Coarctation of the aorta: with report of three cases

Leonard James Chadek
University of Nebraska Medical Center

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COARCTATION of the AORTA

with

REPORT of THREE CASES

LEONARD J. CHADEK

SENIOR THESIS PRESENTED

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INTRODUCTION

The word coarctation is derived from the Latin "coarctio" from "cum" (together) plus "arctare" (to make tight). Dorland (1940) defines it as a straightening or pressing together. A condition of stricture or contraction.

Coarctation of the aorta is synonymous with congenital stenosis of the aortic arch. The degree of stenosis occurring at the isthmus may vary from moderate stenosis to complete atresia (Brotchner 1939). The isthmus is the part of the aortic arch which lies between the left subclavian artery and the insertion of the ductus arteriosus or ligamentum arteriosus.
Altogether too frequently this condition is not diagnosed clinically, the diagnosis being made for the most part during post-mortem examination. It is most often diagnosed as hypertension or a nephritic condition. As Abbott (1928) pointed out in her review of 200 cases "only 19 were diagnosed clinically". It will be pointed out that sometimes the symptoms are vague so as to make the clinical diagnosis difficult. However, the average practitioner should be on the alert for this condition and in cases of hypertension, particularly in young adults, should palpate
the femoral artery for pulsations and take the Blood Pressure reading in the lower extremities for evidence of disparity.

It will be the purpose of this paper to present a review of the literature on coarctation of the aorta with emphasis on the important signs, symptoms and diagnostic points both clinical and radiological in an effort to aid in clinical diagnosis.

HISTORY

This condition was first described as early as 1789 by Paris but by some manner or another was credited to Morgagni in 1760. Meckel and Weirnich were among the earliest writers on the subject. Legrand in 1835 on the basis of collateral circulation and decreased pulse distal to the obstruction was first to diagnose the condition. Reynaud wrote on it in 1828 suggesting its possible etiology, followed by Craigie who promulgated the Skodian theory in 1841. Craigie, Eppinger, Kreigh, Barie, and Bonnet (1903) supplied Maude Abbott 101 cases, 35 cases she abstracted herself, and from King, Blackford, Mexner and others she made a total of 183 cases. Then Blackford reviewed 17 more cases giving her a
total of 200 cases which she studied in retrospect to give the finest report of this anomaly to date.

AGE OF DEATH, INCIDENCE AND SEX

The only statistics of any extent in regards to the age of death, sex, and incidence was compiled by Maude Abbott (1928) in her review of two hundred cases.

<table>
<thead>
<tr>
<th>Age at Death</th>
<th>Total Cases</th>
</tr>
</thead>
<tbody>
<tr>
<td>3-10 years</td>
<td>9</td>
</tr>
<tr>
<td>10-20</td>
<td>45</td>
</tr>
<tr>
<td>20-30</td>
<td>49</td>
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<tr>
<td>30-40</td>
<td>45</td>
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<tr>
<td>40-50</td>
<td>28</td>
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<td>50-60</td>
<td>12</td>
</tr>
<tr>
<td>60-70</td>
<td>10</td>
</tr>
<tr>
<td>70-92</td>
<td>2</td>
</tr>
</tbody>
</table>

From this it is easily seen that the lesion threatens individuals in the prime of youth or early part of middle age. This is the period of greatest economic and physical activity.

Insofar as sex is concerned, Evans (1933) and Abbott disagree. Evans states that this anomaly occurs with nearly equal frequency in males and females whereas Abbott found in a review of 195 cases there was a
disproportion of one female to every three males or a total of 147 males to 48 females. This phenomena cannot be explained but the disparity seems too great to be accidental.

There is a variance in the opinion of writers as to the incidence. It is a comparatively rare condition. Blackford (1928) states that it occurs in one out of every 1550 cases. Tawcett, an analyzing routine post-mortem at Guy's Hospital (1826-1902), could only find eighteen cases out of 22,316, an incidence of one in 1200. Evans, on the other hand, found 26 cases in 19,217 routine autopsies performed at the London Hospital the incidence being about one in 740 cases.

Abbott when she collected her data on 237 cases in 1928 believed these to be the full number of all cases recorded in history to that time. It would seem that this an extremely rare condition but it occurs often enough for practitioners to familiarize themselves with this condition.

TYPES OF COARCTATION

Bonnet in 1903 classified coarctation into two types, the infantile and adult type. These he differentiated according to the site and form of atresia.
The infantile type he described as a diffuse narrowing of the aortic isthmus with the circulation impaired so that the patent ductus arteriosus carries the blood to the lower extremities. This type is not compatible with life, due to its association with an anomaly so severe that there is early termination.

The adult type differs in that rather than a diffuse narrowing there is a marked constriction in the aorta which may amount to complete atresia. The site is usually at the point where the ductus arteriosus is inserted on the aorta or adjacent to it on the caudal side.

Evans (1933) differs with Bonnet (1903) and adopts a different classification. He divides it into six types differentiated by:

1. Site, nature and extent of constriction.
2. Condition of aorta proximal to constriction.
3. Potency or closure of ductus arteriosus.
4. Relationship between systemic and pulmonary circulation.
The types defined are:

1. "Congenital stenosis of the aorta 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."

Types 2 and 3 being more or less compatible with longer life.

Blumenthal and Davis (1941) advocate a different classification, one on a physiological basis rather than anatomical. They suggest the nomenclature "compensating coarctation" for the adult type and "now compensating coarctation" for the infantile type.

They theorize that in the adult type, the patient survives because the development of the collateral circulation is sufficiently extensive to compensate for the defect in the aorta. It is on this basis that they developed their terminology.
THEORIES OF CAUSATION

The theories of causation is probably the most controversial point in the study of this condition. The controversy does not exist as far as the infantile type is concerned, most writers being in agreement that this is a prenatal development. Brown (1943) states that the infantile type unquestionably is of developmental origin and that it has been commonly observed before birth. This then will be a presentation of the opinions of the etiology of the adult type. The first theory of causation put forth was that of Craigie in 1841, and in subsequent years Skoda (1855), Brunner (1888), and Bonnet (1903) gave support to it. This theory was called the Skodian theory and believed the coarctation was developed postnatally. This in effect, that the tissue of the duct wall is continued into the wall of the aorta and that when the lumen of that duct is obliterated in the first six to eight weeks, the ductus arteriosus in contracting due to it's peculiar tissue, causes mechanical traction on the wall of the aorta causing a kinking or constriction.

