HEART BLOCK ASSOCIATED WITH CONGENITAL HEART DISEASE.

BY

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The association of heart block with congenital heart disease has only been recorded in this century. Since then thirty cases have been published. It has usually been assumed that the block is due to a deficiency of the inter-ventricular septum, but the majority of cases of this lesion show no block, and indeed there are cases where the septum is quite absent and yet the auriculo-ventricular node and bundle of His are present and acting smoothly. In this paper there is produced evidence to show that patency of the septum is not the sole cause of the heart trouble.

The case seen by the writer, reported by Firth in 1926, was that of a boy of six, complaining of frequent colds, and palpitation and shortness of breath of two years' standing. He lived in tenement buildings, and found difficulty in climbing the stairs to his flat. He had had diphtheria two years previously, but never rheumatic fever. His appetite was good and he was inclined to be constipated. He had never fainted. He went to a special school.

On examination, the sternum was seen to be depressed. There was no cyanosis. His lips were remarkably red, compared with his pale skin. There was no clubbing of the fingers. Pulsation was visible in the second and third intercostal spaces. The apex beat was in the fourth space, $\frac{1}{4}$ in. beyond the nipple line. The pulse rate was 48. There was a pulmonary systolic thrill, and the shock of the closure of the pulmonary valves could be felt. The area of cardiac dullness extended up to the first intercostal space, two fingers' breadth to the left of the nipple line in the fourth space, and one finger's breadth to the right of the right border of the sternum. A harsh systolic bruit was audible in the pulmonary area. The aortic second sound was blurred and there was marked triple rhythm in the second and third left intercostal spaces.

This developed later into a blowing diastolic murmur replacing the pulmonary second sound, and audible all over the præcordia. The conus arteriosus beat visibly and very forcibly. Blood pressure was 116 mm. systolic and 64 mm. diastolic. X-rays showed the heart to be enlarged, globular, and rather high. The electrocardiogram showed complete heart block, the auricular rate being 75 and the ventricular 48. A later examination showed abolition of the T-wave in lead I; there was a question of a superimposed left ventricular lesion. Red blood cells numbered 4,320,000 per c.mm. and the hæmoglobin was 80%.

It is possible that the heart block was due to diphtheria, but a congenital origin seems more probable.

PREVIOUS CASES.

The first cases reported by previous authors were published by Morquio in 1901. Five members of one family had heart block of undoubted congenital origin, but there were no anatomical deformities of the heart. Two are given here.

CASE 2. (Morquio.) Male, aged 8 years. Full time. Frequent syncope, started by emotion, preceded by malaise and a sensation of emptiness.

CASE 3. (Morquio.) Male, 5 years. Also had attacks of faintness.

Both were apathetic and distrustful. Pulses irregular, unequal and very variable rate—50 to 70. No murmurs. Complete block. Autopsy on elder—heart dilated, otherwise normal macroscopically.

CASE 4. (Scoseria.) Male, aged 4 years. Syncopes from 9 months. Pulse rate 40. Bulging of præcordia, systolic thrill and murmur at base. No cyanosis. ? Complete block.

CASE 5. (V. Stark.) Male, aged 5 years. Pulse rate 28. Some syncopes. ? Complete block.

CASE 6. (Van den Heuvel.) Female, aged 23 years. Syncopes since 2. Pulse rate 34. Some cyanosis. Loud basal systolic murmur. Complete block proved by polygraph. Atropine accelerated auricular rate only. Digitalis slowed it without affecting ventricles.

CASE 7. (Fulton, Judson and Norris.) Male, aged 1 year, with constipation, pallor and sweating. Pulse rate 40 to 50. Heart a little enlarged to right. Systolic thrill and loud murmur. Pulmonary second sound accentuated. At 1½ years, murmur only occasional. Heart enlarged, strong epigastric pulsation. Complete block shown by polygraph. Belladonna did not increase pulse rate. At 3 years, pulse rate 42, thrill and murmur had disappeared. Idea of patent interventricular septum finally abandoned. ? Cause of murmur. Father had a 3 to 1 heart block, and a sister a 2 to 1 block.

CASE 8. (Zahorsky.) Female, aged 15 months. Growing very slowly. Restless and cyanotic; expiratory grunt. Poorly nourished. Pale and cyanotic. Pulse rate 54. Heart enlarged. Loud systolic murmur all over chest. Pulmonary second sound accentuated. Liver nearly down to umbilicus. Spleen not enlarged. Had a "seizure" at 12 months and another after eating popcorn. ? Stokes-Adams' attacks.

