COARCTATION OF THE AORTA (ADULT TYPE). A REPORT OF THREE CASES*

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THREE cases of the adult type of coarctation are reported. Two are new additions to the literature. One has been mentioned previously (Blackford[2]). The latter case is added because of some findings not mentioned in his report.

The history and pathogenesis of coarctation have been so ably reviewed in the literature (Abbott,[1] Blackford[2]) that these features are omitted in this report.

One point which may have a bearing on pathogenesis, particularly for those who still think the condition is of postnatal origin, I wish to mention. Dally[4] in discussing the descent of the diaphragm at birth claims that this descent (contraction) changes the direction of the currents of blood between the auricles and in the ductus arteriosus. He refers to Keith[5] who states: "When the right crus contracts at birth, with the first inspiration, it draws the pulmonary arteries and the fixed margin of the vestibule of the left auricle 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. . . . . Thus the contraction of the right crus, while helping to expand the lung, also closes the foramen ovale and ductus arteriosus."

That this pull exerted at a distance may be one of the jumble of forces acting on the ductus is merely stated. Its influence on coarctation is of course purely speculative. Those who have seen a case of coarctation at the postmortem table can hardly fail to have received the impression that the real cause of coarctation lies in the anomalous redistribution of the primitive arches which go to make up that area of the definitive arch.

The clinical diagnostic criteria for coarctation (adult type) are: (Oppholzer [1848][6]) (1) differences in pulse volume (palpable) between radial and femoral arteries; absence or minimal pulsation of the abdominal aorta; (2) evidence of arterial collateral circulation on chest and abdomen; (3) aberrant systolic noises on chest (Laubry [1926][7]); (4) difference in pulse and systolic pressure between arm and leg, the pulse and systolic pressure being higher in the arm (Railsback and Dock [1929][8]); (5) x-ray evidence of erosion (scalloping) of the ribs.

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CASE REPORTS

CASE 1.—C. S., aged thirty-two years, male, entered the hospital December 31, 1928, discharged January 16, 1929. The patient was sent in for thyroidectomy. Two basal metabolic rates taken at intervals—the latter, one week before admission—were plus 42.

Present Illness: Onset six years ago—dizziness, shortness of breath, palpitation. Hypertension noted six years ago. He tolerates heat well but suffers from the cold. There has been puffiness of the face and hands. During the last three months he has taken Lugol's solution, minims 10, three times daily. The patient was referred to surgery because of the request of his physician, and because he had a thrill over the enlarged thyroid, tremor of the fingers on extension of the hands and a history of increased basal rate readings. A hypertension, recorded as 210/134 mm., was noted, and there was also an enlarged heart with a systolic murmur at the apex which was not transmitted.

The family history is unimportant except perhaps for the fact that the mother died of apoplexy (age not stated). The patient is married. His wife and children are well. He has diseased tonsils and cervical adenopathy, also pansinusitis (x-ray).

When seen by Dr. Karl Anderson of the medical staff it was noted that there was a "mass" under the sternum, a to-and-fro extracardiac murmur heard in the back at the angle of the scapula, also several pulsating vessels in the back. The diagnosis was at the time "hypertension," "mass in upper part of the thorax." "hyperthyroidism, questionable." "Operation should be deferred for more accurate study and to obtain a better understanding of extracardiac murmurs." On comparing the radial pulsation with the abdominal and femoral pulsations a striking difference was noted. Further observation of this man revealed, besides the marked pulsation of the arteries of the neck, the visible pulsation of vessels in the back along the vertebral border of the scapula and palpable vessels which pulsated along the intercostal spaces. When this patient became quieter, no cardiac murmurs were audible. A systolic bruit in the neck vessels was heard and there was a postsystolic murmur in both interscapular areas at the level of the second and third thoracic vertebrae. Deformity of the spine (scoliosis) was noted. The mass under the sternum was interpreted as an enlarged vessel off the arch, because retrosternal dullness varied under rest.

Eye grounds—arteriosclerosis, tortuosity of vessels. Kidney function normal. Blood and urine normal. Basal metabolic rate on January 5, plus 4. Electrocardiogram, January 8, 1929—normal complexes, T in Lead I flat.