Blackford (1928) and Brown (1943) disagree with this theory because they do not believe that such tissue
has ever been satisfactorily demonstrated in the aorta. If it has, an explanation is still lacking for the cases in which there is a patent ductus arteriosus (Brown 1943). His belief is that the site corresponds to the portion of the junction of the fourth and sixth left branchial arches. The sixth arch forms the ductus and it is thought that delay in the involution of these arches and in part, absence of, or imperfect development of the fourth arch together with traction exerted by the obliterating ductus upon these embryonic structures, may be the cause of the occlusive process of the aorta. Assuming the presence of a severe anomaly of the fourth arch in utero a collateral circulation would then develop and be unlikely to cause any great disturbance.

Blumenthal and Davis (1941) believe both types to be congenital instead of the infantile type alone and the adult type developing in extra-uterine life.

Blackford (1928) agrees with Reynaud (1828) that this condition is intra-uterine but disagrees with him on the theory of the influence of the involution of the ductus arteriosus.

Evans (1928) does not promulgate a theory of his own but challenges the Skodiac theory with the following points:
1. In most adult cases the coarctation occurs at some little distance from the site of insertion of the ductus arteriosus.

2. The ductus is patent in some cases.

3. There are other associated anomalies of the cardiovascular system.

4. There have been no cases reported in which the pulmonary artery was constricted.

Ikeda comes forth with some original research by Friedberg (1942) in an effort to establish a definite theory for causation. His work consisted of injecting radio opaque material into the circulatory system of sheep fetus.

In the fetus, the aortic arch is divided into an upper and lower segment by the isthmus, that short segment between the left subclavian artery and the ductus arteriosus. Direct observation of the radio opaque material injected into the inferior vena cava of sheep's fetus reveal the material to pass directly through the foramen ovale into the left heart and aorta, thence into the carotid and subclavian. Little blood was seen to pass through the isthmus into the lower segment. By the same method, most of the material injected into the superior vena cava pass through the right heart, pulmonary artery and ductus arteriosus into the lower segment.
His theory then presupposes a hypoplasia of the aorta or more rarely a constricted area or anomalous vessel in the upper aortic segment as the basic congenital anomaly in coarctation of the aorta at birth, the drop in pressure in the ductus as the pulmonary arterial blood pass into the pulmonary arteries, causes a closure of the ductus through the contraction of it's strong circular muscle fibers. Hypoplasia of the aorta lowers the pressure in the isthmus even more than in the normal and coarctation develops the same way. This theory then explains the fact that the adult type of coarctation of the aorta must occur after birth and is never found in inter-uterine life. It also accounts for the location of the constriction.

Except for the latter theory which is based on original research, we might say that the theories of causation of coarctation of the aorta are largely a matter of conjecture.

ASSOCIATED ABNORMALITIES

The presence or absence of other anomalies occurring with the coarctation are of great interest. Bonnet (cited by Abbott 1928) believes this is an important differential point in that the more serious and complex
anomalies such as bioculate and trioculate heart, transposition of arterial trunks, pulmonary atresia, etc., are very commonly associated with the infantile type but never found in extreme degrees in the adult type. In the adult type only minor defects may be found or they may be absent. One of the most common abnormalities in the adult form is that of bicuspid aortic valve (Sangster (1939), Abbott (1928), Amberg (1932), Weinberg (1938), and Brown (1943). Others not uncommonly found are hypoplasia of the aorta, dilatation of the ascending aorta, congenital weakness of the arterial media, stenosis of Rt. Subclavian artery (Love and Holmes 1939), anomalous origin of the arteries from the arch (Moraques, Moore, Rossen 1942) sub aortic stenosis, defects of aortic septum and an uncommon finding is a patent ductus arteriosus.

Abbott (1928) found that in 155 cases there were 57 cases in which the coarctation was complicated by some associated minor anomaly and in 86 cases the coarctation was the only defect. These anomalies form speculation as to the possibility of developmental arrest as the chief causative factor in the adult as well as in the infantile form.
It must be understood that these complications occur as the cause of death and will be treated as such.

There are various and sundry complications that are found with coarctation. These complications frequently leading to and being the cause of death. Narr and Johnson (1934) found 14 cases of endocarditis, 13 cases of which were at the site of the coarctation. They attributed thus to the dilatation and atheroma of the aorta proximal to the stenosis and to the kinking and deformity which provides a favorable nidus for lodgment of the bacteria. Strang reported 3 cases, 2 of which were complicated by intercurrent infection. Brown (1943) found infective endocarditis which showed to be streptococcus viridans on blood culture.

Myocardial failure is a main cause of death occurring in about 15% of the cases in Lewis (1931) opinion. Atheromatous changes are commonly found in the dilated ascending aorta. Rupture of the aorta or dissection aneurysm are other common complications. Zazlow, and Krasnoff (1943) reported a case in which the aneurysm was distal to the obstruction. There were only two
other cases reported up to 1942. Lichtenberg and Gallagher (1933) state that intercranial hemorrhage was the cause for 10% of all deaths. Strong (1932) reported spontaneous cerebral hemorrhage as a cause of death with confirmation by Amberg (1932).

THE COLLATERAL CIRCULATION

In regards to the collateral circulation, it would be interesting to know whether or not the circulatory efficiency in different cases is related to the anastomotic pattern. Irvine (1942) believes the extent of this collateral circulation is governed by the degree of stenosis present in the aorta. If this is true then those cases with a complete atresia must present an increased degree of efficiency in the collateral compensatory mechanism to sustain life.

It would seem from a purely mechanical standpoint that the greater the obstruction, the greater the collateral circulation and with a lesser degree of obstruction a lesser collateral extent. It cannot be denied however, that there is a relationship hitherto unexplained that lies between the two.