CASE 9. (D'Espine and Cottin.) Male, aged 8½ years. Had had bronchitis and enlarged cervical glands. W. R. +; no other stigmata of syphilis. Feeble and puny. Pulse rate 20 to 32. Heart enlarged to left. Faint apical systolic murmur. No cyanosis nor clubbing. Red blood cells 5,301,500 per c.mm. Hb. 70%. 4 to 1 heart block by polygraph. Atropine did not affect the ventricular rate but slowed the auricles from 104 to 28, abolishing the block. This was not permanent. No explanation is forthcoming. Oculo-cardiac reflex slowed the ventricles from 28 to 24. Amyl nitrite—no change. The authors thought there was a congenital hole in the interventricular septum which was enlarged by a syphilitic process involving the bundle of His.

CASE 10. (Whipham.) Female, aged 2 years. Full time, but backward. Well nourished, good colour, no clubbing. Pulse rate 48 to 80, full and regular. No cardiac symptoms. Heart enlarged to right. Outline globular under X-rays. Loud systolic murmur audible especially on left side from apex to base. 2 to 1 block and right-sided hypertrophy. P.R. interval long—·2 sec. (normal for age ·12 sec.). Red blood cells 8,400,000 per c.mm. and Hb. 110%.

CASE 11. (Whipham.) Female, aged 12 years. Measles at 6. Well grown, not cyanotic. Pain round heart on exertion, for 18 months. Pulse rate slow, 40 to 60, and slightly irregular. Occasionally a run of 3 to 5 beats at 80 to minute. Heart enlarged to left. Impulse heaving. Systolic murmur in 4th space. Rarely a diastolic whiff at apex and just outside it. Pulmonary second sound sometimes reduplicated. Electrocardiogram showed marked auriculo-ventricular dissociation and arrhythmia. Atropine increased pulse rate from 40 to 60. Red blood cells 4,800,000 per c.mm. Hb. 80%.

CASE 12. (Bass.) Male, aged 15 years. Pneumonia at 3, measles at 5. Palpitations and dyspnoea. Poorly nourished. No cyanosis nor clubbing. Under normal height, weight and brain power. Pulse rate 40; 44 on exercise. Heart enlarged to left. Basal and apical systolic murmurs, former very loud and rough. Loud cervical venous hum. X-rays showed left side of heart enlarged. Complete heart block demonstrated by electrocardiogram. Wassermann and von Pirquet reactions negative.

CASE 13. (Gorter.) Male, aged 6 months. Slight cyanosis. Pulse rate 72. Complete block. No syncopal attacks.

CASE 14. (Josué.) Female, aged 18 years. Rheumatic fever at 18 months. Cyanosis on exertion and when moved emotionally; dyspnoea. Clubbed fingers. Heart considerably enlarged. Systolic thrill and murmur at base; thought to be pulmonary stenosis and patent interventricular septum. Electrocardiogram: P-R. interval ·22 sec. (normal ·12 to ·18) and Q-R-S. time ·14 sec., more than one-third of the whole complex (·38 sec.). In Lead I S-wave was very low and jagged and T-wave upright. In Lead III R-wave was very high and jagged

and T-wave was inverted and closer to S than normal. Josue thought that the left branch of the bundle of His was damaged, at the top of a patent interventricular septum, and that the lesion was congenital but might have been accentuated by the rheumatic fever.

CASE 15. (Rosenson.) Female, aged 10. Slight pain and distress over præcordia on considerable exertion. Full time; blue for several hours after birth. Had had pneumonia, diphtheria and whooping cough. No signs of heart trouble till age of 7 years. Apex beat in 5th space outside nipple line. Pulse rate 40 to 50. Heart enlarged to right and left. Long sawing systolic and diastolic murmurs, basal in origin. Electrocardiogram: complete block, auricular rate 100 and ventricular 48; left-sided preponderance. X-ray: right side of heart enlarged. No cyanosis and only slight dyspnœa after hopping 150 times. Thought to be a communication between the aorta and pulmonary artery just above the valves.

CASE 16. (Carter and Howland.) Female, aged 6 years. 6 lb. 9 oz. at birth. Had had a few syncope, but no dyspnœa even on exertion. No cyanosis. Apex beat in 5th space $3\frac{1}{4}$ in. from mid-line. Pulse rate 37. Heart slightly enlarged. Harsh rough systolic murmur loudest at apex but not conducted. Complete block by electrocardiogram. At the age of 10 the murmur was only just audible.

