, thick cheeks, and large mouth; and another, in which the nose is flattened and retracted as a whole. In the former type the bone is of good consistence although somewhat more vascular than normal, while in the latter the bone is soft. No hard-and-fast line can be drawn between the two forms, and Kaufmann regards them as manifestations of the same process, the differences being due to the chronicity and intensity of the process. The retraction of the root of the nose is usually said to be brought about by premature ossification and synostosis of the os tribasilare. The ethmoid is also somewhat shortened, and in some cases may be the only part affected. Klein has observed well-marked craniotabes in some cases. In some, too, there may be beading at the costochondal articulations, and defective development of the pelvis, glenoid, and cotyloid cavities. Lordosis may also be a marked feature, and Regnault (*Bull. et mém. de la Soc. d'Anat.*, 1901, lxxvi., pp. 559–560) has found the vertebræ involved. The disease begins in fœtal life, running its course usually, as it is believed, from the third to the sixth week. As a consequence the bones affected are the base of the skull, the long bones, the ribs, and the pelvis. The bones formed in membrane, those that in late fœtal life are mainly cartilaginous, usually escape. Kaufmann has described three sub-classes in chondrodystrophia: Chondrodystrophia malacica, in which the bone is soft; C. hypoplastica, in which there are evidences of retarded growth; and the C. hyperplastica in which the epiphyseal ends of the long bones are greatly enlarged. The periosteal ossification is normal, so that the bones become plump and thick, although somewhat irregular, but the growth in length is disturbed owing to faulty ossification at the epiphyseal junctures. Ingrowth of the periosteum at the epiphyseal lines may occur. Microscopically the cartilage cells are deficient in growth, being spindle-shaped and irregularly arranged, while the hyaline matrix is more or less soft and homogeneous. It is not improbable, however, although the majority of children thus affected are stillborn, that when the affection is of mild type or arises late on in fœtal life, existence may be prolonged for some time. Parrot has met with an example in a child seven and a half years old, who measured 94 cm. in length. Certain changes in the bones of the hands, notably in the fingers, seem to prove that the disturbances of ossification may go on at a later period than that above indicated. (See Thompson, *Edin. Med. Journ.*, 1892–93, vol. xxxviii., pp. 1109–1113, and Turner, *Practitioner*, 1899, vol. lxiii., pp. 263–277.) Thompson (*loc. cit.*) has met with two instances at the ages of thirty-six and thirty-nine, and others have been described by Osler (Trans. Amer. Cong. Phy. and Surg., 1897, vol. iv., pp. 190–192), Abt (*Archives of Pediatrics*), Cestan, "Nouvelle Iconographie de la Salpêtrière," 1901, xiv., 277–289), Apert (*Ibid.*, pp. 290–298), and Baldwin (*Medical News*, 1890, vol. lvii., pp. 138–141).

The exact relationship of the disease, called by Vrolik and Stilling (*Virch. Archiv*, 1899, cxv., SS. 357–370) osteogenesis imperfecta, to chondrodystrophia, is still *sub judice*. The studies of Stilling, Hildebrandt (*Virch. Archiv*, 1899, clviii., SS. 426–444), and Harbitz (Ziegler's "Beiträge," 1901, xxx., SS. 605–628) go to show that it is a definite intra-uterine process. There is clearly some defect in the process of ossification, for the bones are soft and brittle, with the result that fractures and deformities are common. Microscopical study has shown that the trabeculæ are few in number, irregular, and imperfectly formed. There is no continuous system of trabeculæ with Haversian canals and lamellæ as in normal bone. A point of great interest is the extraordinary manner of ossification of the skull. The calvarium is formed not of continuous bony plates, but of a multitude of small mosaics, sometimes touching one another, but also attached by bridges of periosteum and dura. In a remarkable case described by Stilling, the cranial covering consisted of a membranous sac which contained scattered bony spicules. So far as is known there is no synostosis of the basal synchondrosis in this disease. While the affection has hitherto been found only in the new-born or in very young infants, it is not necessarily fatal, and Harbitz is inclined to think that some cases of dwarfism that have been regarded as examples of fœtal rickets or chondrodystrophia may have been osteogenesis imperfecta. As in chondrodystrophia porosity of the bones occasionally appears to depend on hereditary conditions, and Bircher (Lubarsch u. Ostertag, "Allg. Aetiologie," Wiesbaden, 1896, S. 53) has found osteoporosis in the case of a chondrodystrophic dwarf (*cf.* also the case of Ekmann, referred to above). We are, therefore, not prepared to

believe with Paltauf ("Ueber den Zwergwuchs," Wien, 1891) that the two affections are not related.