Blood pressure readings in arms and legs:

		Left	*Right*
March 3, 1929	Arm	186/108	194/112
	Leg	124/118	114/ ?
April 14, 1929	Arm	190/120	188/120
	Leg	120/110	120/110
May 24, 1929	Arm	168/102	170/102
	Leg	130/120	120/92
January 26, 1930	Arm	208/120	198/126
	Leg	140/120	140/118
May 18, 1930	Arm	220/115	210/115
	Leg	140/125	140/120

Physical findings on January 26, 1930, about one year after discharge from the hospital: Congestion of face and slight cyanosis. Pupils equal, react to light and accommodation. Some visible pulsation of vessels of the neck. Some venous

dilatation. Some asymmetry of thorax—left bulges slightly, moves freely. Some cardiac enlargement. Abdomen is normal. Extremities of good color, veins are quite numerous and visible. Percussion of thorax—clear, no retrosternal dullness found. Patient feels well. Has no roaring in his ears, no headaches. Legs tire when he goes upstairs. Arm and leg pressures noted in table of pressures.

The patient had been working steadily for one year at fairly hard labor in a steel mill.

X-ray Report (Dr. Rigler): January 5, 1929, Ad 6390, Chest and Spine: Fluoroscopic and film examination of the thorax was made. There is a shadow in the superior mediastinum which pulsates slightly. This may be due to a substernal thyroid, but its appearance is not characteristic, and the possibility of its being due to dilated vessels cannot be ruled out. The heart appears to be within normal limits in size, shape and position, but there is a marked distortion of the spine which causes the heart to protrude to the left side. Only the arch and ascending aorta can be fairly visualized. The descending aorta was not well visualized. Plates of the spine reveal multiple deformities, the third and fourth dorsal vertebrae being used together, and the seventh and eighth being very much deformed. Its appearance is characteristic of a congenital lesion, and suggests intercalation. A further study of the spine is advisable. There is a marked deformity of the ribs on the right side, secondary no doubt to the spinal deformity and some scoliosis. Erosion of the ribs along their inferior margins is shown. This is characteristic of dilated pulsating arteries. *Conclusions:* Congenital deformity of spine. Possible substernal thyroid. Possible dilated vessels of neck. Erosions of ribs secondary to dilated intercostal vessels.

January 29, 1930, Ae 864, Chest: Reexamination of the chest and heart with the barium filled esophagus was made. The findings are much the same as last reported. There is little or no distortion of the esophagus except that it is displaced somewhat to the right in the anteroposterior view just below the arch of the aorta. The aorta could not be clearly visualized and suggests an atresia. The distinct erosion of the ribs previously reported is again shown and is due to pulsation of the tortuous intercostal vessels. The deformity of the spine previously reported is again shown. *Conclusions:* Multiple congenital deformities of spine. Erosion of ribs secondary to tortuosities of intercostal arteries. Distortion of esophagus secondary to abnormality of aorta. Enlarged heart, left ventricular type.

Comment: The x-ray report of erosion of the ribs in this man with the correct interpretation of its cause, was made previous to our knowledge of the publication of Railsback and Dock's[8] article. It was decided at the time of the observation to wait for verification of this finding in other cases before calling attention to it. This was neatly done by Railsback and Dock. The syndrome of hyperthyroidism in this case and its appearance in cases of coarctation are discussed subsequently.

CASE 2.—Miss S., aged twenty-one years, patient of Dr. M. W. Alberts, St. Paul.* The patient was seen in January, 1930. She is a young woman in apparently good health attending the university. She gives a history of having had symptoms of hyperthyroidism five and a half years ago. Thyroidectomy was performed without results. One year later another portion of the thyroid was removed. This improved the patient's nervous symptoms but did not relieve her hypertension which had been present throughout this period. It was then noted and there was a marked

*We again take occasion to thank Dr. M. W. Albert for his permission to use this case as clinical material and for the opportunity to mention it in this report.

difference in pulsation of the brachial and femoral arteries. The diagnosis of coarctation was made. Since her operation four years ago she has been slightly restricted in her activities.

