the terms "blue disease" and "morbus cæruleus" are practically synonyms for congenital heart-disease. The lividity in a majority of cases appears early, within the first week of life, and may be general or confined to the lips, nose, and ears, and to the fingers and toes. In some instances there is in addition a general dusky suffusion, and in the most extreme grades the skin is almost purple. It may vary a good deal and may only be intense on exertion. The external temperature is low. Dyspnœa on exertion and cough are common symptoms. The children rarely thrive and often display a lethargy of both mind and body. The fingers and toes are clubbed in a grade rarely met with in any other affection. The cause of the cyanosis has been much discussed. Morgagni referred it to the general congestion of the venous system due to obstruction, and this view was supported in a paper, one of the ablest that has been written on the subject, by Moreton Stillé. Morrison's recent analysis of 75 cases of congenital heart-disease shows that closure of the pulmonary orifice and patency of the foramen ovale and the ventricular septum are the lesions most frequently associated with cyanosis, and he concludes that the deficient aëration of the blood owing to diminished lung function is the most important factor. Another view, advocated by William Hunter, was that the discoloration was due to the admixture in the heart of venous and arterial blood; but lesions may exist which permit of very free mixture without producing cyanosis.

Diagnosis.—In the case of children, cyanosis, with or without enlargement of the heart, and the existence of a murmur are sufficient, as a rule, to determine the presence of a congenital heart-lesion. The cyanosis gives us no clew to the precise nature of the trouble, as it is a symptom common to many lesions and it may be absent in certain conditions. The murmur is usually systolic in character. It is, however, not always present, and there are instances on record of complicated congenital lesions in which the examination showed normal heart-sounds. In two or three instances fœtal endocarditis has been diagnosed *in gravida* by the presence of a rough systolic murmur, and the condition has been corroborated subsequent to the birth of the child. Hypertrophy is present in a majority of the cases of congenital defect. It is impossible in the scope of a work of this sort to enter upon elaborate details in differential diagnosis between the various congenital heart-lesions. I here abstract the conclusions on this question given by Hochsinger in his recent monograph:*

"(1) In childhood, loud, rough, musical heart-murmurs, with normal or only slight increase in the heart-dulness, occur only in congenital heart-disease. The acquired endocardial defects with loud heart-murmurs in young children are almost always associated with great increase in the heart-dulness.

"(2) In young children heart-murmurs with great increase in the cardiac dulness and feeble apex beat suggest congenital changes. The increased dulness is chiefly of the right heart, whereas the left is only slightly altered. On the other hand, in the acquired endocarditis in children, the left heart is chiefly affected and the apex beat is visible; the dilatation of the right heart comes late and does not materially change the increased strength of the apex beat.

"(3) The entire absence of murmurs at the apex, with their evident presence in the region of the auricles and over the pulmonary orifice, is always an important element in differential diagnosis, and points rather to septum defect or pulmonary stenosis than to endocarditis.

"(4) An abnormally weak second pulmonic sound associated with a distinct systolic murmur is a symptom which in early childhood is only to be explained by the assumption of a congenital pulmonary stenosis, and possesses therefore an importance from a point of differential diagnosis which is not to be underestimated.

"(5) Absence of a palpable thrill, despite loud murmurs which are heard over the whole præcordial region, is rare except with congenital defects in the septum, and it speaks therefore against an acquired cardiac affection.

"(6) Loud, especially vibratory, systolic murmurs, with the point of maximum intensity over the upper third of the sternum, associated with a lack of marked symptoms of hypertrophy of the left ventricle, are very important for the diagnosis of a persistence of the ductus Botalli, and cannot be explained by the assumption of an endocarditis of the aortic valve."

Treatment.—The child should be warmly clad and guarded from all circumstances liable to excite bronchitis. In the attacks of urgent dyspnœa with lividity blood should be freely let. Saline cathartics are also useful. Digitalis must be used with care, and it is sometimes beneficial in the later stages. When the compensation fails, the indications for treatment are those of valvular disease in adults.

* Die Auscultation des kindlichen Herzens, Wien, 1890.

III. DISEASES OF THE ARTERIES.

I. DEGENERATIONS.

Fatty degeneration of the intima is extremely common, and is seen in the form of yellowish-white spots in the aorta and larger vessels. *Calcification* of the arterial wall follows fatty degeneration, atheromatous changes, and sclerosis. It occurs in either the intima or the media. In the latter it produces what is sometimes known as annular calcification, which occurs particularly in the middle coat of medium-sized vessels and may convert them into firm tubes. Calcification of the intima is a common terminal process of arterio-sclerosis.