Since there has been but little work done on the
pathways of collateral circulation and due to the lack of uniformity in the collateral circulation pathways, this will only show the extent to which it takes place in one case and will not be dealt with controversially.

In 1941 Bramwell and Jones admitted to a London hospital a patient in whom the lesion was diagnosed in 1938. She was admitted suffering from a subarachnoid hemorrhage from which she died. This accident gave them an opportunity of studying the collateral circulation in a patient who exhibited no signs of heart failure.

The following tabulation is the various collateral channels they thought of importance. This is theoretical because the blood has a choice of routes whereby to reach the aorta beyond the coarctation.

1. The Scapular and Cervical Anastomosis.
   A network around the scapula and the cervical region.
   (a) Suprascapular and transverse colli (from the thyroid axis).
   (b) Postacapular and superficial cervical (from the transverse colli).
   (c) Long thoracic and subscapular with its dorsalis and scapular branch (from the axillary artery).

From this network descending branches anastomose with the lateral and dorsal branches of the aortic intercostals.
2. The Internal Mammary Anastomosis.
   (a) Superior epigastric to the deep epigastric branch of the external iliac.
   (b) Musculophrenic to the phrenic branch of the aortic intercostals.
   (c) Mediastinal branches to the mediastinal branches of the aorta.
   (d) Anterior intercostalis to the terminal branches of the aortic intercostals.

3. The Intercostal Anastomosis.
   (a) The terminal branch to the intercostal branch of the internal mammary.
   (b) Lateral branches to the subscapular artery and the long thoracic artery.
   (c) First and second intercostal artery (arising from the subclavian by the superior intercostal artery) to the upper aortic intercostal artery.
   (d) Dorsal branch to the posterior scapular artery.
   (e) Each intercostal to those above and below it.

4. The Spinal Anastomosis.

   The vertebral artery which arises from the first part of the subclavian reinforces the spinal arteries in which the blood flows downward to reach the spinal branch of the aortic intercostals. These pass through each intervertebral foramen. There are also branches from the inferior thyroid which pass through the intervertebral foramina in the neck to join the spinal arteries.

   This is the report of the above case in which the patient at post-mortem was injected with barium enema paste in an effort to study the anastomosis of the
collateral circulation. From this study, while it is only a single case, the extensive collateral circulation that is set up gives one an idea why there are no diagnostic clinical manifestations until such time that there is a secondary cardiac embarrassment caused by the individual.

A cannula was introduced in the left common carotid artery and the opaque paste injected proximal to the coarctation. This was done under radioscopic control in order to insure sufficient material to enter the arteries to outline them satisfactorily without obscuring the larger vessels by filling the arterioles and capillaries. Approximately 700-800 cc. of barium enema paste was used.

From their case study, they found this circulation established to compensate for the stenosis of the aorta:

(a) The scapular and the cervical branches of the subclavian and axillary arteries anastomose with the lateral and dorsal branches of the aortic intercostals forming a network around the scapula and in the neck.

(b) Anastomosis by the musculophrenic branch of the internal mammary to the inferior phrenic branch of the abdominal aorta follow forming a network above and below the diaphragm.

(c) The anastomosis that extends from the superior
intercostals to the upper two intercostals to the upper aortic intercostals, is the one that produces the notching of the ribs.

Their findings agreed with some of the theoretical patterns. They found that the importance of the anastomosis between the superior and deep epigastric arteries which has been stressed in some accounts was in their case insignificant. Likewise the anastomosis between the anterior intercostals (arising from the internal mammary) and the terminal branches of the aortic intercostals was poorly developed, the great enlargement of the internal mammary being due chiefly to the extensive diaphragmatic anastomosis.
This diagram illustrates the available collateral channels. All of these are not necessarily enlarged in any particular case.
FIG. 8.—Tracing from Fig. 7.
CA—Injection Canula.

ARTERIES.
AA—Ascending aorta.
IN—Innominate.
SC—Subclavian.
LT—Long thoracic.
DS—Dorsalis pedis.
LA—Anastomosis of subscapular with aortic intercostals.
, 6, etc.—Aortic intercostals. 
ending aorta.

CC—Common carotid.
IM—Internal mammary.
SS—Subscapular.
M.Ph.—Musculo-phrenic forming diaphragmatic anastomoses.
CO—Communicating branches between intercostals.
XX—The points at which the second aortic intercostals (supplying the fifth rib) notch the ribs.
The preceding diagram is a tracing from a radiogram following the injection of Barium paste.

The intercostal arteries have filled and are very tortuous. The notching of the ribs by the arterial loops is obvious. The internal mammary arteries are enlarged and the extensive diaphragmatic anastomosis formed by their musculophrenic branches and the phrenic branches of the aorta is well seen on the left side. The tortuous enlarged subscapular artery is visible on the right border of the chest; its dorealis scapulæ branch encircles the neck of the scapula. Just medial to its origin is the long thoracic artery. The aortic intercostales supplying the fourth and fifth spaces are much enlarged near their origin from the aorta. This appearance is not a filling artefact, for all the barium was injected above the coarctation. The intercostals, therefore, filled by their collateral channels from the scapular network and the superior intercostal, and the distension near the descending aorta could not have been due to forcing barium into them from the aorta distal to the coarctation.
SIGNS

Physical signs are of more importance in the diagnosis of this condition than are the symptoms (Sangster 1939). Some of the more prominent signs will be herewith described. As would be expected from the high blood pressure in the upper body, the face may appear flushed, this is often a common occurrence. This is explained by reflex from the carotid sinus (Lewis 1933). He believes this hypertension in the head and neck acts as a stimuli to the carotid sinus, this reflex producing a loss of arterial tone and vasodilation. This may also account for the warmth and sweating of the upper extremities.

In the absence of a diastolic murmur one cannot overlook, on examination of the neck, the importance of carotid pulsation.

Blood pressure readings are important signs. The readings in the upper extremities showing a marked hyperiesis where as the readings in the lower extremities are considerably lower or not obtainable. By contrasting the radial and femoral pulses, it is found that the radial pulse is full and hard in contrast to the femoral pulse which may be barely palpable, retarded,
or even imperceptible. Abbott (1928) attaches so much significance to this sign with dilated pulsating vessels on arterial murmurs in the upper body, that she believes it to be pathognomonic of coarctation.