Physical Examination: The patient is a well-nourished, well-developed young woman. There is a full thyroid; the vessels pulsate in the neck but there is no thrill. The heart is not enlarged. All over the chest a systolic bruit is heard. Besides this, cardiac sounds are well heard all over the chest. Along the sternum on the right a systolic murmur is heard which can be traced far down the abdomen to the level of the umbilicus. A similar murmur is heard on the other side of the abdominal wall. Along the right axillary area this systolic murmur is pronounced. In the back, pulsation of intercostals can be made out on palpation from midscapular area to as low as the ninth interspace on both sides. A systolic bruit is heard along the inner margin of the scapula on both sides. The characteristic interscapular systolic noise is not so obvious as in Case 1. There is absence of palpable pulsation in the abdominal aorta and very feeble pulsation of the femorals.

Eye grounds, beginning arteriosclerosis, tortuosity of the vessels. Blood pressure readings:

		Left	*Right*
January, 1930	Arm	190/100	200/110
	Leg	130/110 difficult	130/110
In May, 1930	Arm	170/90	170/90
	Leg	128/100	120/95

X-ray Report: February 3, 1930, Ae 1023, Chest and Esophagogram (Dr. Rigler): The heart shows only a very slight enlargement, chiefly in the left ventricle. The aorta shows some slight dilatation of the ascending portion, but the arch is small and the descending portion can be very poorly visualized. It can, however, be made out. A definite area of constriction in the aorta cannot be clearly visualized. There is no particular distortion of the esophagus. A definite erosion on the inferior surfaces of the ribs on both sides extending down to the eighth rib posteriorly is clearly made out. This is characteristic of the type of erosion which is clearly made out. This is characteristic of the type of erosion which is due to tortuosities of the intercostal arteries. There is a shadow in the superior mediastinum very ill-defined but suggesting enlarged dilated vessels. *Conclusions:* Slight cardiac enlargement, left ventricular type. Erosion of ribs from pressure of vessels; coarctation of aorta (clinical).

Comment: Blackford[2] reported this case, we assume as No. 7. Since he gave no differential blood pressure readings nor mentioned the erosion of the ribs, we have incorporated this data. This is the second case which has come to our notice presenting at some time in the history evidence of a hyperthyroid complex. The possible explanation for this will be given in the discussion.

CASE 3.—O. J., aged twenty-three years, admitted March 6, 1930.

Present Illness: The patient felt very well until February, 1929, when he developed an inguinal hernia on the right side. This did not trouble him very much so he did light work. The hernia increased during the summer of 1929. He worked in the harvest fields in the fall of 1929. He felt very well when lying on his back and the hernia was reduced. Swelling of the abdomen began in February, 1929. It was gradual and painless. No jaundice was noticed at the time. There was no edema of the ankles, no dyspnea, no cyanosis. He was confined to bed for one month before admission.

Past History: His previous health had been good. Apparently he had a normal childhood. He ran and played with the other children. He never was short of breath. The mother noticed that he was usually pale. He never flushed when running. He catches "cold" easily. There have been no headaches, no palpitation. No dyspnea was noticed until six weeks before admission. There is no history of rheumatism. The family history is negative.

Physical Examination: On admission there was slight dyspnea, and he was able to lie flat in bed without discomfort. Sclera icteric; slight acrocyanosis. Thorax: Lungs clear; marked enlargement of the heart to the right and left; shape of the heart probably altered by intra-abdominal pressure. A rough systolic thrill was felt at the apex. At the base there was a rough diastolic murmur occupying the whole of diastole evident in the pulmonic area. No other murmurs were noticed. Pulse 41 to 56; occasional extrasystoles. Blood pressure 154/96 mm. Abdomen: Marked ascites; liver down to umbilical level. Liver seems firm, not tender. Spleen not felt. Right inguinal hernia with fluid, easily reducible. Extremities: Reflexes diminished. Slight edema of the legs. Slight cyanosis; radials sclerotic. No clubbing of fingers present.

Paracentesis on March 7, 1930: Liver now made out as enlarged, with round hard edge; surface feels granular. Spleen palpable and hard. Lungs clear. Heart: Murmurs have changed; now a rough systolic murmur was heard over the base. Pulsation was made out on vessels of the back particularly along the vertebral borders and in the intercostal spaces. Pulsation of the abdominal aorta was not found. Pulsation of the femorals was very faint. The ascites was controlled by novasural and ammonium chloride. On March 29 the patient was up and about. No edema of the legs. On sitting up, there was systolic retraction at the apex. On palpation a short rough thrill was felt at the apex.