Hyaline degeneration may attack either the larger or the smaller vessels. In the former the intima is converted into a smooth, homogeneous substance, and it is commonly an initial stage of arterio-sclerosis. In the smaller arteries and capillaries the hyaline degeneration is often seen, particularly in the glomeruli of the kidney. Its exact production is still a matter of some doubt. "It appears to arise principally by homogeneous coagulation of an albuminous fluid, either within the vessels or infiltrating the cells and the hyaline transformation of proliferating cells and of leucocytes."

II. ARTERIO-SCLEROSIS (*Arterio-capillary Fibrosis*).

The conception of arterio-sclerosis as an independent affection—a general disease of the vascular system—is due to Gull and Sutton.

Definition.—A condition of thickening, diffuse or circumscribed, of the intima, consequent upon primary changes in the media and adventitia. The process leads, in the larger arteries, to what is known as atheroma or endarteritis deformans.

Etiology.—(1) As an involution process arterio-sclerosis is an accompaniment of old age, and is the expression of the natural wear and tear to which the tubes are subjected. Longevity is a vascular question, and has been well expressed in the axiom that "a man is only as old as his arteries." To a majority of men death comes primarily or secondarily through this portal. The onset of what may be called physiological arterio-sclerosis depends, in the first place, upon the quality of arterial tissue (vital rubber) which the individual has inherited, and secondly upon the amount of wear and tear to which he has subjected it. That the former plays the most important *rôle* is shown in the cases in which arterio-sclerosis sets in early in life in individuals in whom none of the recognized etiological factors can be found. Thus, for instance, a man of twenty-eight or twenty-nine may have arteries of sixty, and a man of forty may present vessels as much degenerated as they should be at eighty. Entire families sometimes show this tendency to early arterio-sclerosis, a tendency which cannot be explained in any other way than that in the make-up of the machine bad material was used for the tubing.

More commonly the arterio-sclerosis results from the bad use of good vessels, and among the circumstances which tend to produce this condition are the following:

(2) *Chronic Intoxications.*—Alcohol, lead, gout, and syphilis play an important *rôle* in the causation of arterio-sclerosis, although the precise mode of their action is not yet very clear. They may act, as Traube suggests, by increasing the peripheral resistance in the smaller vessels and in this way raising the blood tension, or possibly, as Bright taught, they alter

the quality of the blood and render more difficult its passage through the capillaries.

The poison of syphilis and of gout may act directly on the arteries, producing degenerative changes in the media and adventitia.

(3) *Overeating.*—Many authors attribute an important part of the etiology of arterio-sclerosis to the overfilling of the blood-vessels which occurs when unnecessarily large quantities of food and drink are taken. Particularly is this the case in stout persons who take very little exercise.

(4) *Overwork of the muscles*, which acts by increasing the peripheral resistance and by raising the blood-pressure.

(5) *Renal Disease.*—The relation between the arterial and kidney lesions has been much discussed, some regarding the arterial degeneration as secondary, others as primary. There are certainly two groups of cases, one in which the arterio-sclerosis is the first change, and the other in which it appears to be secondary to a primary affection of the kidneys. The former occurs, I believe, with much greater frequency than has been supposed.

Morbid Anatomy.—Thoma divides the cases into *primary* arterio-sclerosis, in which there are local changes in the arteries leading to dilatation and a compensatory increase of the connective tissue of the intima; *secondary* arterio-sclerosis, due to changes in the arteries which follow increased resistance to the blood-flow in the peripheral vessels. This increased tension leads to dilatation and to slowing of the blood-stream and a secondary compensatory development of the intima.

In a recent study of 41 autopsies upon arterio-sclerotic cases from my wards, Councilman * follows the useful division into nodular, senile, and diffuse forms.

(*a*) *Nodular Form.*—In the circumscribed or nodular variety the macroscopic changes are very characteristic. The aorta presents, in the early stages, from the ring to bifurcation, numerous flat projections, yellowish or yellowish white in color, hemispherical in outline, and situated particularly about the orifices of the branches. In the early stage these patches are scattered and do not involve the entire intima. In more advanced grades the patches undergo atheromatous changes. The material constituting the button undergoes softening and breaks up into granular material, consisting of molecular *débris*—the so-called atheromatous abscess.

In the circumscribed or nodular arterio-sclerosis the primary alteration consists in a degeneration or a local infiltration in the media and adventitia, chiefly about the vasa vasorum. The affection is really a mesarteritis and a periarteritis. These changes lead to the weakening of the wall in the affected area, at which spot the proliferative changes commence in the intima, particularly in the subendothelial structures, with gradual thick-

* Transactions of the Association of American Physicians, vol. vi.