The presence or absence or diminution of the femoral pulse bears no relation to the degree of stenosis. It may be perceptible in cases of complete atresia and absent in cases with mild stenosis, (Abbott 1928). Bonnet (1903) explained this on the basis that the sensation imparted to the palpat ing finger from the pulsating vessel is not dependant on the amount of blood in the vessel but on the more or less sharp repercussion of the wave that passes along the artery as it is propelled from the aorta. So that when the blood reaches the aorta or artery through a large series of collaterals, the ascent will be more gradual and the pulse below it correspondingly weaker.
Sphygmographic tracings from the left brachial, right brachial, and femoral arteries. Note difference in form and amplitude of the brachial and femoral tracings.

SYMPTOMS

Dependant on individual cases the symptoms may be absent and the patient enjoy a vigorous and healthy life and may be by chance picked up at routine examination. The subjects are usually robust, rather plethoric looking, athletic young individuals with good musculature and intelligence often above average (Abbott 1928).

The patient may present himself for evaluation of symptoms that resemble ordinary cases of hypertension. Dyspnea on exertion, precordial pain, headache, vertigo,
throbbing in the neck and head and fatigue (Ash 1941). Eppinger and Middlefart (1933) say that in the adult form there are no symptoms referable to the cardiovascular system in children or youth. Blue (1943) substantiates this with his report of a case of a 14 year old athlete who led a vigorous life being active in football, basketball, and swimming. He had no symptoms or complaints. His condition was picked up when he applied for life insurance but not diagnosed.

Gross (1945), however, reported that one of his operative cases, the 5 year old boy, had frequent epistaxis for four years, but on the other hand his second operative patient, a 12 year old girl, had no symptoms referable to the cardiovascular system.

DIAGNOSIS

In 1933 Lichtenberg and Gallagher found that in 250 cases reviewed only about 25% was diagnosed clinically and confirmed at autopsy. In 50 other cases the diagnosis was made during life with some fair degree of certainty.

With these figures in mind, an attempt will be made to present the major clinical and radiological findings so that diagnosis may be made more readily during life.
Bramwell and Jones (1941) in a single case made an effort post-mortem to show that the extensive collateral circulation that is set up is the cause for the lack of diagnostic clinical manifestations until such time that there is secondary complications or cardiac embarrassment caused by an increase of work by the individual. The results of this experiment will be discussed elsewhere in this paper.

1. Radiological.

Perelman in 1944 presented a review of 13 cases in which the following radiological signs were found and which he considers to be pathognomonic:

1. Aortic Knob absent in all cases.
2. Aortic dilatation
   (a) None--3 cases
   (b) Slight--5 cases
   (c) Moderate--5 cases
3. Rib Erosion
   (a) Slight--5 cases
   (b) Moderate--7 cases
   (c) Morbid--1 case
4. Increased Left Ventricular diameter
   (a) None--4 cases
   (b) Slight--5 cases
   (c) Moderate--3 cases
   (d) Morbid--1 case

Wilson (1932) agrees with Perelman (1944) but offers another finding, a break in continuity of the aorta with a defect in the arch. This view is substantiated by Purks and Roberts (1935) but they add
that the view should be taken in the left posterior-anterior oblique view and state a segment of 1-3 cms. in the descending arch cannot be seen. Ash (1941), however, disagrees and states that the radiological diagnosis shows only two positive signs. (1) An erosion of the ribs from the 4th to the 9th ribs along the inferior aspect and (2) narrowing or complete absence of a portion of the aorta in the region of the isthmus. He then presented the following radiological signs as a suggestive diagnosis (1) absence of an aortic knob (2) left ventricular hypertrophy (3) widening of ascending aorta and (4) increased width in the shadows of the great vessels to the neck.

Gladnikoff (1946) does not believe that changes on the ribs occur in every case and that they are not pathognomonic because the same changes have been seen in diseases of the aortic and mitral valves.

In cases where no rib defects show up roentgenologically, he believes the diagnosis can be made and is pathognomonic by the following signs:

1. Dilatation of left subclavian artery along with change of position of the aortic arch and descending aorta toward the media stinum.

2. A consequent reduced possibility to see the parts of the aorta.
2. Clinical Manifestations.

The most common diagnostic mistake is to label this condition essential hypertension. Coarctation should always be thought of and eliminated in essential hypertension especially in young adults. In these cases where hypertension is present it should not be too difficult for the practitioner to accustom himself to palpate both the radial and femoral pulses simultaneously to detect any difference in pulse waves.

When any case of coarctation is suspected collateral circulation should be searched for both clinically and radiologically. Clinically these may be palpated as thrills or heard as bruits.

One of the most important diagnostic signs is the disparity of blood pressure in the upper and lower extremities. The pressure in the upper extremities may vary from 170 mm. Hg. to 290 mm. Hg. in systole and from complete absence to 110 mm. Hg. in systole in the lower extremities. There is little or no palpable femoral, popliteal or dorsalis pedis pulse. With this decreased blood flow to the lower extremities, there is a complaint of coldness of the lower extremities with a feeling of warmth or even perspiration of the upper portion of the body. Adding to this is the evid-
ence of collateral circulation obtained when pulsations are seen or felt in places where none usually occur. These may occur in the neck or thorax. Bruits are often heard and thrills felt over such vessels. Systolic murmurs are often heard over the precordium, the upper thorax and characteristically in the interscapular region (Amberg 1932).

Since difference in blood pressure in the extremities may be the only sign, it is important that the practitioner think of this condition and be on the lookout for it.

TREATMENT

Until 1944 the only treatment for coarctation of the aorta was palliative, being much the same therapy as encountered in vascular hypertension. This consisted of avoidance of responsible occupations (owing to the risk of sudden death), protection against physical and mental strain (Blue 1941). Foci of infection had to be watched for due to the possibility of some pathogenic organism setting up at the site of coarctation or at the aortic valves.

