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Coarctation of the aorta

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COARCTATION of the AORTA

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INTRODUCTION

The term coarctation is derived from the Latin (co- cum, together and arcta're, to press or make tight) meaning a localized narrowing or constriction of the aorta, Trautman (1937). Coarctation of the aorta is synonymous with congenital stenosis of the aortic arch. It may be defined as a narrowing of that part of the thoracic aorta known as the isthmus, or the part immediately distal to this. The degree of stenosis may be so severe as to actual obliteration.

Only ten percent of the cases of aortic coarctation have been recognized ante mortem, and its discovery at post mortem is often a surprise. According to statistics the condition is very rare. To the present time there have been approximately two hundred and seventy five cases reported in the literature. It can be shown, however, that coarctation is not so rare as was formerly thought. Blackford (1928) indicated its occurrence about one in every 1,550 autopsies. Clinical diagnosis was made only in 19 cases reviewed by Abbott (1928). This means that the great majority of such cases are being overlooked.

It is of my opinion that many cases of coarctation are being wrongly diagnosed as essential hypertension, chronic nephritis or basophilic adenoma of

the pituitary. How many physicians take the arterial tension in the lower extremities, or feel for the femoral and popliteal pulse in a routine physical examination? I would say very few. In coarctation one will always find hypertension in the arms and hypotension in the legs with diminished femoral pulse. Many cases of coarctation probably would be recognized if arterial tension in the legs would be taken after finding high tension in the arms. I think it is indicated, any way it would give one a lead to study the other possible findings and rule out essential hypertension or chronic nephritis with hypertension. Inexplicable hypertension in youths is extremely suggestive of coarctation.

Symptoms are not of note in this congenital anomaly and the diagnosis is made from physical findings and roentgenalogical studies. It has been my purpose to emphasize the important points in diagnosis so that cases of coarctation will be clinically recognized and not be treated as essential hypertension or kidney disease. It is true that chronic nephritis and coarctation may occur together.

Dr. Maude Abbott (1928) of McGill University has done more to advance the subject of coarctation than

any other scientist. I refer any one interested in this subject to read her beautiful masterpiece in which she reviews two hundred cases of coarctation of the aorta. Due credit belongs to Blackford (1928), King (1928), Railsback and Dock (1929), Fray (1930).

HISTORY

It was not until nearly the beginning eighteenth century that the medical world became conscious of the clinical entity, coarctation of the aorta. A French prosector in Paris by the name of Paris was first to describe a case of coarctation. However, Morgagni described in 1760 a true case of coarctation from a post mortem on a Monk. Morgagni should be given full credit because Paris' case was doubted.

Legrand in 1835 was the first to diagnose this developmental anomaly on evidences of collateral circulation above the aortic obstruction, diminished pulse below the stenosis. He considered these cardinal features pathognomonic. This diagnosis was made in a man 48 years old who was shown at autopsy to have an extreme stenosis of the thoracic aorta. The actual diagnosis of stenosis of the aorta at the insertion of the ductus, i.e., true coarctation was made by Apploger

in two cases published by Hamernik in 1848. In 1855 Skoda argued that fibers from the ductus arteriosus extended into the aortic wall and that these fibers contracted with the contraction of the ductus in the process of its obliteration.

Scalloping of ribs was first described in 1929 by Railsback and Dock and again by Fray (1930), although it had been recognized in gross necropsy specimen in 1827 by Meckel.

AGE, INCIDENCE and SEX

In considering the ages of patients with this anomaly, it should be said that the unfortunate is born with this condition, the "infantile" type being incompatible and the "adult" type compatible with life. The infantile coarctation patients die in a few years and the average age of the adult form diagnosed is 23 years (Evans 1933). In 198 cases reviewed by Abbott (1928), 129 cases occurred in patients over one year old.

King (1926) recorded four instances of coarctation in patients 35, 40, 54, and 58 years of age, drew the attention to two critical periods through which patients pass, i.e., early infancy and third decade.

The predilection for this congenital abnormality has been noted in the male sex. In 136 cases of uncomplicated coarctation in adults reviewed by Bonnet (1903), there were 96 males and 37 females.

The incidence of cases diagnosed clinically and at post mortem show the condition to be rare. Fawcett (1905) expressed the rarety of the lesion. He reported that there were only 18 cases of coarctation out of some 22,316 autopsies performed since 1826 at Guy's hospital.

Evans (1933) presented 28 cases; 26 cases were found among 19,217 routine necropsies conducted at Bernhold Baron Institute of Pathology, London.

Abbott (1928) in her magnificient review of 200 cases reported that only 19 cases had been diagnosed clinically. Blackford (1928) reported coarctation occurs once in about 1550 cases at autopsy. If this is true, many cases are clinically missed.

ANATOMICAL FEATURES and its RELATION TO CLASSIFICATION

Physicians and scientists have long read and heard of the classification of coarctation of the aorta as differentiated into the two types, infantile and adult.

The infantile type is incompatible with life and is a congenital anomaly from mal-development embryologically. The theories of this condition will be brought out under pathogenesis. Briefly this type anatomically is narrowing of the fetal isthmus of the aorta. The isthmus lies between the origin of the left subclavian and the insertion of the ductus arteriosus in the descending aorta. The constriction of the infantile type very rarely begins abruptly; it generally is found to diminish progressively in caliber to maximal narrowing which is maintained for a certain distance. McKenzie (1927) said that the isthmus has acquired that name from the fact that during fetal life, the isthmus is used very little and at birth is generally found to be smaller than the aorta proximal and distal to it. Normally, this narrowing disappears soon after birth except as McKenzie (1927) states that in 6% of individuals a slight constriction of the isthmus persists. Unless the constriction amounts to more than 2 mm it is not considered abnormal.