To rickets chondrodystrophia bears much resemblance, although there are several differences. In both there is enlargement of the epiphyseal ends of the long bones (chondrodystrophia hyperplastica), the long bones may be curved, the ribs beaded, and there may be spinal and pelvic deformities. In rickets, however, the arms are usually long. Periosteal bone formation is also interfered with. The periosteum is readily stripped off and the underlying bone is softer and more spongy than normal. As the condition heals the bone becomes unusually plump and dense. Basilar synostosis does not occur in rickets. Microscopic study has shown that the epiphyseal zone of proliferation in rickets is thicker than normal, irregular in outline, soft, and very hyperæmic, so much so that Kassowitz believed the lesions to be of inflammatory origin.

The microscopical appearances of the growing ends of the bones in chondrodystrophia are, however, quite different from those in post-natal rickets. No clear evidence of the existence of rickets originating during intra-uterine life is forthcoming, yet there is a parallel between chondrodystrophia and rickets, for, as is well known, post-natal rickets, if severe, results in the stunting of the growth of the affected individual, and cases of extreme dwarfism have been met with. The lesions are characteristic. The skull is large, although the face is relatively small. The fontanels remain open for a prolonged period, and bone in certain regions, especially the occipito-parietal, may be so thin as to give way under the pressure of the finger (craniotabes) The forehead is usually large and square, owing to the formation of flat hyperostoses over the frontal eminences. The sternum projects and the sides of the chest are drawn in (pectus carinatum). The spine is often curved and the extremities become greatly deformed owing to the weight of the body and muscular action. The pelvis is contracted; dentition is delayed and the teeth are small and badly formed. The condition is by many believed to be due to infection or possibly auto-intoxication. This view is supported by the experiments of Morpurgo (*Centralbl. f. Path.*, 1902, xiii., S. 113), who showed that rickety changes in the skeletons of young white rats could be produced by the injection of cultures of a diplococcus. The lesions in rickets, however, are so unlike those of chondrodystrophia in important particulars that it is unlikely that an infective cause is at work in the latter disease. Differentiation between chondrodystrophia and rickets may be made by attention to the following points: The dwarfism in the former is micromelic; in the latter not. Periosteal bone formation is not interfered with in chondrodystrophia, while it is in rickets. In healed rickets the bone is abnormally dense; not so in chondrodystrophia. Basilar synostosis does not occur in rickets. In rickets you do not get "trident deformity" of the hands. Developmental anomalies, like cleft palate and polydactylism, are not found in rickets. Osteogenesis imperfecta is recognized by the fragility of the bones, the peculiar ossification of the cranium, and the lack of basilar synostosis.

It is a suggestive and a striking fact that while we are able to draw these distinctions between the type cases of chondrodystrophia, osteogenesis imperfecta, and rickets, there exist borderland cases in which some of the features of these diseases may be combined. Such a one is that reported by Hektoen ("Anatomical Study of a Short-limbed Dwarf," *Amer. Jour. Med. Scien.*, May, 1903), in which in a micromelic dwarf, forty-five years of age, there were one hundred and seventy-two Wormian bones in the skull, without premature synostosis of the os tribasilare, with shortness and curvatures of the long bones, enlargement of the articular ends, curvatures of the spine, deformity of the pelvis, osteoporosis and multiple fractures, and a fibroid thyroid. A case presenting features both of chondrodystrophia and osteogenesis imperfecta has been described also by the Countess von Geldern-Egmond ("Beitr. zur Casuistik der sogenn. fötalen Rachitis," Inaug.-Diss., Zurich, 1897)

The etiology of these interesting conditions is still largely unknown. Heredity appears to be a factor in some cases. In one case, recorded by Porak ("Beitrag zur Histologie und Funktion der Schilddrüse," Inaug.-Diss., Königsberg, 1892) a dwarf gave birth to one healthy child and a second with all the signs of the so-called "fœtal rickets." Guéniot (*Bull. et mém. de la Soc. Obstet. et gynécol. de Paris*, January, 1893) recounts the operation of Cæsarean section on an achondroplastic dwarf, the child being similarly affected. The parents of chondrodystrophic dwarfs as well as their children are nevertheless often healthy. Heredity seems then to play a minor rôle. The influence of heredity in the case of polydactyly has already been dwelt upon, and it is a curious fact that polydactyly may sometimes be associated with dwarfism. An instance of this will be found in *Hutchinson's Archives of Surgery* for April, 1893, where there is given an illustration of a micromelic polydactylous dwarf, copied from Theodore Kerckring's "Spicilegium Anatomicum," published in 1670. The drawing is not entirely to be relied upon, but it is clear that the limbs were too short for the trunk. Both the hands have seven digits; the right foot has eight and the left nine. The condition is not perfectly symmetrical, for in some cases two of the metacarpal or metatarsal bones are welded together. The long bones are plump, and, so far as it is safe to judge from the imperfect sketch, the ends appear to have been enlarged. The orbits are deformed, the two halves of the lower jaw are already united, and the ribs are short and badly formed. It is clear that here we have an abnormality of development in few respects comparable to rickets. It may be that here, as in simple polydactylism, we are dealing with a primary vitium of development inherent in the germinal cell. This view is supported also by the observation of Kirchberg and Marchand (Ziegler's "Beitr. zur path. Anatomie," Bd. v., 1889), who found cleft palate in a chondrodystrophic infant. One of Bowlby's cases, also, reported as congenital cretinism but really an instance of chondrodystrophia, had a cleft palate and a deformed nose. The right foot had six metatarsal bones but seven toes; the left foot had seven toes.