In 1944, Crafoord and Nylin in Sweden and in 1945
Gross and Hufnagel in the United States considered the possibilities of surgical correction of coarctation.

Crafoord in 1935-36 found that on experiments on dogs he was able to ligate the aorta for as long as thirty minutes without any signs of organic damage, provided an adequate blood supply was maintained to the brain. On the basis of these observations he ligated the aorta on certain patients with patent ductus arteriosus, above and below the duct and kept it ligated until the duct was ligated and the aorta sutured. With this in mind he considered the possibilities of using this method to treat coarctation of the aorta surgically.

When two patients came into the Sabbotsberg Hospital, Crafoord and Nylin with the grave prognosis in mind thought it justifiable to attempt surgical intervention.

It must be understood at this point that only the adult type is of interest insofar as surgical correction is concerned.

Case I concerned a twelve year old male whose systolic pressure varied from 140-190 mm. Hg. since 1942. His heart was not enlarged, but there was a loud blowing systolic murmur heard loudest in the second
interspace. On pre-operative examination his blood pressure was 170/105 in his arms and 100/70 in his legs. Oscillography showed increased blood pressure in his arms and good oscillations. In his legs the pulsations were very small and the blood pressure below 100 mm. Hg. Post-operatively both systolic and diastolic blood pressures increased in the legs and decreased in the arms.

Case II had to do with a 27 year old male, who, early in 1944 had a blood pressure in his arm of 210/110 mm. Hg. His heart was enlarged with a loud systolic murmur in the second intercostal space near the sternum. The blood pressure in his legs was below 100 mm. Hg. systolic with no pulsations. On pre-operative oscillography there showed an increase in blood pressure in the arms and good oscillations. The leg pulsations were small and showed a blood pressure of below 100 mm. Hg. in systole. Post operatively both the systolic and diastolic blood pressures was increased in the legs and correspondingly decreased in the arms.

Gross (1945) felt as Nylin and Crafoord (1945) did, that with the variable prognosis he would be justified now to attempt a similar procedure on human
beings.

Within the period of two weeks in the summer of 1945, Gross had the extremely good luck to have two patients with coarctation admitted to the Children's Hospital in Boston, Mass. The first patient a five year old male, was admitted on June 28 for excision of the anomalous portion of the aorta. This segment was excised and the remaining ends of the aorta anastomosed by continuous mattress-type suture which included all layers of the aorta. When the aorta clamps were released, there was satisfactory hemostasis at the suture line but the quick release of the blood into the great vascular bed of the lower part of the body imposed an enormous burden on the heart which dilated rapidly and the patient died, all efforts at resuscitation having failed.

Case II was a twelve year old female operated on July 6, 1945. Her pre-operative blood pressure in the upper extremities was 190/100 mm. Hg. and none obtainable in the lower extremities. Cyclopropane was the anesthesia of choice. A cannula was inserted into the ankle vein to administer fluids and blood during and post-operatively.
A long curved incision was made beginning just to the left of the spine, starting at the level of the second rib and running downward and outward to reach the posterior axillary line at about the level of the inferior tip of the scapula. The muscles divided and the 3, 4, 5, & 6th ribs were transected at their angles. The aorta was exposed showing a marked constriction of the descending aorta about 1.5 cm. below the origin of the left subclavian artery, the external diameter being no more than 3 or 4 mm. in diameter. Palpation of the constriction showed it to be a firm fibrous cord with little or no lumen. The aorta above the constriction was 15 mm. in diameter and showed a marked heaving pulsation which was transmitted into the dilated left subclavian artery. Below, the constriction was 13-14 mm. in diameter and had no visible or palpable pulsation. Care was taken to avoid injury to the vagus nerve, left recurrent nerve and the thoracic duct. The aorta was clamped just above and below the constricted zone. Application of these clamps (specially designed) produced absolutely no changes in the heart rate or activity. A segment of aorta, 1 cm. long, was excised so that the constricted portion was completely excised and the ends then reunited as in Case I. The lower clamp
was then removed and as blood flowed back from the lower segment up into the anastomosis there was no bleeding at the suture line. With the experience of Case I, great care was exercised in three ways to ensure that there would be no abrupt shift of the circulating blood which might embarrass the heart. First, the remaining aortic clamp was released very slowly over a period of 10 minutes so that the lumen of the aorta was opened very gradually. This was done in order to avoid a great rush of blood into the lower part of the body. Second, the table was tilted to place the patient in a mild Trendelenberg position to diminish the pooling of blood in the leg and lower part of the body and to increase the return of venous blood to the heart. Third, donor blood was pumped into the ankle vein to insure an adequate blood volume for the heart to circulate. The distal aorta now showed a visible and palpable pulsation. The wound was closed and healed per primam.

In the 19 post operative days there was a gradual rise in the blood pressure in the lower extremities. This gradual rise was probably due to a smaller vascular bed distal to the constriction and the time was required to dilate it. There was also a simultaneous
diminution of the blood pressure in the arms. At discharge the blood pressure in the upper extremities read 140/80 mm. Hg. and 145/105 in the lower extremities.

In view of the fact that only about 25% of all cases are diagnosed clinically this procedure would hardly alter the grave prognosis.

PROGNOSIS

Of the two types of coarctation of the aorta, the infantile type is 100% fatal, the patient dying in infancy (Blalock 1928 and Sangster 1937). Blue (1943) states that they always die before nine months. The prognosis in the adult type is varied (Gross 1945), patient may live to a ripe old age even with a life of a heavy manual laborer. In others the prognosis is poor enough to justify attempts at surgical correction (Nylin and Crafoord 1944). Reynaud (cited by Blackford 1928) reported a patient who lived a vigorous life and died at 92. As Maude Abbott (1928) stated in her classical review of 200 cases "there is no other pathological condition compatible with life with fewer evidences during life." Among these 200 cases re-
viewed she also found that 148 (74%) died before or during the 40th year of life. In all of these 200 cases 77% of the deaths was attributed to coarctation or a complicating cardiac lesion.