The adult type is a congenital, acute constriction of the isthmus of the aorta at or near where the ductus arteriosus joins the aorta. Abbott (1928) reported that 90 cases out of 183 studied showed the stenosis at the insertion of the ductus and 43 other cases just below the tissue of botalli: In Abbott's review, 19 cases the exact seat of the stenosis was not mentioned and in 18 others, below the subclavian.

Evans in 1933 propounded a new classification. It is his idea to do away with the etiological background as to classifying cases as to adult and infantile. His cases are allotted to different groups according to the nature of the anatomical deformity present and the arrangement of the associated developmental lesions. From this basis he has defined six separate types of congenital stenosis and atresia. Evans (1933) brought out that in order to allocate a case to its particular group it is necessary to consider the following data:

(1) "The site, nature and extent of constriction, (2) condition of the aorta proximal to the site of the stenosis, (3) patency or otherwise of the ductus arteriosus, (4) relationship between systemic and pulmonary circulation."

The types defined by Evans (1933) are:

(1) "Congenital stenosis of the aortic arch-- ductus arteriosus patent-- hypoplasia of the proximal portion of the aorta.

(2) Congenital stenosis of aortic arch-- ductus arteriosus closed-- hypertrophy of proximal portion of aorta.
(3) Congenital atresia of distal portion of aortic arch. Ductus arteriosus closed-- hypertrophy of proximal portion of aorta.
(4) Interruption of aortic arch in its distal portion-- ductus widely patent-- hypoplasia of proximal part of aorta.
(5) Congenital atresia of proximal portion of the aortic arch-- ductus arteriosus patent.
(6) Congenital absence of ascending aorta-- ductus arteriosus patent."

Gitlow (1937) reported that types (2) and (3) are compatible with more or less prolonged life.

PATHOGENESIS

Coarctation of the arch of the aorta below the origin of the left subclavian has been differentiated by Bonnet into two distinct groups, classified by him as "infantile" and "adult" types, according to the site and form of the atresia. His infantile form means narrowing of the thoracic aorta between the origin of the left subclavian artery and the insertion of the ductus arteriosus, this region being known as the fetal isthmus. This type may be regarded as a persistence of the anatomical relations that exist before birth.

Bonnet (1903) termed the adult type, a pathological condition which does not exist in normal intra-

uterine life, consists of a sharp constriction, in some cases to complete obliteration of the lumen of the descending aorta, at, just above or below the insertion of the ductus arteriosus.

Various theories have been propounded for the explanation of this congenital anomaly. The first theory was promulgated by Craige in 1841. His theory is, "The region of the obstruction corresponds exactly to that of the junction of the ductus arteriosus with the aorta, and the contracted point, though a little lower down, was still slightly connected with the trunk of the pulmonary artery. It seems therefore, that the obliterating action which has taken place in the ductus arteriosus had been for some peculiar cause, prolonged into the aorta and had then given rise to contraction and then to obliteration of the coats of the vessel." This theory has been named the Skodaic theory.

Reynaud (1828) considered coarctation a persistence of the fetal state which was influenced in some way by the involution of the ductus. According to Blackford (1928) the ductus arteriosus in no was deals in the pathogenesis, and that the anomaly could be traced back to intra-uterine life.

Prenatal development of coarctation in all cases has never been universally accepted. Others attribute it in some cases propagation of an inflammatory process in the ductus botalli to the wall of the aorta.

Blackford (1928) states there is no question infantile type is a result of prenatal mal development.

He challenges Bonnet's (1903) deduction that the adult type is the result of post nasal contraction of Botallian tissue in the wall of the aorta. He says, that it has been shown that fibers from the ductus arteriosus do not normally extend appreciably into the wall of the aorta. It has not been demonstrated that they ever do.

Strong reports in 1932, "The cause of coarctation is still a matter of conjecture." According to him, it is congenital malformation arising as a result of factors acting during intra-uterine life.

John Fawcett writing in 1905 published his idea that the anomaly is due to defective development of the positions of the branchial arches which go to form the aorta, between the left subclavian and the ductus arteriosus.

A report published in 1900 by Herman Schlesinger reports that the anomaly is a persistence of the so called isthmus aortae-- "that narrow vascular tube which during fetal life unites the thoracic aorta below the left subclavian with the point of origin of the abdominal aorta, the latter, as is well known, having its blood flow at this time from the right ventricle thru the ductus Botalli. In extra-uterine life there is a dilatation of the isthmus aortae. If, however, the isthmus does not dilate, or does so insufficiently, a stenosis of the aorta forms, which on further growth of the individual causes increasingly disproportion between the size of the arch and of the isthmus."

Evans (1933) believes the deformity due to congenital maldevelopment and challenges the post-nasal or Skodaic theory. His arguments are (1) in most of the adult cases the constriction occurred at some little distance from the site of insertion of the ductus, (2) ductus remains open at its aortic extremity in many cases reported, (3) ductus at times remains patent, (4) there are other anomalies, usually cardio vascular, (5) there never has been any report of constriction in the pulmonary artery.

There is another theory which Ulrich (1932) tends to disprove the Skodaic theory. The descent of the diaphragm changes the direction of the currents of the blood between the auricles and the ductus arteriosus. Ulrich states, "When the right crus contracts at birth with the first inspiration, it draws the pulmonary arteries and the fixed margin of the vestibule with it, but the aorta is fixed otherwise and scarcely yields. Hence a decided traction is exercised on the ductus arteriosus enough, I believe, to stop the flow of blood from the pulmonary artery to the aorta and turn it into the lung which at the same time is expanding-- -- --. This contraction of the right crus, which helping to expand the lung, also closes the foramen ovale and ductus arteriosus."