There is, however, another possible explanation. Recent observations have shown that many forms of hypoplasia, non-closure of sutures, and, in fact, the most extreme deformities may result from intra-uterine pressure. The simple weight of a limb resting on a band has been sufficient to erode the soft tissues to the bone. Twisting of the cord about a limb may lead to dwarfing or amputation. The pressure of bands traversing the amniotic sac is also a well-recognized cause of even more extreme deformities, such as anencephaly and craniorachischisis. Dareste believed that a contracted amnion was an important cause of hypoplasia.

In Klebs' text-book of "General Pathology" (vol. ii., 1889, Jena) is an interesting illustration representing a microscopical section through the proliferating end of one of the long bones in a micromelic dwarf. The growing cartilage cells are closely packed together, flattened, with their long axes at right angles to the long axis of the bone. The appearances could not be due to alterations in the ground substance which seemed to be normal, but could be attributed only to the effects of external pressure. This would, of course, lead to inhibited and imperfect ossification. The effects of intra-uterine compression are well illustrated by the experiments of Fol and Warynski (Thèse de Genève), who brought external pressure to bear upon a growing embryo. They found that the head was the part most amenable to pressure, and could to some extent be restrained in its growth. Warynski, also, by exerting pressure at the site of fusion of the two primitive cardiac rudiments, was able to produce a double-hearted monster. Klebs (*loc. cit.*, p. 306) refers to a most remarkable case in which a contracted sac led to a singular malformation. In an ectopic gestation the fœtus was found in a tight sac within the transverse mesocolon. There was syndactylism in the upper extremities and polydactylism in the lower. From the

recorded cases it would appear that contraction of the amnion does not lead to dwarfing of the fœtus as a whole, but that certain parts are more liable to be affected, as the extremities and head. In this connection also may be remembered the fact that hydramnios is apt to be associated with grave deformities, such as exencephaly and spina bifida, and has been found in a case of osteogenesis imperfecta. The effect of intra-uterine pressure has also been emphasized by von Franqué ("Ueber sogenan. fœtale Rachitis," Sitzungsberichte der physicalisch-mediz. Gesellsch., Wurzburg, Jahrg. 1893, S. 80).

It is on the ground of the obvious errors in development which are sometimes associated with chondrodystrophia, and which can only be referred to peculiarities of the Anlage, that Virchow objects to the term chondrodystrophia introduced by Kaufmann. Virchow points out that the condition imperceptibly shades into a pronounced developmental anomaly (*Missbildung*), which is finally represented by the phokomelia of Saint Hilaire.

The small stature, the peculiar facial configuration, and the enfeeblement of mental power, with other minor abnormalities found in some cases of chondrodystrophia, have induced the suspicion that some of these cases of dwarfism are really cases of cretinism. The difficulties in the way of differential diagnosis are great. Thus Dolega, Bernard, and Bircher have pronounced certain cases to be instances of cretinism, and yet a subsequent examination proved them to be Kaufmann's chondrodystrophia, and conversely, Neumann, Scholz, and others have described instances of cretinism as fœtal rickets. The difficulties in the way will be readily comprehended if one considers for a moment the lesions in a typical case of cretinism. In the cretin the stature is almost always stunted, Maffei in twenty-two cases out of twenty-five finding the height to be less than 140 cm., while several were under 95 cm. The limbs and trunk are disproportionate. The head is usually relatively large, the top flat, and the occiput prominent, although microcephaly has been found. The fontanels and sutures remain open for a long time. The root of the nose is retracted, and the organ is short and thick with large wide nostrils. The lips and tongue are enlarged. The teeth appear late and are large. The first dentition usually persists throughout life. There is as a rule no hair on the pubes and in the axillæ. The sexual organs are poorly developed, and puberty, if it occur at all, is late. With regard to the finer structure of the bones in cretins, Langhans (*Virch. Archiv*, 1892, cxxviii.) found that the cartilage cells were small, spindle-shaped, and anomalously arranged, being longitudinal to the axis of the columns. The rows were also often interrupted. The bony trabeculæ were shortened and the marrow spaces in the youngest portions of the bone were large and widely separated. The resemblance to Class I. of Kaufmann's chondrodystrophia is close. Typical cretinism is endemic and associated with goitre. Chondrodystrophic dwarfism is sporadic. Can, then, chondrodystrophia be sporadic cretinism? The recorded cases of chondrodystrophia go to show that the disease is of intra-uterine origin, and so far no evidence is forthcoming to prove that the affection ever arises subsequently to birth. Cretinism is said to be congenital in some cases, but the symptoms usually appear some five to eight months after birth, or even later. It is now believed that the changes in the skeleton of cretins are due to delayed ossification of the cartilages. Hofmeister (*Fortschritte auf dem Gebiete der Roentgen-Strahlen*, Bd. i., Hft. i., 1897), studying a case of cretinism with the *x*-rays, found that the epiphyseal ends of the bones grew slowly, and that the epiphyseal plates persisted for a long time. The bones were small. Ossification may in time be completed, but the process may take years; in fact, cretins may continue to grow until they are thirty or forty years of age. Periosteal osteogenesis is normal, or may be in excess. The peculiar appearance of the nose is due to premature synostosis of the bones at the base of the skull, although, as Niepce, Stahl, and Klebs have shown, this anomaly of bone formation does not always occur.