So on a basis of these figures and on single cases reported by various authors (Moragues (1940), Taylor (1943), Grishein (1947), the prognosis is extremely grave.
CLINICAL HISTORY

This is the report of C. F., a 25 year old colored male, who entered the Tulane University Hospital on April 11, 1943. He died May 7, 1943.

Chief Complaint.

High fever and heart trouble.

Present Illness.

The patient was playing football about six years ago and had a routine physical examination and was told that he had a bad heart. At this time he had no symptoms and felt well. About one month ago he began to have fever in the afternoons, accompanied with pains in the knees and arms. Right after the fever he became very nervous. He had a cough and spit up some heavy sputum. He had lost his appetite since the fever began but had not been constipated. He had lost about 40 pounds in weight in the past two months. He perspired profusely and seemed to be thirsty all the time.

Past History.

Negative.

Social History.

Negative.
Family and Marital History.

Negative.

Physical Examination.

Temperature 100.8°F. Pulse 105. Respiration 25.

Blood Pressure: Left arm 180/65, right arm 182/65, left leg, 125/100. The patient was a 25 year old, well developed and well nourished, colored male, who was lying in bed. He was extremely nervous and appeared to be acutely ill and was very apprehensive. Eyes, ears, and nose were negative. Mouth and throat normal. There was an abnormal arterial pulsation bilaterally in the neck. The lungs were crepitant and there were a few moist rales over both lung fields posteriorly on deep respiration. There was an enlargement of the heart to the left and downward. The Point of Maximum Intensity was in the sixth interspace outside the midclavicular line and somewhat diffuse. There was a palpable diastolic thrill over the apex of the heart. There was a friction rub heard best over the apex of the heart in the anterior axillary line in the fifth interspace. Superimposed on the friction rub was a rumbling diastolic murmur heard best at the mitral area. There was a loud, harsh, systolic murmur over the aortic area which was transmitted into the neck, and a short diastolic murmur of less intensity over the same area. The abdomen was
negative. The upper extremities revealed a water-hammer pulse. There was a very coarse tremor to the fingers. Femoral pulsations could not be felt.

Laboratory Findings.

On April 12, 1943, hemoglobin was 8.7 Grams, R.B.C. 3,200,000, W.B.C. 15,700. Subsequent examination showed no marked change.

Urine showed a 2+ albumin. Alpha streptococcus was isolated in blood cultures on four occasions. X-Ray, April 12, 1943, showed marked enlargement of the cardiac shadow with generalized enlargement of all chambers of the heart. The aortic knob could not be well seen. There was suggestive evidence of notching of the ribs. Right and left oblique views of the chest shows the enlargement of the cardiac shadow. The arch of the aorta could not be definitely made out. The right anterior oblique view showed the usual clear space between the transverse aorta and spine obscured. General Impression: Marked cardiac enlargement mainly in left ventricle; possible anomaly of the course of the aorta in that it may pass to the left instead of the right.

Clinical Course.

Blood Pressure in left arm 180/70, in the left popliteal space 125/10.
On April 21, 1943, the patient showed marked dyspnea. Pitting edema of both ankles was noted. His condition was afebrile and satisfactory until May 7, 1943 at which time his respiration ceased very suddenly. He was pronounced dead.

POST-MORTEM REPORT

Pericardial Cavity.

The cavity is obliterated. There are marked adhesions present throughout the entire cavity.

Heart: (After fixation).

The entire pericardial cavity is obliterated as the result of fibrous adhesions. The combined thickness of the visceral and the parietal pericardium is about 2 - 3 mm. The heart weighs 1345 Grams. This weight includes the parietal pericardium but all of the excess fat is removed from this structure. The great vessels leave and enter the heart in a normal fashion. The chambers of the heart are open. The tricuspid and the pulmonary valves are normally developed and show no thickening. The mitral valve is composed of two cusps in the normal position, but several of the chordae tendineae are slightly thickened, measuring as much as
1.5 mm. in diameter in their central portion. The aortic valve is deformed, as a result of congenital malformation and acquired disease. It consists of two cusps, a posterior cusp whose transverse diameter at the superior margin is 4 cm. The right anterior and the left anterior cusps are represented by a single cusp which likewise measures about 4 cm. in diameter. This cusp is divided into two almost equal halves by a medial raphe in the base of the cusp and a cord-like structure which begins at approximately the point of a normal commissure and inserts into the sinus aside of the cusp at a point about 2 mm. inferior to the superior margin. There is an aperture between this cord-like structure and the raphe which measures approximately 0.7 cm. in diameter. The posterior cusp presents an ulcerated area at its inferior margin which measures 2.5 cm. in its transverse diameter and 1.5 cm. in its supero-inferior diameter. The margins of this ulcerated area are elevated, the superior lip protruding for a distance of 0.7 cm. from the valve and the inferior lip for a distance of 0.2 cm. from the surface of the valve. In the center of the ulcerated area there is an ovoid aperture which measures 2 cm. in transverse diameter and 0.6 cm. in supero-inferior diameter. This aperture communicates
with a cavitation which extends superiorly for a distance of about 4.5 cm. between the system and the pulmonary aorta. At the mouth of the ulceration this cavitation measures about 3 cm. in its transverse diameter. It is estimated that the total volume of this cavitation is 7 cc. by measurement. The posterior cusp, which is not involved in this ulcerative process, shows slight diffuse scarring. The sinus of Valsalva is intact except for a small area measuring about 2 mm. in diameter at the superior margin of the ulcerated area previously described. Here there is an aperture which communicates with the large ulceration which actually has eroded into the tissues at a point immediately inferior to the most inferior portion of the valve. The other cusp is a deformed cusp. Its origin at the commissure is about 0.5 cm. inferior to the posterior cusp on either side, so that the supero-inferior measurement from the superior margin of the cusp to the bottom of the sinus of Valsalva is approximately 1 cm. as compared to the posterior cusp which measures 1.5 cm. The cusp also is scarred to the extent that it resists movement but is not rigid. Both coronaries arise above this cusp, one on either side of the raphe which has been previously described. What is evidently the left coronary arises directly above and to the left of the
commissure of the posterior cusp. The stoma of this left coronary measures 1 cm. in transverse diameter but rapidly narrows to about 0.6 cm. in a distance of about 1 cm. The aperture of the right coronary measures about 0.3 cm. which rapidly narrows to a diameter of about 0.2 cm. Two aortic lesions are present immediately above the aortic valve. The first of these lesions is a diamond shaped lesion whose long axis is parallel to the circumference of the base of the aorta. This consists of a transverse laceration of the intima of the aorta which measures 2.2 cm. in its transverse diameter and 1 cm. in the supero-inferior diameter at the central portion. Both ends taper off to a point. The amount of tissue involved in the laceration of the intima is not known from the gross examination, but the base of the laceration is somewhat irregular. However, it is smooth and glistening as if covered with intima. The second lesions is located immediately above the raphe which divides the abnormal cusp into two equal portions. This lesion is also an intimal laceration which is also roughly diamond-shaped and measures 1.5 cm. in its transverse diameter, which is parallel to the circumference of the base of the aorta, and 0.8 cm. in its supero-inferior diameter in its widest central portion.
This laceration appears to involve somewhat more than the intima also and the base likewise is irregular, but smooth and shiny as if it’s covered by intima. The arch of the aorta shows a very small intimal atheromatous plaque. The ascending portion appears to be dilated, the circumference measuring about 8.5 cm. There is a coarctation of the aorta which is located at a point 2 cm. distal to the insertion of the ligamentum arteriosum. The ligament measures 1.4 cm. in length and 0.4 cm. in diameter. It is completely occluded. The circumference of the coarctation is 0.4 cm. The diameter of the aorta about 1 cm. above the coarctation is 3 cm. and below the coarctation at a distance of about 2 cm., the circumference is 5 cm. Immediately below the coarctation, there is an elevated plaque which measures 2 x 1.2 cm. The long axis of this plaque is in the long axis of the aorta. The thickness of the wall of the aorta including the plaque is about 0.4 cm. In the center of the plaque there is a granular vegetation. Otherwise the plaque is covered by smooth connective tissue. On section through the vegetation, it is noted that the vegetative material extends deeply into the plaque for a distance of almost 2/3 the distance through the plaque. The chamber of the left ventricle is
practically rounded. The columnar chordae carneae are all thickened. The apex is definitely rounded. The left ventricular wall in the region of the interventricular septum measures 3 cm. in thickness and laterally, it measures 2 cm. The musculature is firm and of a good color. The right ventricle wall measures 1 cm. in thickness. It likewise is firm and has a good color. The specimen is on display in L. S. U. Museum.