Blackford (1928) summarizes that etiology of adult coarctation, narrowing or obliteration of the aorta in the region of the mouth of the ductus arteriosus or its vestige as believed to be due to absence, atrophy, or imperfect development of the left fourth

branchial arch.

There has been little evidence to show that syphilis can obliterate a large artery.

PATHOLOGY

Blackford (1928) states that the classification into infantile and adult type simplifies the pathology of coarctation. Abbott (1928) said the so called adult type, a pathological condition exists without counter part in normal intra-uterine life, which consists of a sharp constriction, amounting in some cases to complete obliteration of the lumen of the descending aorta, adjacent to the insertion of the ductus arteriosus, either immediately at this point or just above or below it. Abbott in her analysis of 200 cases found 37subjects had complete atresia of the aorta. In marked cases of the adult type, the constriction is so sudden and deep that the descending arch appears as though a ligature has been tied around it. In moderate cases there is a gradual diminution from the innominate or left subclavian, rapidly narrowing after the origin of the latter vessels to the point of constriction. Due to this external strangulation, there is a narrowing of the lumen of the vessel.

Frequently, there is a fold, septum or a diaphragm which stretches across what remains of the aortic lumen, either closing this or leaving a small central or lateral aperture.

Schlesinger in 1900 reported that "one-third of the cases showed atheroma immediately above the stenosis. Much more striking is the repeated finding of the atheromatous degeneration immediately below the contraction. These conditions may be due t o the fact that blood flows into the abdominal aorta by means of a collateral circulation and then takes a course opposite to the normal in the wide tube of the aorta back to the obstruction; for, according, to a well known physical law, when the liquid in a tube is under pressure all parts of the tube are subject to equal pressure. If, when this reserved blood flow reaches the stenosis, the same condition of poor nutrition exists below as above it, the arterial wall is subject to a strong and continuous pressure and degenerates on this account by an atheromatous process."

The ductus which is usually obliterated fixes the contiguous part of the aortic wall and may even appear to draw it toward the heart. The heart may be normal in every way; developmental anomalies are rare in the adult form other than bicuspid aortic valve. Lecount (1913) states that lesions of the aortic valves are common with stenosis at the isthmus or coarctation of the aorta. These changes in the valves in many instances of coarctation are probably the result of repair, of thrombosis and organization in minute rupture of the leaflets. In contrast to inflammatory condition such

as infectious endocarditis, the alterations in the aortic valves with coarctation are generally within the sinuses and not on the ventricular surfaces. However, Fawcett said in 1905, the constant strain to which the aortic valves are subjected leads in the majority of cases to a chronic inflammatory change, with the result that the valves become thickened or adherent or the orifice stenosed. In no less than 10 cases reportedly by him were aortic valves affected and in six of them the mitral valve showed a similar change. He reported 18 cases.

Blackford (1928), Abbott (1928), Dickinson and Fenton (1900) report that hypertrophy and dilatation occurs in the majority of cases. Abbott and Blackford have analyzed that three-fourths of the cases show hypertrophy of one or both ventricles. Lewis (1931) says that the enlargement is usually due to hypertrophy rather than dilatation.

Blackford (1928) says the ascending aorta is often dilated to a varying degree and may be the seat of an aneurysm, and sometimes there is an aneurysm distal to the stenosis. The aortic wall is at times thicker than normal, but is usually thinner. According to Dickinson and Fenton (1900) the constriction is generally produced

by a fold of the inner coats of the aorta, the outer coat being continued over; very rarely the aorta has been found reduced to a fibrous cord. The inner surface may be normal, or it may show any degree of arteriosclerosis, sometimes with extensive calcification. G. R. Murray reported a case in 1906 when the whole of the aorta was lined by a calcareous plate.

Microscopic study of the stenosed and degenerated area itself has revealed little besides connective tissue. Many authors believe that diminution of elastica with replacement by connective tissue is the essential feature. Oppenheim (1918) reported a case in which he considered the aorta essentially normal both grossly and histologically.

The infantile pathology makes this condition incompatible with life. As found in fetuses, still born and young infants, the coarctation is usually a diffuse narrowing of the isthmus of the aorta, this segment may be represented by a fibrous cord or be altogether lacking. According to Blackford (1928),

"The ductus is patent in almost inverse ratio to the lumen of the isthmus and when the latter is atresic, the pulmonary trunk is continued with undiminished diameter into descending aorta."

There is no collateral circulation.

ASSOCIATED ANOMALIES

Coarctation of the aorta is frequently associated with some other congenital deformity of the cardiovascular system, but each may exist as the sole manifestation of faulty development. Applebaum and Kalkstien writing in 1938 reported that the major or main complex anomalies are more commonly combined with the infantile type, while minor anomalies are more frequently found in the adult form. The outstanding major anomalies are bilaculate or triaculate heart, transposition of the arterial trunks and pulmonary atresia. The more important of the minor anomalies include bicuspid aortic valve, anomalous origin of the arteries of the arch, persistent left superior vena cava, defects of the aortic system, sub-aortic stenosis and minor aneurysms.

W. Dickinson and Fenton (1900) in studying necropsies reported twelve chief associated malformations: (1) patent ductus arteriosus, (2) transposition of the heart (3) two aortic cusps, (4) patency of the undefended space, (5) absence of musculi papillares in the left ventricle, (6) hair lip and cleft palate, (7) hypospadias, (8) deformity of the lower extremity, (9) two anterior communicating branches in the circle of Willis, (10) innominate and left carotid arteries arising from a common trunk, (11) triple division of

the aorta at its origin, (12) left carotid and subclavian arising from a common trunk.