The stunted growth is believed by Bircher (*loc. cit.*) to be due to defective development of the cartilage cells, as was found occasionally by Virchow and Klebs at the base of the skull. We find, therefore, that cretinism and the so-called chondrodystrophia have much in common. The resemblance can, however, be made out clearly only in the case of chondrodystrophia of Kaufmann's first group. Cretinoid chondrodystrophia might be explained as cretinism that had become marked at a very early period of intra-uterine life.

Further information might be obtained by an inquiry into the condition of the thyroid gland in chondrodystrophia. Hofmeister ("Experimentelle Untersuchungen über die Folgen des Schilddrüsenverlustes," *Beiträge zur klin. Chirurgie*, 1894) holds that the changes in the bones produced by thyroidectomizing rabbits are identical with those found in chondrodystrophia as described by Kaufmann, H. Müller, Kirchberg, and Marchand. Although we must admit the great similarity of the lesions in the two cases, this, of course, does not prove identity. Leblanc (Comptes rend. de Soc. de Biol., 1902, liv., 88–89) states that chondrodystrophia is often associated with myxœdema and that disturbance of the thyroid is the causa morbi. Nasan (*Rév. de Neurologie*, 1901, p. 549) is of the same opinion. There is no need of entering here into the question of the identity of myxœdema and

Case.	Observer.	Type of Disease.	Thyroid.	Reference.
I..	Bowlby	Cretinoid with some signs of myxœdema.	Absent	Trans. Path. Soc., 1884, p. 450.
II..	Kirchberg and Marchand.	Not cretinoid.....	Not enlarged	Ziegler's "Beiträge zur path. Anat.," Bd. 5, 1889.
III..	Dolega	Cretinoid	A few atrophied remains without colloid found.	Ziegler's "Beitr. zur path. Anat.," Bd. 9, 1891.
IV..	Paltauf.....	.....	Very small	"Ueber den Zwergwuchs," Wien, 1891.
V..	His (quoted by Paltauf).	.....	Apparently normal.	
VI..	Bernard.....	Cretinoid.....	Thyroid absent, but a very small accessory found.	"Die Cretine Pohl," Inaug.-Diss. Würzburg, 1892.
VII.–XIX.	Kaufmann	Eight cretinoid	Small in two; without change or at least not enlarged in five.	"Untersuchungen über die sogenannte foetale Rachitis," Berlin, 1892.
XX..	Kaufmann	Not cretinoid; C. hyperplastica.....	Normal	Ziegler's "Beitr.," 1893, xiii., S. 32.
XXI..	Scholz	C. hypoplastica.....	Vascular struma	"Ueber fötale Rachitis," Inaug.-Diss. Göttingen, 1892.
XXII..	Johannessen ..	C. hyperplastica, not cretinoid.....	Normal (microscopical examination).	Ziegler's "Beitr.," Bd. 23, 1895.
XXIII..	Salvetti	C. hypoplastica; cretinoid with some signs of myxœdema.	Hypertrophied	Ziegler's "Beitr.," Bd. 16, 1894, S. 29.
XXIV..	Grotthoff	C. malacica.....	Normal size.....	"Ueber einen Fall von sogen. fötaler Rachitis," Inaug.-Diss., Berlin, 1895.
XXV..	Bircher	.....	Normal size	Lubarsch u. Ostertag's "Allg. Aetiologie," 1896, S. 51.
XXVI.–XXVIII.	Legny and Regnault.	.....	Normal in all.....	Comptes rend. de Soc. de Biol., 1902, liv., 567.
XXIX..	Hektoen.....	Mixed type.....	Atrophy and fibrosis; hypophysis enlarged; iodine not determinable in thyroid.	*Amer. Journ. Med. Sciences*, May, 1903.

cretinism, further than to say that it is proved practically beyond doubt that the two are one and the same thing, and due to defect in the function of the thyroid. Any differences are to be explained in respect to the time at which the athyroidea first makes itself manifest. Myxœdema is cretinism manifesting itself in the adult; cretinism is myxœdema of childhood. In opposition to the views of Leblanc and Nasan may be cited the experience of Cestan ("Nouvelle Iconographie de la Salpêtrière," 1901, xiv. 277), who found a condition of chondrodystrophy in a girl nine and a half years old, unimproved by thyroid feeding for nine months, and of Marie (*Presse médicale*, Juillet, 1900, vol. iv.), who in two cases found no benefit from the use of thyroid extract. We should observe, too, that the lesions of chondrodystrophia are said to develop in the earliest period of fœtal life before the thyroid is properly formed.