This shows the degree of hypertrophy of the left ventricle. The coarctation is visible with the size of the lumen demonstrated by the passage of a plunger of a tuberculin syringe.
Lungs.

The right lung weighs 790 grams; the left, 730 grams. There is a mottled reddish brown and purple color over all. They are boggy. Dark airless areas are scattered over all lobes. The cut surface shows confluent reddish brown areas of normal lung tissue.

Spleen.

The spleen weighs 215 grams and measures 13.5 x 9 x 5 cm. The capsule is smooth, moderately soft and reddish purple. The cut section shows a reddish purple color with a granular appearance. Many gray malpighian corpuscles project slightly above the cut surface.

Liver.

The liver weighs 1615 grams and measures 26.5 x 15 x 8 cm. The capsule is smooth and glistening. The outer surface is grayish brown. The cut surface shows a grayish brown liver tissue. The central veins show no pathology. The gallbladder measures 7 x 3.5 x 3 cm. The mucosa is smooth. The gallbladder contains about 15 cc. of dark green thick bile. The mucosal surface shows a bright yellow glistening elevation. The walls are not thickened. The extrahepatic bile ducts are patent. No stones are found.
Aorta.

A plaque of 2 cm., in diameter, which is raised and granular, suggests evidence of subacute bacterial endocarditis in the area of coarctation. The coarctation is at the base of and distal to the left subclavian. The lumen of the aorta measures 0.6 cm. in diameter. The circumference is 5.5 cm. below the coarctation. The diameter is 1.5 cm. above the coarctation. The ductus arteriosus is closed.

All other organs were essentially negative and non-contributory.
REPORT OF A CASE WITH DIAGNOSIS MADE CLINICALLY

On November 11, 1940 W. G., a white married male laborer of 27 years, presented himself to his local doctor for evaluation of the following symptoms:

1. Palpitation
2. Fast heart
3. Cold feet

All for a duration of about one year.

Past History.

Essentially negative. He had a mild case of scarlet fever as a child.

Family History.

Father--Died accidentally at 50.

Mother--Living and well. Has high Blood Pressure. She had a stroke at 29 years and now at 70 shows no residual.

Sisters--5, Living and well.

Brothers--9, Living and well.

Wife--Living and well.

Children--2, Living and well.

Present Illness.

This patient has always been well except for a short spell of palpitation about one year before he was first seen on February 13, 1939. In February 1939, he was working very hard being employed shoveling coal.
He developed palpitation and precordial pain and consulted his local doctor who discovered he had hypertension and referred him to Dr.--- in Omaha for treatment.

While here, he was in Methodist Hospital for one week, November 16, 1940 to November 23, 1940. Since leaving the hospital, he has tried various jobs but had to give them up because he could not find any work that did not require manual labor.

He had precordial pain which radiated to his left shoulder and left scapular area, but did not radiate down the arm. The Blood Pressure in the right arm was 190/110, left arm 180/106, right leg 116/100, and left leg 116/100.

Physical Examination.

The physical examination was essentially negative except for the following findings: The apex beat was best heard 10 cm. to the left of the midsternal line in the sixth interspace. A loud, blowing, systolic murmur was heard over the apex and referred to the entire precordium, axilla, and left scapular area. There were prominent pulsations of the vessels in the neck, particularly the carotid artery and of the precordium.
The pulsations could be felt through the vessels over the back of the upper chest. Femoral pulse was barely perceptable as was the dorsalis pedis.

X-Ray. (November 20, 1940).

There is cardiac enlargement with preponderance of the left ventricle. The cardiac pulse was unusually vigorous and rapid. The aortic notch was not particularly prominent. The ribs show definite notching along the inferior border on the 3, 4, 5, 6, 7, 8, 9, ribs on the left and 4, 5, 6, 7, 8, 9, on the right.