Hardaway and Sawyer (1934) in reviewing 22 cases showed that bicuspid aortic valve occurred in four cases while patent ductus was present in two cases. Wechsler and Gustafson (1937) reported a case with congenital bicuspid aortic valve to show the tendency for the malformed valve to become the seat of an infective endocarditis. Benkwitz and Hunter (1937) wrote that the bicuspid aortic valve is the most common of the associated anomalies.

SYMPTOMS

The subjects are usually robust, rather plethoric looking, athletic young individuals with good musculature, and intelligence often above average (Abbott 1928). Symptomatology may be divided into four groups according to Farris in 1935. (1) Symptoms associated with hypertension in the upper part of the body, such as nervousness, headaches, epistaxsis, dizziness, flushing of the face and a constant high color. Shapiro (1933) said that these patients may be unusually bright, due to the excessive blood supply to the head and he also states that because of the increased blood supply to

the upper part the patient may be mistaken for hyperthyroidism. (2) Symptoms due to low blood pressure and poor circulation in the lower extremities, such as numbnness, cold feet, tiredness and vague pains in the legs and intermittent claudication. (3) Symptoms of over action of the heart and later, cardiac failure. (4) Symptoms due to cerebral hemorrhage.

King (1926) in reviewing cases of coarctation writes that most cases show symptoms of myocardial insufficiency, that is, dyspnea, palpitation, edema. He states that intermittent claudication is a rare symptom. King (1926) says nocturia is common and believes it an important symptom, especially as it usually occurs without evidence of nephritis.

Blumgart, Lawrence and Ernstene (1931) published a series of cases and their second patient had subnormal temperature of the feet and occasional nocturnal cramps in the legs.

In some of the most marked cases symptoms have been absent throughout life, but more times than not there are symptoms of cardiac insufficiency. Many authors have reported such cases as Eppinger and Medlefart (1933) when there were no symptoms referable to cardio vascular system. R. O. Moon 1912 writes cyanosis is said to be rare.

According to Narr (1929) symptoms of coarctation when present are; (1) dyspnea usually not as marked as in other forms of congenital heart disease, (2) Results of an unequal circulation with unequal pressure, such as headache and coldness of lower extremity.

In the infants, wrote Shapiro in 1933, "The exact nature of the lesion can rarely be established; a tentative diagnosis of congenital heart disease is usually made." According to Shapiro the symptoms of numbness and weakness in both legs when ever the patient assumes an upright posture after reclining for some little time in the horizontal position was pathognomonic of coarctation of the aorta.

Abbott in 1928 placed patients according to symptoms in three groups: (1) Those in whom symptoms are absent, (2) those in whom symptoms are late in developing, (3) those in whom symptoms are present throughout life.

Rumold and Schwartz (1934) in discussing symptoms said that the latency of symptoms is accounted for by the marked and extensive collateral circulation, the freedom of injuries to the myocardium and the remarkable ability for the heart to compensate.

Waltman and Sheldon (1927) reported a review of cases stating the neurological symptoms. The following percentages of symptoms occurred: Headache 44%, hemiplegia 34%, convulsions 19%, rapid fatigue and sensation of said in lower extremities 10%, vertigo 12.5%, tinnitus 9.4%. However, these authors explained that these symptoms should be considered neurological complications of coarctation.

DIAGNOSIS

Purks and Robert in discussing diagnosis of coarctation in 1935 said, "Congenital heart disease is a subject that holds little interest for the general medical profession." Such a feeling has in general been due to and in a measure justified by the fact that in the hands of the most familiar with congenital heart disease, clinical and post mortem diagnoses have shown poor conformity and because therapy of all types of lesions is unsatisfactory.

As brought out previously, the statistical studies indicate there occurs one case in every 1550 autopsies. Maude Abbott's beautiful study in 1928 reported that clinical diagnosis had been proven by autopsy in only 19 cases reviewed by her. This shows that a great

majority of cases of coarctation are being over looked. According to Farris (1935) the most severe case of coarctation may be difficult to diagnose as the collateral circulation follows a relatively direct course through widely dilated vessels, so may not affect the leg pressure and pulse as would be expected. Other conditions that tend to avoid detection of coarctation is that great majority of patients show vigorous health, complaining of no symptoms. To show that in former years the diagnosis was clinically missed, of the 18 cases collected from the records of autopsies at Guy's Hospital by Fawcett in 1908, not a single case had been diagnosed during life.

Abbott (1928) states that the "characteristic signs of coarctation when present, are vascular rather than cardiac, the effects of the gradually extending collateral circulation." There is usually a period of latency which often lasts on into early adult life, and that these signs frequently appear for the first time, accompanied by slight dyspnea and palpitation on exertion. These cardio-respiratory symptoms occur after an intercurrent infection has crippled the myocardium or some undue physical strain has lessened the cardiac reserve.

Statistical studies of coarctation show the rarity of the diagnosis clinically. John King 1926 reported only four cases have been recognized during life at Johns Hopkins Hospital. However, post mortem findings indicate that the condition is not extremely uncommon, so writes Ernstene and Robins in 1931.

Granting, then, that this form of congenital heart disease is not uncommon and that it can be diagnosed, why is it important to make such a diagnosis? Most of the cases that are over looked pass under a diagnosis of chronic nephritis with hypertension or as essential hypertension. Each of these offers a lower hypertension. The patient is usually advised that his or her span of life will probably not exceed a few years. Such a pessimistic attitude is not warranted for most cases of atresia of the isthmus of the aorta.

In youths where hypertension is encountered, Purks (1937) writes a differential diagnosis should be made between, (1) chronic nephritis, (2) basophilic adenoma of pituitary and (3) coarctation of the aorta.