With regard to the anatomical peculiarities of the thyroid in chondrodystrophia it is not always possible to obtain accurate information. So far as I have been able to trace them they will be found in the preceding table.

Unfortunately, not having access to the original papers in all instances, I am not able to give complete information in the above table, and the number of cases might be added to. We see, however, that out of twenty-nine cases the thyroid is abnormal in eight, either absent, atrophied, or hypertrophied. And it is further striking that in all the cases in which the thyroid is obviously diseased, with one exception, the clinical type has been cretinoid; with, in two cases, some additional features resembling myxœdema. The one exception, Hektoen's case, is probably to be explained on the ground of a compensatory action of the hypophysis, which was considerably enlarged, but otherwise of normal structure. In Bowlby's case, which is recorded as a case of cretinism, being observed before the distinctions between the different forms of abnormal osteogenesis were so closely drawn as they are at present, the skin was œdematous and semitranslucent, the nose was broad and flat, and there were no eyelashes or eyebrows. In Salvetti's case the skin was also œdematous. In the one case in which vascular struma is stated to have been present (Scholz), there were apparently no signs of cretinism; but this anatomical condition of the thyroid does not of course imply deficiency of the secretion; rather is it akin to what is found in exophthalmic goitre or hyperthyroidism. This association of defective thyroid secretion, which must be admitted where the thyroid structure is so extremely altered, with the cretinoid type of Kaufmann's chondrodystrophia can hardly be fortuitous, especially when we note that in the cases that are distinctly without cretinoid features, the thyroid has been, so far as we can judge from the imperfect information that we are given, practically normal. In settling this latter point we can admit as conclusive evidence only cases that have been controlled by post-mortem examination. Still, the position is supported to some extent by clinical cases. It is, it must be remarked, difficult to draw conclusions as to the functional efficacy of the thyroid from an estimate of its size alone derived from palpation. Osler (Trans. Congr. Amer. Phys. and Surg., 1897, vol. iv., pp. 190–192) describes two cases of chondrodystrophia in French-Canadian children, who did not present the cretinoid facies, in whom the thyroid was not enlarged on palpation. Joachimsthal (*Deut. med. Woch.*, 1899, S. 288) also reports a case of chondrodystrophia hyperplastica, without the cretinoid appearance, in which also the thyroid was normal on palpation. As corroborative evidence, also, it may be noted that Virchow (*Virch. Archiv*, 1883, xciv., S. 183), in the canton of Glarus, Switzerland, where goitrous cretinism is endemic, found not a few cases of chondrodystrophic dwarfism without the gross signs of cretinism. It would seem, so far as we can judge from so limited a number of observations, that the cretinoid type of chondrodystrophia, as described, is simply a form of cretinism, or at least due to thyroid inadequacy. If this be the case, it then follows that Kaufmann's chondrodystrophia fœtalis is probably not a distinct disease entity, but that several differing conditions are included under the one name. Some stress has been laid on the peculiar retraction of the root of the nose, which is so evident a feature in typical cretinism, as an aid in the diagnosis of these conditions. It should be said that this feature is not always present in undoubted cretinism and, moreover, the explanation given of it, namely, that it is due to premature synostosis of the os tribasilare, does not invariably hold good. Some of Kaufmann's cases show that retraction of the root of the nose may be present without premature ossification of the os tribasilare. In these exceptions he attributes the appearance to shortening of the bony parts in front of the os. Conversely, the synostosis may take place without producing any effect on the condition of the nose. Consequently too much weight should not be laid on this point.