Paracentesis on April 30, 1930: Murmurs at base have disappeared. Posteriorly the murmurs persist "postsystolic in time." The point of greatest intensity of this murmur is at the level of the third thoracic spine but can be heard as far down as the seventh spine in both intrascapular spaces. The pulsation of the neck vessels was very forceful but did not have the celerity felt in the other cases of coarctation we have seen. In fact, at times the radial pulse suggests aortic stenosis. There was a systolic retraction in the lower intercostal spaces on the left, in the axillary line. Conjunctivae were slightly yellow.

Eye Grounds: "O. D. vessels, particularly the smaller arteries and veins, very tortuous; some change in vessel walls, particularly arteries. O. S. similar to O. D. Tortuosity of small vessels marked." (Dr. Lane.)

Laboratory: Blood normal. Icterus index March 11, 1930: 12. Icterus index May 7, 1930: 20. Urine: Urobilin, P. S. P. 80 per cent two hours. Electrocardiogram March 11, 1930: Bradycardia, left bundle-branch block.

Blood pressure ranged from 154/96, 144/86, to normal readings in arms. Leg readings were not obtainable. In September, 1930, readings were obtained in his legs.

	Left	*Right*
Arm	140/78	140/82
Leg	104/90	104/90

X-ray Reports: March 10, 1930, Ae 1894, Six-foot Chest (Dr. Rigler): The heart is greatly enlarged to the right and left with a marked bulging in the region of the conus pulmonalis and considerable enlargement of the left ventricle. The aorta is very poorly visualized, especially in its ascending portion, but even in the descending portion it is very difficult to make out. There is evidence of a marked pulmonary congestion on both sides. The diaphragms are very high. A definite erosion of the inferior surface of the posterior ribs is shown, characteristic of

coarctation of the aorta and due to the enlarged intercostal vessels causing pressure on the ribs. The esophagus shows a definite posterior displacement due to enlargement of the left auricle. Otherwise there is little or no evidence of change.

After evacuation of the fluid from the abdomen, the diaphragm has come down somewhat, and the heart does not appear quite so enlarged but is still massive, and the pulmonary congestion is still very prominent. The findings in the ribs are again shown, and the marked enlargement of the left auricle is definitely shown. The whole appearance suggests a coarctation of the aorta with some other lesion in addition, the appearance suggesting most strongly a marked degree of mitral disease in addition to the aortic disease. There is a marked enlargment of the left ventricle also.

Operations: May 14, 1930. Talma-Morison operation under spinal anesthesia. Piece of the liver examined microscopically. Report of frozen section: cirrhosis of the liver (Bell). (There was a slight infection of the bronchi following this operation; temperature 102°; leucocytes 15,500.) No pulsation on direct palpation of the aorta reported by the surgeon (Wangensteen) at the time of the operation.

June 13 to 18, tapping of hydrocele.

Discharged June 24, 1930; patient up and about.

Reentered hospital November 4, 1930. Died November 7, 1930.

After the Talma-Morison operation there was relief of ascites for a brief period. The patient was up and about. The abdomen was tapped at intervals of two and three weeks after leaving the hospital in June. On October 25, 1930, the patient had an attack of weakness, he was conscious of his heart ("pounding fast") and some precordial pain. The attack lasted eight minutes. He has had similar attacks since. These attacks never last more than ten minutes. After each attack the patient feels weak temporarily. Between attacks he does not "know he has a heart." His general health, appetite, sleep, and bowels were normal.

His physical examination on November 4, 1930, revealed no new data. When seen in one of his attacks his heart was very rapid and irregular. There was dyspnea, cyanosis and sweating. Venesection was done in his fatal attack. Two minutes after venesection the patient gasped suddenly, vomited, and his respiration became spasmodic. The heart stopped before respiration. Electrocardiogram some five hours earlier had shown tachycardia, rate 200, possibly nodal in origin.

Necropsy (Dr. O'Brien.)—O. J., A 31-1662. The body is that of a young white male, 175 cm. in length, weighing approximately 165 pounds. Nutrition and development are good. No edema or jaundice. The abdomen is distended. The veins over the anterior and lateral chest wall and abdomen are prominent.