In many cases of stenosis of the aortic isthmus of the adult type, according to Weber (1927) there are clinical signs of aortic regurgitation but without history of acute rheumatism or evidence of past syphilis.

The aortic regurgitation is probably due to excessive strain on the proximal aorta and simple atheromatous or sclerotic changes in the valves. Furthermore, aortic regurgitation is apt to be diagnosed in these cases when it is not really present.

BLOOD PRESSURE:

According to many authors, Purks and Robert (1935), Blackford (1928), Lewis (1931), it is believed the best single lead in arriving at the diagnosis clinically is the finding of an elevated blood pressure. Not all but certainly the majority have a hypertension in the upper extremities. There have been cases reported, however, in which the blood pressure is low and cardiac failure with congestion, or auricular fibrillation is present.

It is to be assumed that if high pressure is found in the adult case, it has been present, continuing from the first years of life.

Lewis concluded in 1931-33 that most cases of coarctation are uncomplicated and present high blood pressure; the statement applies to both systolic and diastolic readings.

The pathogenesis of arterial hypertension in coarctation was studied by Rytaud in 1938. He per-

formed experiments on rats showing that hypertension in stenosis of the aortic isthmus has the same pathogenesis as the hypertension in a partial constriction of the renal artery. The criterion of the hypertension was the presence of cardiac hypertrophy. Partial occlusion of the left renal resulted in cardiac hypertrophy 28% in rats. Partial occlusion of the aorta between the kidneys resulted in cardiac hypertrophy in 26%. Partial occlusion of the aorta above both kidneys resulted in cardiac hypertrophy. This situation is analagous to that existing in coarctation of the aorta. Rytaud 1930 concludes that "partial (or even complete) occlusion of the aorta in rats produces hypertension only if there is living renal tissue distal to the occlusion just as there must be a kidney beyond a partially occluded renal artery in order to produce hypertension in a goldblatt dog. The same degree of mechanical obstruction due to stenosis of the aorta and the presence of collateral bed never results in hypertension when all of the renal tissue is above the site of occlusion."

The earlier writers on this subject believed that the hypertension was due to the fact that the blood pressure is always high, so as to overcome the difficulty of getting blood through the narrow arterial channels, so wrote Gossage in 1908.

King (1928) reported that in 66 cases in which the blood pressure was recorded in only 10 of these was there an appreciable difference in the blood pressure.

Gossage in 1908 reported that the pressure in the right arm is usually higher than the left.

Hills wrote in 1938 that anatomical narrowing of the mouth of the left subclavian artery does occur in coarctation of the aorta and to him it seemed reasonable to assume that this is the explanation of the occasionally finding in such cases of normal blood pressure in the left arm associated with marked hypertension in the right arms.

Study of coarctation clinically and in post mortem study has provided research work on the relation between arterial pressure and the degree of arterial sclerosis. Blumgart, Lawerence and Ernstene (1931), and Stewart and Bellet (1934) reported that in patients with essential hypertension, arterio sclerosis appears early and progresses rapidly. It is still not clear whether this is due to increased arterial pressure or to the underlying pathalogic process. These authors said that it was remarkable that both of their patients of coarctation, arterial hypertension had been present for forty years above the site of stenosis. Physical examination and x-ray studies failed to disclose any appreciable difference in degree of arterio sclerosis in the upper and lower parts of the body.

Rooke (1938) states high blood pressure as a regular feature of coarctation of the aorta. Sir Thomas Lewis (1933) wrote, "We still lack records covering the periods of childhood and adolescence during which seemingly very few cases are diagnosed. While it may be highly probable that coarctation means high pressure from a time shortly after birth to the time when cardiac failure supervenes or death occurs from other cause, the gap that is still present in our evidence forbids us to draw the correspondent conclusion. High pressure is life long in coarctation and is of value because these cases are useful in studying the effects of long lasting high pressure of a relatively uncomplicated kind."

Cases of coarctation have not yet been recorded without high pressure but with normal heart and weight after death, it is possible that they will be recorded. Cases do occur and are not infrequent in which with very high pressure, the heart presents no signs of enlargement clinically, even when examined by x-ray.

COLLATERAL CIRCULATION:

As has been brought out hypertension is a lead to diagnosis of coarctation. Having discovered the latter, one looks for evidence of collateral circulation in the thorax and neck. The carotid arteries will often be dilated and tortuous and pulsate forcibly. One will often find abnormal pulsations or bruits in the inter scapular region or axillae.

It should be brought out here as propounded by Applebaum and Kalkstein (1938) that the diagnosis of coarctation of the aorta was first suspected because of the presence of external developmental defects such as a pulsating vascular artery.

This collateral circulation is brought about by the stenosis of the aorta and the degree of superficial pulsating arteries is due to amount of atresia. Many of the cases that have been diagnosed were given by lead of a pulsating palpable vessel. These latter vessels are found perhaps as stated above in the neck and thorax but most frequently found along the dorsal border and angle of the scapula. In many cases pulsating internal mammary vessels are palpable. The superficial pulsating arteries in the axilla and subscapular regions flow obliquely from above downward into the intercostal arteries. Herman Schlesinger in 1900 reported a case that on palpation of the left axilla, lying against the wall of the thorax, found a group of cord like vessels. They were pulsating blood vessels of the thickness of a pen holder.

The collateral circulation is brought about by anastomosis of the upper three aortic intercostals with the superior intercostal artery; dilated,

tortuous internal mammaries with deep epigastrics; transverse cervicals, suprascapular of the subclavian, and branches of the axillary artery with the intercostals. It is these pulsating intercostals that produce a diagnostic finding in many cases, i.e., notching of the lower borders of the ribs.