It would, perhaps, be also premature to conclude that the other forms of chondrodystrophia may not be dependent on thyroid dystrophy. It is true that Cestan, Marie, and Christopher have failed to get benefit from thyroid feeding in chondrodystrophia. As the disease, however, originates during intra-uterine life, by the time the cases come to observation the main damage has been done, and it is not to be expected that any form of medication would have a marked effect on bony structures once they were formed. The only possible chance would be if the cases could be treated from birth; but, so far as I am aware, this has not been done. This fact weakens the therapeutic argument very much. Further, it does not follow that because the thyroid is of normal size and appearance that it is competent to perform its function. While an absolute deficiency in the amount of secretion furnished by the thyroid will undoubtedly produce the symptoms that constitute cretinism or myxœdema, it is quite possible that a relative inadequacy will produce the same symptoms. This aspect of the case, which has been emphasized by Prof. J. G. Adami ("Internal Secretions Considered in their Physiological, Pathological, and Clinical Aspects," Trans. Congr. Amer. Phys. and Surg., vol. iv., 1897) has been constantly overlooked by writers on the subject. The doctrine of internal secretions supposes the existence of some substance, the product of metabolism, for the secretion to act upon. The thyroid, therefore, may be normal, but if the substance upon which it acts is produced in excess, then the gland will be insufficient for its work, and the various symptoms of athyroidea may make their appearance. In some few instances in which the hypophysis has been invaded by a tumor, symptoms of myxœdema have occurred with an intact thyroid, the thyroid and the hypophysis being, as is now well recognized, more or less complementary organs. Relative inadequacy of the thyroid—and, *mutatis mutandis*, the same remark applies to the hypophysis and other glands furnishing an internal secretion—is a factor that can by no means be neglected in these obscure disorders of development. Until, however, we know much more about the nature, chemical and otherwise, of metabolic processes, we must leave such questions to the realm of speculation.

In concluding, we should not forget to say a word or two in regard to the relationship between anomalies in structure and function of the genital tract and the general question of somatic development. It is a matter of common knowledge that the changes in the genital organs which occur at puberty are coincident with an increased nutritional and functional activity of the body as a whole, as is evidenced by the rapid growth, the consolidation of the figure, the production of hair, the deposit of fat, the changes in the voice, and the altered mental characteristics, all of which together constitute the adult type. Certain isolated facts bring this relationship into still greater prominence. Genital hypoplasia, with its associated sexual torpidity, amenorrhœa, and even sterility, has been found in certain dwarfs, in myxœdema and cretinism, in acromegaly, and in some forms of gigantism. This would suggest that there is some

functional relationship between the thyroid gland, the pituitary body, and the genital organs. The almost constant genital insufficiency found in conditions of athyroidea is so well recognized that I need not do more than mention it. That a similar peculiarity may be associated with lesions of the pituitary body is perhaps not so widely known. Some years ago I performed an autopsy on a female about thirty years of age, who died with symptoms of a basal tumor of the brain. The genitalia were markedly infantile in size and appearance. A tumor of the hypophysis was found, although there were no signs of acromegaly. A similar case is one reported by Babinski (Society of Neurology, June 7th, 1900), who found signs of infantilism, viz., lack of body hair, amenorrhœa, an excess of fat, in a girl of seventeen, but without acromegaly, who post mortem exhibited a tumor of the pituitary. Analogous peculiarities have been found in the male in such cases. While it is true that children the subjects of atrophy of the genitals are often small, instances of the reverse are not unknown. Eunuchs are often of more than normal height. The increase in length is most manifest in the lower limbs, a peculiarity that is to be observed also in the capon, or castrated chicken, and in the ox. The relationships suggested by the above observations might be depicted graphically in the diagram (Fig. 5124).

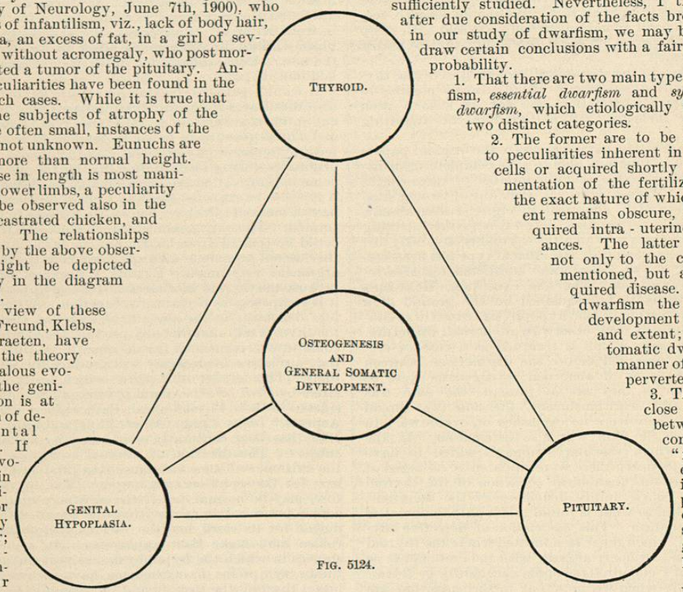

FIG. 5124.