The peritoneal cavity is filled with 6000 c.c. of clear straw colored fluid. The liver is firm and extends well below the costal margin.

The pleural cavities are free of adhesions and effusion. The pericardial sac contains approximately 100 c.c. of clear straw colored fluid.

The heart shows generalized enlargement. It weighs 870 grams. It is 20 cm. in width, the thoracic width at this level being 27 cm. All chambers are enlarged, especially on the right. The left ventricular walls vary from 1.5 to 3 cm. The right varies from 1.0 to 2.0 cm. in thickness. The auricular walls are thickened and distended. The mural endocardium is smooth and shows no thrombosis. The valve edges are free and leaflets are thin. The interauricular and interventricular septa are intact. The coronary arteries show slight internal atheromatous deposits, but no interference with the lumina. There are diffuse yellowish deposits in the pulmonary artery, root of the aorta, base of the aortic valve and the aortic leaflet of the mitral valve. The muscle is slightly cloudy but shows no evidence of fibrosis.

The aortic arch rapidly tapers down to a moderate constriction proximal to the left subclavian artery. There is a more marked constriction near the junction

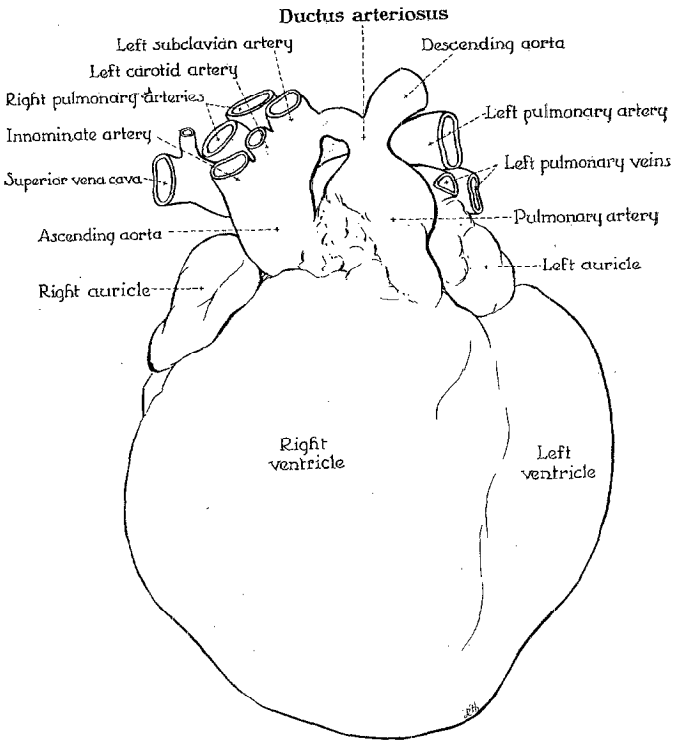

Fig. 1.—Viewed from above and slightly to the left, this drawing is an excellent proportionate outline of the heart and vessels. The arch is somewhat displaced to the left due to fixation. The descending aorta is also distorted by the removal of the specimen from the body and by fixation. The narrowing beyond the left subclavian is marked but does not give an adequate idea of the extent of the atresia because the vessels have not been opened. (See report of the pathologist.)

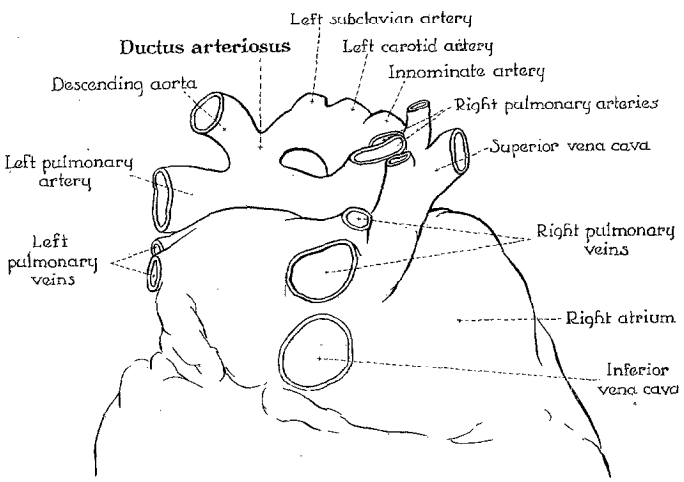

Fig. 2.—Viewed from below and behind. The same distortions of the arch and descending aorta (for similar reasons as in Fig. 1) are noted.