It should be remembered that even in the most extreme cases the diagnosis may be made difficult because the collateral circulation follows a relatively direct route through widely dilated vessels. Also, the absence of collateral circulation does not rule out coarctation. Blackford (1928) reported an interesting case in which a mediastinal tumor produced typical collateral circulation.

Collateral vessels, says Abbott (1928), transmit a systolic or post systolic murmur and frequently a fine accompanying thrill over the areas occupied by the main and terminal branches of the subclavian arteries. When the arteries involved in the anastomoses lie deeply below the surface, as in the cases of those scapular branches coursing along the posterior and inferior borders or in the infra spinous fossa of the scapula, or of the internal mammaries which are screened by the costo sternal articulation, a systolic murmur and

sometimes also a thrill may exist without other visible or palpable evidence.

CARDIAC SIGNS:

As we shall see most cases of coarctation reveal a hypertrophied heart rather than a dilated heart. In 200 cases analyzed by Maude Abbott (1928) 75% of the subjects show hypertrophied hearts. A usual finding, the supra cardiac dullness is increased. A systolic murmur is almost invariably heard over the precordium, usually best heard at the base. Abbott (1928) says the murmur has its maximum intensity along the left sternal border, which is thought to be generated at the constricture. The signs over the heart other than those already mentioned are those complicating valvular lesion which is so frequently associated, or they are produced in the dilated and tortuous collaterals adjacent to the precordium. A murmur so originated differs from a valvular one in being usually somewhat prolonged and beginning a little after the first sound, i.e., post systolic rhythm, so wrote Abbott in 1928. Such murmurs also differ somewhat in timber from the ordinary valvular murmur, being usually high pitched and of a more rasping quality. According to Purks (1935) a diastolic murmur of aortic insuffic-

iency (due to bicuspid valves or endocarditis) is occasionally present.

Blackford (1938) reported that precordial murmurs are of little consequence as they are sometimes absent. He says they may be caused by super imposed endocarditis. The murmur in the inter scapular space is of a greater importance.

VASCULAR SIGNS:

In many cases of coarctation a curious feature is sometimes noted between certain physical findings in the upper and lower parts of the body. Abbott (1928) described a boy of five, a peculiar "vascular surcharge of the head and chest and atony of the subdiaphragmatic viscera, slight edema of the lower extremities in the presence of marked ascites." In some cases the upper part of the body is better developed than the lower. One case reviewed by Abbott showed marked disproportion in the crural pulse and muscular energy in the lower extremities. Another patient complained of coldness of the feet. One patient complained of pain in calves of the legs, swelling of the feet on walking. Intermittent claudication was present in two cases diagnosed clinically by John T. King (1926).

Over taxing of the circulation in the upper part of the body is usually brought forth when a break in compensation is impending, by sense of fullness in head, buzzing of the ears, flushing and lividity of the face, stabbing pains in the back, shoulders, extremities and over the precordium.

PULSE:

The tension and form of the pulse in the upper and lower extremity is by far the most constant and distinctive clinical feature of adult coarctation according to Abbott in 1928. Like lower arterial tension in lower extremities the femoral, popliteal, dorsal pedis pulse is diminished or absent. Blumgart, Lawrence and Ernstene writing in 1931 said that the retardation and diminution or absence of the femoral pulse are not constant either in this presence or degree. The amplitude of the femoral pulsation is affected not only by the degree of patency of the aorta but also by the caliber of the collateral pathways and by the directness of the route taken by the blood back to the thoracic aorta.

Lewis in 1931 took records simultaneously from two vessels in his patient, in these records the femoral pulse was seen to rise much more slowly than the radial.

In normal subjects the femoral upstroke begins first because the vessel is a little nearer the heart, about .01 seconds before the radial upstart. In cases of coarctation the radial pulse begins in the average .03 seconds before the femoral, so writes King (1926) Abbott (1928), Blackford (1928) and Lewis (1931).

Blumgart, Lawrence and Ernstene (1931) wrote that the velocity of the blood flow in the larger arteries of the legs was reduced and the arterial--arteriolar difference was greatly diminished. According to measurements of oxygen in the blood, they found the blood supply to the tissues under resting conditions and was never the less within the limits of normal. Gitlow (1937) reported that the femoral oxygen saturation was less than that of the brachial.

ROENTGENOGRAPHIC DIAGNOSIS:

In 1926 King said "Roentgenograms are disappointing." Blackford in 1928 said "Roentgenographic examination of the thorax is of value in excluding anneurysm which has been the most common erroneous diagnosis, but the presence of aneurysm does not exclude coarctation." The earliest writers on coarctation were well aware of the fact of the erosion of the ribs being often present. They attributed this to

dilatation and tortuosity of the intercostal arteries which plays a great part in establishing collateral circulation. It remained, however, for Railsback and Dock in 1929 to call attention to this roentgenalogic finding as being pathognomonic of coarctation. Railsback and Dock (1929) stated that "occasionally a local expansile pulse aneurysmal to feel and sometimes strong enough to gradually wear away the ribs may be fealttfrom place to place in the intercostal vessels."

Fray, writing in 1930, said if stenosis of the aortic isthmus is suspected, a very careful roentgenoscopic examination of the thoracic aorta should be made from all angles, searching particularly for a defect in the continuity of the arch, unusual pulsations and dilatation of the ascending aorta, with possible aneurysm from dilatation of the weakened wall proximal to the obstruction. One should look for collateral circulation changes with enlargement of the vessels in the superior mediastinum and finally erosion of the ribs. According to Ernstene and Robins (1931) bi-lateral erosion of the inferior borders of the ribs occurs only in coarctation. The usual routine postero-anterior film and left postero-anterior oblizue of the chest should be obtained. The latter should be made with the patient in proper position to outline

the aorta to its greatest extent. This will usually be obtained when the patient is rotated clockwise from the usual postero-anterior position 35 to 60 degrees, depending upon its appearance during the roentgenascopic examination.