Impression: coarc of Aorta with hypertension and development of collateral circulation through the intercostal arteries.
Unfortunately there has been no follow-up of this patient due to the fact that he moved out of the state and left no record of his whereabouts.
REPORT OF CASE II WITH DIAGNOSIS MADE CLINICALLY

The patient, Mrs. E. C., a 56 year old, white, married, female, who presented the interesting clinical features of this comparatively rare condition came into the University of Nebraska Heart Clinic for the first time on April 27, 1935 complaining of:

1. Shortness of breath
2. Pain in left shoulder

Family History.
Essentially negative.

Past History.
This revealed that she had attacks of Rheumatic fever in 1919, 1920, and again in 1927.

Physical Examination.
On inspection, she appeared to be a moderately obese, well, and healthy female weighing 204 lbs.

Other findings were essentially negative except for the chest findings. Her heart was enlarged to the left, the apex beat being visible in the 5th interspace 6 inches left of the mid line. A marked thrill was felt over the left chest. A marked systolic murmur was heard best over the tricuspid area. There were distinct carotid pulsations in the neck.
The Blood Pressure in her upper extremities at this time was 290/130.

She was put on a low caloric diet and told to return in two weeks.

On May 4, 1935, she weighed 200 lbs. and her Blood Pressure in the upper extremities was 270/128. She was diagnosed as an essential hypertension and a sympathectomy was recommended. Electrocardiogram showed left ventricular preponderance with right coronary arterial pathology.

She was hospitalized at the University of Nebraska Hospital on May 31, 1935 and underwent a right thoracic sympathectomy and splanchectomy on July 5, 1935 and a left thoracic sympathectomy and splanchectomy on July 11, 1935. Post-operative course was uneventful and she was dismissed on July 31 with a Blood Pressure reading of 170-196/70-90 in the upper extremities.

One month later, on August 21, 1935, she came into the heart clinic for post-operative check-up. She complained of palpitation on exertion and headaches. At this time a systolic blow was heard in the interscapular area but no thrill was felt. Standing, her Blood Pressure was, in the right arm 180/120, left arm 200/150, right
leg 128/92, and in her left leg 120/100. At this time her condition was diagnosed as coarctation of the aorta.

One week later, on August 28, the diagnosis was confirmed by X-Ray. The systolic readings in her upper extremities was elevated.


The left cardiac border was 9.5 cm. from the mid line, the right cardiac border was 4.5 cm. from the mid line and the internal thoracic diameter was 29 cm. giving a thoracic ratio of .52. The aortic shadow does
not appear prominent in the antero-posterior projection and is faintly visualized down to the pulmonic conus.

There is definite notching of the 7, 8, and 9th ribs posteriorly, somewhat more pronounced on the left. Impression: There is relatively deep notching of the 7, 8, and 9th ribs bilaterally, somewhat deeper on the left associated with moderate cardiac enlargement of the left ventricle consistent with coarctation of the aorta.

She was treated with Nitroglycerine, Digitalis gr. iss, t.i.d. and Phenobarbital gr. l/4 t.i.d.

Her blood pressure was down on October 16, 1935, the reading being 180/120. She complained of dyspnea, palpitation, headache, and a roaring sensation in her head. The treatment of 8/28/35 was continued and she was advised to avoid all exertion and to rest as much as possible. Physical Examination at this time revealed a visible pulsation in the right interscapular area.

As she had no complaints she was not seen again until November 13, 1942 when she was admitted to the University Hospital with the diagnosis of coarctation of the aorta with hypertensive heart disease. On admission her Blood Pressure reading was, in her left
enlarged for the patient's height and weight. The arch of the aorta appears narrow and elongated.

Impression: Suggested coarctation of the aorta.

From December 1942 until January 4, 1947, she visited the Heart Clinic at various times with much the same symptoms as she previously presented. Upon the latter date she came into the University Hospital for the third time for the evaluation of her cardio-vascular symptoms. She was put on complete bed rest, Digitalis gr. iss/daily, Phenobarbital gr. ss t.i.d., and Aminophylin gr. iss t.i.d.
films indicate a progressive increase in the size of the heart from 135 mms. in 1939.

Irregularities of ribs again noted.

Impression: Coarctation of the Aorta with probable bifurcation at the level of the arch with the right branch being the largest. This forms essentially a right sided aorta.

At this writing, this patient is still in the University Hospital. She shows some signs of cardiac decompensation, she has pitting edema of both legs, and fluid in her abdomen.

With her cardiac reserve reduced to almost zero, her prognosis is indeed grave.
CONCLUSIONS

1. This condition is not as rare as previously believed. The rarity, I believe, is attributed to the fact that the diagnosis is frequently missed clinically. With the advent of routine chest films, the diagnosis will be more easily recognized and we can then more accurately ascertain the incidence.

2. The adult type is the only one of interest, the infantile type being incompatible with life due to the major associated abnormalities.

3. The theories of causation are largely a matter of conjecture. The adult type must occur after birth and is never found in intra-uterine life.

4. In the adult type only minor defects may be found or they may be absent. The most common type of abnormality being bicuspid aortic valve.

5. Myocardial failure is the main cause of death, followed by rupture of the aorta and dissecting aneurysm.

6. There is a relationship between the degree of obstruction and the extent of the collateral circulation which has hitherto been unexplained. The extensive collateral circulation that is set up may be why there
are no diagnostic clinical manifestations until there is secondary cardiac embarrassment caused by the individual.

7. Clinical Diagnosis can be made by the disparity of blood pressure readings in the upper and lower extremities. There is little or no femoral pulse. Radiologically the diagnosis is made by the absence of the aortic knot, aortic dilatation, rib erosion and increased ventricular diameter.

8. Treatment in a few select cases by a skilled surgeon is surgical excision of the constricted part, otherwise it is palliative.

9. The prognosis for a long and active life is poor.

10. In the two cases whose diagnosis was made clinically, the diagnosis was made on a basis of the clinical and radiological manifestations as was presented in this paper.
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