of the arch with the descending portion. The ductus arteriosus is patent and connected with aorta, at the site of the marked constriction. A probe is passed through the ductus arteriosus and passes readily into the descending aorta. It is practically impossible to pass a very fine probe through the distal stricture of the aorta. The descending aorta is smaller than normal. There is a dilatation of the proximal portion of the descending aorta for a distance of 5 cm. Beyond this it is uniformly narrowed, averaging 1 cm. in diameter. The common iliac vessels are smaller than normal but show no other abnormalities. The left carotid is normal in size. The left subclavian artery is dilated and thickened. The innominate artery is distended and has a thin wall. All of the intercostal vessels are markedly dilated, thickened and tortuous. The change is most marked in the first intercostal vessel on the left side. This vessel is approximately the same size as the right carotid artery. Both subclavian arteries are marked dilated. The internal mammary arteries are dilated and tortuous. The axillary arteries are apparently normal in size. The pulmonary artery and conus are markedly dilated. The two pulmonary arteries arise from the posterior superior portion of the distended trunk. The pulmonary vessels show no dilatation nor sclerosis.

The spleen weighs 500 grams and is definitely enlarged. The surface is covered with a diffuse hyaline change. Multiple adhesions between the spleen and stomach and abdominal wall are present. On cut section marked chronic passive congestion is found.

The liver weighs 2,725 grams. Slight hyaline perihepatitis is present, and the surface is roughened due to the liberation of adhesions. The organ is firmer than normal. On cut section marked darkening of the centers of the lobules is seen. There is increased resistance probably due to fibrous tissue deposits. Slight fatty metamorphosis is present throughout.

The right kidney weighs 230 grams and the left 250 grams. The capsules strip without difficulty, and the presenting surface is dark red and hemorrhagic in appearance. There is faint fine pitting over the surface. Section shows the cortices of fairly uniform width.

Microscopic Examination: Sections of the heart muscle are normal. No evidence of pulmonary arteriosclerosis is seen. The spleen shows marked chronic passive congestion. The liver presents marked congestion and atrophy of the cords in the liver lobule, especially in the central portion. There is a moderate increase in the portal connective tissue and lymphocytes, presenting the picture of a moderate portal cirrhosis. The kidneys are carefully studied from the standpoint of possible arteriolar sclerosis but none is found.

Diagnosis: (1) Coaretation of the aorta. (2) Patent ductus arteriosus. (3) Marked dilatation of the subclavian, carotid, innominate, internal mammary, intercostal and pulmonary arteries. (4) Marked narrowing of the descending aorta and common iliac arteries. (5) Cardiac hypertrophy and dilatation. (6) Chronic passive congestion of all viscera. (7) Ascites. (8) Portal cirrhosis. (9) Vascular adhesions of the stomach, spleen, liver and peritoneal cavity (operation).

ASSOCIATED ANOMALIES

Case 1 had a spinal deformity in the thoracic area; the third and fourth ribs were pressed together and the seventh and eighth being much deformed having on the right an intercalated vertebra with an an extra rib. This type of somatic deformity is new, using Maude Abbott's[3] report as a criterion.

The aorta in Case 3 certainly suggests aplasia, although the pathologist does not stress this point.

DISCUSSION

While the clinical criteria are sufficient to make a diagnosis, hemiplegia, or symtoms of hyperthyroidism with increased blood pressure in the young should make one alert as to the possibilities of coarctation. Two of our three cases had evidence of hyperthyroidism. Both Case 1 and Case 2 had definitely increased metabolic rates. In Case 2 the patient had her thyroid operated on twice before she was pronounced normal. Case 1 escaped surgical interference. Rest, with assurance that his condition had been properly diagnosed, and the withdrawal of Lugol's solution reduced the patient's metabolic rate to normal. Lorrigia[9] was the first to mention, and Blackford[2] later noted, hyperthyroidism in connection with this defect. Is the overactivity of the thyroid due to the increased vascularity of the gland? There is an apparent increase of blood flow in the areas above the clavicles. The inferior thyroid comes off the subclavian. The subclavian and its branches are the usual arteries of collateral circulation. That increased blood supply may be a factor in the overactivity of the thyroid in these cases is a reasonable hypothesis. That it may be a factor in other cases reported with this defect is also a legitimate assumption. If this is correct, then ligation of the inferior thyroid may be all that is necessary to relieve the hyperthyroid symptoms in some of these cases.