It is in view of these facts that Freund, Klebs, and Verstraeten, have advanced the theory that anomalous evolution of the genital function is at the bottom of developmental dystrophy. If genital evolution be in excess, gigantism or acromegaly will occur; if defective, infantilism or dwarfism results. It seems to me, however, that this is much too strong an assertion, and one that is not supported by the facts, as we know them. Such a theory supposes the existence of an internal secretion in the case of the genital organs, to wit, the testes and ovaries, the evidence for which is not beyond question. Further, the association between genital anomalies and developmental peculiarities is not necessarily direct, as cause and effect, but may be indirect through some third factor. We have experimental evidence to show that defect of the thyroid will produce hypoplasia of the genitalia, but not the converse, and in this case as a matter of fact genital hypoplasia is simply part and parcel of a systemic developmental defect. In the case of the pituitary evidence is scanty, inasmuch as experimentation, except in the single particular of pituitary feeding, is extremely difficult. The evidence, so far as I can gather it from autopsies, seems to favor the view that certain lesions of the pituitary, provided they arise early enough, may prevent the proper development of the genital organs. There is absolutely no evidence to prove that hypoplasia or any other pathological condition of the genitalia has produced disease of the pituitary. Could it do so, one would suppose that, considering the very respectable number of cases of aplasia and hypoplasia of the genital organs that have been described, some abnormality of the pituitary body would occasionally have been noted. This is, however, not the case. It would seem more probable that genital hypoplasia, if not indeed always an effect, is an associated condition merely. In regard to the factors at work in this very interesting but obscure condition of anomalous development, the sum and substance of the whole matter is that our views cannot attain finality until we know much more of the chemistry of the internal secretions and of metabolic processes generally. Hitherto the anatomical peculiarities and the pathological chemistry of the thyroid and pituitary glands in cases of dwarfism and giantism have not been sufficiently studied. Nevertheless, I think that after due consideration of the facts brought out in our study of dwarfism, we may be able to draw certain conclusions with a fair degree of probability.

1. That there are two main types of dwarfism, *essential dwarfism* and *symptomatic dwarfism*, which etiologically belong to two distinct categories.

2. The former are to be attributed to peculiarities inherent in the germ cells or acquired shortly after segmentation of the fertilized ovum, the exact nature of which at present remains obscure, or to acquired intra-uterine disturbances. The latter are due, not only to the causes just mentioned, but also to acquired disease. In true dwarfism the defect of development is in time and extent; in symptomatic dwarfism the manner of growth is perverted as well.

3. There is a close association between such conditions as "fœtal rickets," cretinism, osteopsathyrosis, osteoporosis, osteogenesis imperfecta.

4. Fœtal rickets so called has nothing to do with rickets as it is ordinarily understood.

5. The term *chondrodystrophia fœtalis* is a misnomer, inasmuch as it implies a local lesion, and, moreover, attributes it to a nutritive cause, whereas not only the cartilage but the bone and soft tissues are involved. Virchow is probably correct in regarding it rather as a developmental anomaly.

6. The three types, chondrodystrophia hyperplastica, chondrodystrophia hypoplastica, and chondrodystrophia malacica, are probably not varieties of the same disease.

7. Chondrodystrophia of Kaufmann's first or cretinoid type is probably a modified cretinism.

Albert George Nicholls.

EMBALMING.—This article is intended to furnish useful information to the medical practitioner who may be called upon to preserve a dead body for a limited period of time. This duty can occur only when it is not possible to secure the services of the professional embalmer.

When the object in view is simply to keep the dead human body from undergoing decomposition for a few days, there are a number of methods from which the physician may make a choice. Thus, for example, when it is possible to obtain ice in plenty and the services of a person who can be trusted to renew the supply as frequently as may seem desirable, this method of preventing or at least postponing decomposition is probably as good as any other that can be suggested. But it is often impossible to obtain ice in the desired quantity, or the period of time during which the dead body must be preserved is longer than is compatible with the employment of this method. Under these circumstances it becomes necessary to fall back upon some other procedure, one, for example, which depends for its efficacy upon the employment of chemical reagents. Among the large number of such procedures the physician may be compelled, by reason of the circumstances in which he happens to be placed, to select one of the simpler and less efficacious methods. I will therefore mention a few of these first, reserving to the last an account of the steps which one must take if it be desired to preserve the dead body in good condition for a reasonably long period of time—for months or even years. It is only to such a procedure that the term embalming may with propriety be applied.

Methods Suited to the Preservation of a Dead Body for a Short Period of Time.—(*a*) Place the body, without opening it, and as soon as possible after death, in sawdust mixed with powdered zinc sulphate. The skin, it must be remembered, has a tendency, under continued exposure to this reagent, to assume a yellow color.

(*b*) Open and clean the body and viscera, and keep all the parts that can be reached saturated with a corrosive sublimate solution (1 in 1,000) or a formalin mixture (of a strength of from five to ten per cent.). Cloths saturated with the same fluid should be wrapped around the body, and enough of the preservative should be added from time to time to keep the cloths constantly moist.

(*c*) Open a femoral artery at the apex of Scarpa's triangle and inject a solution of arsenous acid (eight drachms in 9 kgm. of alcohol; cinnabar, q. s.). Then open the abdominal cavity, wash out the cavities of the stomach, bladder, and intestines, and inject the preservative solution into them freely.

(*d*) Camphor dissolved in alcohol, in the proportion of 1 part of the former to 6 of the latter, can be used effectively as a preservative fluid. Another mixture is the following: Oil of turpentine, 5 pints; Venice turpentine, 1 pint; oil of lavender, 2 ounces; oil of rosemary, 2 ounces; vermilion, q. s.