To review the fundamental points of roentgenalogical studies are five:

(1) Absence in aortic know in postero-anterior view.
(2) Defect in Aortic arch in left A P oblique.
(3) Rib erosion.
(4) Dilated ascending aorta.
(5) Left ventricular hypertrophy.

The usual postero-anterior, of a normal adult chest shows a rounded eminence due to the left spine at the level of the fifth dorsal vertebrae. The aortic knob, representing the junction of the transverse portion and descending limb of the arch is absent in cases of coarctation because it represents the site of the isthmus. This finding in the P A view affords direct evidence and leads to a complete cardiac roentgen study with oblique views.

Fray (1930) stated that "The great advantage of of the oblique view is obvious in the study of coarctation in Youth. The absence of the aortic knob in P A film in youth is not a reliable sign because of its unconspicious character in normal subjects, but the demonstration of a local defect in the oblique view makes the diagnosis certain."

Gitlow (1937) emphasized

that the absence of aortic knob is not pathognomonic.

Lewis writing in 1931 says it is important to recognize that A P silhouette is valuable but has no very definite characteristics. It is usual for the ascending aorta to contricute to the right margin and the bulge of this vessel to the right may be conspicious. Lewis (1903) reports that there are certain constant features that can be regarded as significant. First, there is the increased breadth and density of the shadows of the basal vessels; this is associated with their dilatation, but he says it is not characteristic of coarctation because the dilated basal vessels occur in cases of simple high blood pressure. Secondly, Lewis says, their inability to trace out the aortic arch is seen, a fact particularly significant when it applies to the left oblique position. According to Lewis, it is more noteworthy because a normal aorta under strain of high internal pressure is wide and throws a dense shadow.

A defect or break in the continuity of the arch is pathognomonic of coarctation according to Fray (1930). No other condition ofwhich radiologists are aware will produce this result. Arteriosclerosis, dilatation secondary to hypertension and aneurysm produce radically

different shadows. It is thought that the latter two tend to accentuate the aortic shadow. In most instances a localizing defect in the descending aortic arch will aid in differentiating coarctation from the generally hypoplastic aorta.

Rib erosion was said to be pathognomonic by Railsback and Dock in 1929 and this condition was first recognized by Meckel (1827) early in the mineteenth century. Dock reported in 1932 that he had obtained silhouettes typical of coarctation in patients with normal blood pressure, no rib erosion. He said then that the silhouette was not pathognomonic of rib erosion.

Fray (1930) reported characteristics of rib defects in cases of coarctation: (1) "The lower margins of the rib only is affected.
(2) The sulcation is smoothly curvilinear.
(3) The defects are multiple, affecting more than one rib and not infrequently producing multiple defects in the same rib.
(4) Simple erosion of the rib occurs when it is in contact with the artery without any alteration in bone trabeculation or other evidence of pathological change elsewhere in the rib.
(5) New bone formation is essentially absent though occasionally the sulcated margin may be brought out into striking relief by a smooth line of increased density in the deeper portion of the sulcation.
(6) They are bi-lateral, affecting particularly the posterior portion of the ribs.
(7) Pathological fracture has not been noted though erosion may involve half the diameter or more."

Not every case of coarctation will show a collateral

circulation, therefore perhaps no extreme rib defects. This will depend to a large extent upon the degree of stenosis and in part on its duration, as believed by Sangster (1937).

In a case reported by Ernstene and Robins (1931) opportunity was offorded by these men to corraborate the roentgenographic and roentgenoscopic findings by vecropsy. After death, the arterial vascular system was injected with a suspension of barium sulphate in agar and sodium iodide solution and additional roentgenograms were taken to verify certain of the observations made during life.

MORTALITY AND DEATH

The infantile type of coarctation (that type which is a congenital narrowing of the arch of the aorta between the origin of the left subclavian and the insertion of the ductus arteriosus) is not compatible with life and usually die within a few years. This type is more commonly associated with other deformities and the prognosis is very grave.

The adult form of this anomaly is not diagnosed early because the condition is compensated by adequate collateral circulation. These patients usually enjoy

good health and are physically fit and robust. It is not until undue strain is put on the circulatory system that symptoms appear. Maude Abbott (1928) in her analysis of 200 cases from the literature shows that spontaneous rupture of the heart or the aorta took place in 40 cases-- 20%, while in four others rupture of a mycotic aneurysm of the aorta occurred, making a total of 44 fatalities from rupture of the aorta. In 1938 Register and Innes reported a case of extreme stenosis (adult) of the arch with death caused by spontaneous rupture of the ascending portion and resulting hemo-pericardium. This patient had suffered neither cardiac embarrassment nor disability of any kind previous to sudden fatal attack. Pierce (1934) in reviewing the literature reported that he found six cases in which death occurred from a rupture of a mycotic aneurysm. In ten of the two hundred cases analyzed by Abbott there was a mycotic endarteritis in the descending arch, at or just below the coarctation; 24 of the 200 died from the effects of a cerebral lesion, most likely hemorrhage. The most common cause of death is usually cardiac failure. Abbott (1928) reported 60 patients dying from failing compensation. Seventeen cases died suddenly from cardiac asystole or without

apparent cause. Schroder (1936) reports a case of a bright lad, eight years old who dramatically dropped dead. He emphasizes the fact that death may ensue from aortitis, plaques, abscess in the wall of the aorta with destruction.