All three cases, aged respectively nineteen, twenty-two and thirty-one years, had evidence of tortuosity and beginning arteriolar changes in the retina. If, in essential hypertension, arteriolar changes of the vessels in the retina are indicative of arteriolar changes in the vessels leading to the glomeruli in the kidney (Bell), it would be of interest to examine the preglomerular vessels in these cases as they come to the postmortem table. Our single postmortem examination showed no changes in the kidney.

Students of vascular pathology have an important and striking "set up" in these coarctation cases to study the effect of work or tension, plus wear and tear or pulse pressure in the upper part of the body as compared with work or tension in the lower part. It is to be noted that in these three cases while the systolic pressure in the arm is higher than in the leg, the diastolic is the same or even higher in the leg. In other words, there is hypertension in the lower extremities. The observations of Blumgart et al.[10] differ on this point. In one case their readings given for the lower extremity show a lower diastolic pressure than normal. In their second case no diastolic readings in the leg are given. All of our readings were made with a 20 cm. cuff. We found a high normal diastolic (90 mg. of mercury) or above. Blumgart's arteriolar pressures were normal in the legs. Is the high diastolic pressure in the lower portion of the body a compensatory phenomenon to insure adequate arteriolar pressure?

Those interested in the pulse may learn something for or against

their theories as to the factors in its mechanism. The pulse is a distention of the wall of the vessel induced by the increased volume of blood propelled by the systolic force of the heart. In Case 3, during the operation for relief of his portal circulation, the surgeon had an opportunity to feel the aorta in the abdomen. He reported there was no pulsation. I know of no other observation of its kind in coarctation. That this is a constant finding cannot be true. It was usually difficult to get the blood pressure readings in the legs in Case 3, but occasionally they were obtainable. If there was a pulse pressure in the femorals at times, there must have been a pulse in the abdominal aorta at those times. The loss of pulse in Case 3 was due to extreme atresia and the dissipation of the systolic wave in the collaterals. That there was adequate supply of blood flow must be conceded. Not one of these three cases had symptoms of weakness(atrophy or loss of function in the legs. Blumgart et al. suggest from their blood gas studies a low gas reserve in the legs.

All three cases showed the characteristic erosion of the ribs described by Railsback and Dock.[5] This finding has been confirmed by others. The relation of age to the appearance of this defect is pertinently discussed by Fray.[11] If we look on page 385 of THE AMERICAN HEART JOURNAL, Vol. 3, 1927-28 (Hamilton and Abbott[8] "Coarctation of the Aorta"), the x-ray plate of the fourteen-year-old boy shows some of the characteristic erosions of the ribs. Realizing the fact that erosion is not an absolute pathognomonic sign, we have studied the esophagogram and its relation to the arch, without any definite results. We do not wholly agree with Fray that the main reliance (roentgenologically speaking) should be placed on the defect in the arch in the left oblique view.

SUMMARY

Three cases of adult type of coarctation are reported. The postmortem findings are given in one case.

Case 1 had a somatic anomaly, involving the thoracic vertebrae. This has not been reported previously in the literature.

The cirrhosis of the liver in Case 3 may have had a basis in the modified blood supply.

The diagnostic importance of x-ray evidences of erosion (scalloping) of the ribs is emphasized.

The findings of increased basal metabolic rate in two cases is mentioned. A possible explanation is suggested for this increase.

Attention is called to the "set up" which these cases afford for the study of vascular problems. The work (tension) in the vessels, plus increased pulse pressure in the head, neck and arms, as contrasted with work (tension) without pulse pressure in the legs is stressed. In the patient who died the arterioles of the eyes (clinical) and kidneys (anatomical) are compared.

Attention is called also to the high diastolic pressure in the legs. The impression from these three cases is that this is a compensatory phenomenon.

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