Another mixture, which has been used with some measure of success in England, is the following:—To one pint and a half of glycerin add three quarters of a pound of white arsenic (arsenous acid) and boil; afterward add one gallon of pure glycerin. Still another useful mixture is one composed of alcohol and glycerin in equal parts (each representing forty-seven and a half per cent. of the entire bulk), and carbolic acid to the extent of five per cent. A small quantity of arsenic (about one per cent.) may, if desired, be added to the mixture.

All of the ingredients mentioned in the preceding paragraphs are, as a rule, easily obtainable, and the manipulations required call for no special skill. At the same time it must be remembered that these procedures can at best retard decomposition for only a relatively short time. Thoroughly satisfactory results can be obtained only through the more elaborate and painstaking methods adopted by the professional embalmer.

An Effective Method of Embalming.—Mr. Frank E. Campbell, of 241 West Twenty-third Street, New York City, a recognized authority in embalming, has very kindly allowed the use of the following description of the method of embalming which he prefers:

"I. Bathe and dress (in underclothing) the body to be embalmed, and place it upon a board supported in an inclined position. Close the mouth and eyes, and securely plug all orifices.

"II. Place within easy reach all the instruments and various objects that will be needed, viz., scalpel, aneurism needle, blunt hook, scissors, arterial tube, five six-inch lengths of linen thread for tying about the vein and artery when raised, a threaded needle suitable for sewing up incisions, a bulb syringe, a large bottle filled with the embalming fluid, a sponge, a supply of cotton, etc. Sink the suction end of the tubing of the bulb syringe into the embalming fluid contained in the bottle and squeeze the bulb several times in order both to fill it with the fluid and to force out the air which it contains. When these objects have been accomplished the discharge end of the syringe should also be introduced into the bottle, in order that no leaking may take place.

"III. Connect the aspirating pump, by means of tubing and a gooseneck, with an empty aspirating bottle suitable for holding the blood to be withdrawn from the body. Then attach to the free end of the tubing a long silk-covered vein tube, or, better still, a flexible wire catheter.

"IV. Expose the femoral vessels in Scarpa's triangle over an area two or three inches in length and dissect them clean. Then, with one of the lengths of thread already prepared, tie the artery at the lowest possible point with a firm knot, for the injection is to be made only in an upward direction.

"V. Next, pick up the vein and treat it in the same manner as the artery, remembering, however, that the structure of the vein is fragile and that it may easily be punctured or torn. Make a slit, about a quarter of an inch long, lengthwise in the vein. As blood will escape from the vessel when it is thus opened, it is well to prevent this by making pressure upon the vein with the finger or with a blunt hook. Insert the vein tube into the opening in the vein and push it upward to or near the right auricle of the heart. Pass a piece of the thread snugly, but not too tightly, around both vein and tube, and tie it with a simple knot. Attach the tubing of the aspirating pump and begin to pump the air from the bottle. When the air has been withdrawn from the bottle, the vacuum will draw the blood from the body by way of the vein tube. If no blood flows into the bottle after the air is pumped out, it may be assumed that a clot of blood has probably entered the free end of the tube in the vein, or else that there was no blood at the point where the end of the tube rested. Draw the tube slowly downward, and, if there is still no flow of blood, push the tube as far back into the vein as it will go, detach the rubber tubing from it, and, by means of the bulb syringe, inject a bulbful of aqua ammoniæ, for the purpose of dissolving the clotted blood. After this has been done, resume the operation of aspirating. When all the blood that can possibly be removed in this manner has been aspirated, withdraw the tube for about one-third its length, and let it remain in the vein. (Always keep a vacuum in the bottle.)

"VI. Next, pick up the artery, slit it, and insert the arterial tube with care. Then pass the remaining piece of thread around it as far up as you can, and tie it with a simple knot. Attach the bulb syringe (previously made ready) to the tube and begin the injection slowly. When about one-third of a quart of the preservative fluid has been injected, stop and begin once more to aspirate the vein, for by this time the fluid injected into the artery is forcing the blood ahead of it, and the blood will flow again more freely.

"VII. Administer the injections and the aspirations in alternation until a quantity of the preservative fluid varying from three pints to five or six pints, according to the size of the body, shall have been injected, and also until a clear fluid, and not a bloody one, escapes from the vein tube as a result of the aspiration. Any discolorations of the face, ears, or neck, will probably by this time have disappeared. If it should be found, however, that a slight discoloration still remains in the lower part of the ears, or in the neck, a slight rubbing will in most cases remove it.

"VIII. After it is seen that only clear fluid flows into the blood bottle, draw out the vein tube and tie the vein tightly.

"IX. Continue the injection of fluid into the artery until the veins of the temple, the large vein on the forehead, and the jugular vein, as well as the veins in the arms and hands, indicate that they are filled. Then re-