Evans writing in 1933 represents the average life span as 23.8 years. Lebman (1902) reported the average duration of life was 34 years. The highest mortality is in the second and third and forth decades according to Abbott (1928). She reports seven patients died in the sixth decade and six patients lived to be over three score and ten years. This has been one instance-- 92 years of active life. He was a patient of Reynaud (1828) and at autopsy showed no ventricular hypertrophy so common to this condition. Of the 200 cases of Abbott's 27% died before the end of the second decade and 26% lived over forty years. Hardaway and Sawyer (1934) reviewed 32 cases subsequent of Abbott's 200 that the majority of cases deaths occurred in the third decade.

The high mortality that attends coarctation of the aorta during the prime of early middle life, together with the peculiar latency of its symptomatology in most cases before this period is reached, makes this

condition one of great importance from a medicolegal stand point.

PROGNOSIS

In considering the prognosis of the adult type of coarctation, it is more evident among authors that there is a tend to be more optimistic as to the number of years of life the patient has left. A case diagnosed in the early teens without complication will probably live to be forty years old, if he limits his activities in accordance with his cardiac tolerance and perhaps longer. Several cases have been reported to have lived to be over 70 years and one lived to be 92 years old.

Infantile types usually are fatal in infancy because the pathology and associated anomalies are not compatible with life. Better prognosis in the adult type is due to the position of the constriction, forcing the establishment of the collateral circulation during early fetal life at a time when it can be most readily established, so with Ballantyne in 1935.

The physical signs bear no constant relation to the symptoms but may present when the latter are lacking. Perhaps in no pathologic condition are more

extensive changes compatible with fewer evidences of disease during life.

To summarize-- prognosis depends upon the degree of stenosis of the isthmus and the adiquacy of the collateral circulation.

TREATMENT

Recently, interest has boomed among surgeons to study the possibilities of surgery to alleviate this condition. Up to date no surgery has been attempted, undoubtedly due to the fact of the rarity of the anomaly and the condition being incorrectly diagnosed as essential hypertension. This new interest is due to the successful ligature of a case of patent ductus arteriosus.

Unfortunately very little can be done in the way of treatment. Activities and exercise should be limited and owing to risk of sudden death. Responsible occupations, such as that of a bus driver should not be allowed to the affected person.

On account of the danger of infective endocarditis all foci of infection should be dealt with. In this respect, as in mitral stenosis wholesale extraction

of septic teeth should not be carried out, owing to the risk of flooding the circulation with pathogenic organisms. In a case of coarctation this organism may set up an infective focus at the site of constriction or at the aortic valves, especially if a bicuspid condition be present.

Light work to keep the patient's mind occupied should be encouraged. Appropriate treatment for circulatory failure, the earliest signs and symptoms of which are to be looked for in the legs, once any other complication should be instituted as soon as they manifest themselves so wrote Sangster in 1937.

The main principle is to live within the cardiac tolerance and by appropriate means to aid the circulation in the lower part of the body with relief of circulatory stress. S. Amberg (1932) deduced this result from the observation of persistent treatment with diathermy the pulse in the lower extremity became palpable and the blood pressure increased so that it could be determined by ausculatory method.

CONCLUSIONS

(1) Predelection for this congenital abnormality is for the male sex, three to one.

(2) Coarctation occurs once in about 1550 cases at autopsy.

(3) Coarctation is classified into two types, "infantile" and "adult". "Infantile" type is the congenital narrowing of the isthmus of the aorta proximal to the insertion of the ductus arteriosus. "Adult" type is the abrupt, ligature-like constriction at, just proximal, or distal to the insertion of the ductus arteriosus.

(4) Etiology is still a matter of conjecture but it is believed that adult coarctation, narrowing or obliteration of the aorta is due to absence, atrophy or imperfect development of left fourth branchial arch.

(5) Pathology of adult type is a sharp constriction amounting in some cases to complete obliteration. Due to external strangulation there is narrowing of the lumen. Frequently then is a fold, septum or a diaphragm which stress across the lumen.

(6) Infantile type is a diffuse narrowing of the isthmus of the aorta, this segment may be represented by a fibrous cord, or be altogether lacking. This type is incompatible with life.

(7) Associated anomalies are more commonly combined with infantile type. Aortic bicuspid valve

frequently found with the adult type.

(8) Symptoms are divided into three groups: (a) those in whom symptoms are absent (b) those in whom symptoms are late in developing (c) those in whom symptoms are present throughout life. Symptoms are usually those of myocardial insuffiency.

(9) Characteristic signs of coarctation when present are vascular rather than cardiac, the effects of the gradual extending collateral circulation.

(10) Hypertension in the upper extremities in the best single lead in arriving at a diagnosis.

(11) Diagnosis may be difficult because the collateral circulation follows a relatively direct route through widely dilated vessels.

Palpable collateral circulation is a direct lead to diagnosis.

(12) 75% of cases show hypertrophied hearts.

(13) The femoral, popliteal pulse is diminished or absent. Radial pulse begins .03 sec. before the femoral.

(14) There are five diagnostic roentgenological findings: (a) Absence of aortic knob in postero-anterior view, (b) Defect in the aortic arch in left antero-posterior oblique view. (c) Rib erosion

(d) Dilated ascending aorta, (e) Left ventricular hypertrophy. (b) and (c) are pathognomonic.

(15) The majority of cases die from failing compensation.

(16) Highest mortality is in the first year of life, second, third and fourth decade.

(17) Prognosis depends upon the degree of stenosis ofthe thoracic aorta and the efficiency of collateral circulation.

(18) Treatment is palliative.

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