CASE 17. (Smith.) Male, aged 20 years. Syncope from 3 till 9 years, but could now play games and dance, and was robust and moderately muscular and well developed. Apex beat circumscribed, distinct and striking. Pulse rate 43. Heart enlarged to the left. Apical and basal systolic murmurs. Electrocardiograms showed complete heart block except in forced expiration when there was none. Under X-rays the heart was seen to be higher and more horizontal in forced expiration, therefore there was less tension on the heart, and this tension on the interventricular septum was thought to be the "last straw" to complete the heart block. Atropine did not affect the rhythm of the ventricles. Wassermann reaction negative. Red blood cells, 5,120,000 per c.mm., Hb. 98%, white cells 9,100 per c.mm.

CASE 18. (White, Eustis and Kerr.) Female, aged 4 days. A seventh child; full time; parents, brothers and sisters all well. Irregularity of heart noted before birth. Pulse rate varied rapidly between 80 and 160, at both of which rates it was regular; but at times there was bigeminy at 105 or trigeminy at 120. Cyanosis at first, not later. Electrocardiogram showed a variable block of 2 or 3 to 1 and less: right-sided preponderance. No murmur. At 5 weeks the child was seen to be a Mongolian idiot. No clubbing; cyanosis again. Blowing basal systolic murmur. No block; pulse rate 160. At eleven weeks the child was under nourished and apathetic. No cyanosis and no clubbing. Heart very large and globular. At 5 months she weighed 10 lbs.; the pulse rate ranged from 70 to 140. The condition of the block was sometimes 2:1, sometimes 4:3 and sometimes the block was absent. At 8 months a faint diastolic murmur was heard at the base. The pulse was regular at 110 and there was no block; the P-R. interval was .1 sec. (normal for her age). The authors thought the ductus arteriosus was patent.

CASE 19. (Same authors.) Male, aged 6. Full time. Wassermann reactions of parents positive, but boy appeared healthy. Broncho-pneumonia at 6 years. He had failed to gain in weight since tonsillectomy at 5. Very nervous and weak. Dyspnœa and cyanosis on exertion, but no clubbing. There was visible contraction over the præcordia and a marked carotid pulse. The apex beat in 6th space was in the nipple line and there was systolic retraction. The beat was slow, forceful and heaving in the epigastric angle. No thrill. Pulse rate 46 when he was erect, 60 after exercise. Heart enlarged to right and left. Loud apical systolic murmur after exercise only, not well conducted. The pulmonary second sound was reduplicated. B.P., 90 mm. systolic, 65 diastolic. The liver and spleen were not palpable, but the superficial lymph glands were enlarged and hard. The von Pirquet test was negative, but the Wassermann reaction was strongly positive. X-rays showed a large round heart, central in position. Complete block was shown by electrocardiogram, auricular rate being 120 and ventricular 45. Thus the condition is only probably of congenital origin, as a gumma may have destroyed part of the bundle of His or the A-V. node.

CASE 20. (Willius.) Male, aged 20. Cyanotic and fretful in first 2 years. Influenza at 17. Had a troublesome cough. Was pale in childhood and developed slowly. Pulse rate 63. Heart enlarged on both sides. Many extra systoles and a systolic thrill. A loud rough reverberant systolic murmur was audible all over the præcordia, up the neck, especially on the left

side, and out to the left shoulder. B.P., 112 mm. systolic, 82 diastolic. X-rays showed dilatation of right ventricle. The lesion was thought to be pulmonary stenosis and a patent ductus Botalli. In Lead II of the electrocardiogram the P-wave was inverted, because of a change in the position of the pacemaker node. This doubtless accounts for the extra systoles. The T-wave in Lead II was very exaggerated and peaked. Heart block complete, auricular rate 57, ventricular 63. This is very remarkable and presumably due to the failure of the sino-auricular node. The fingers were clubbed. The liver was not felt. Red blood cells, 7,400,000 per c.mm. ; Hb. 95% ; white cells 9,200 per c.mm.

CASE 21. (Barbier, Lebée and Mouquin.) Male, aged 15. Complained of dyspnoea on exertion for 3 weeks : had never fainted and was not cyanotic. The apex beat was in the nipple line in 6th space. Pulse rate was 30. Heart enlarged to right. Loud harsh basal systolic murmur conducted to left. X-rays showed the heart to be globular and enlarged. Complete block by electrocardiogram. When the eyes were compressed the pulse rate slowed down to 20 and the boy fainted. He was thought to have a patent interventricular septum.

CASE 22. (Meyer.) Female, aged 18. Seventh child ; others all healthy. Lips, hands and feet were blue and congested in infancy. Fingers clubbed early. She could not play or run at school because of dyspnoea. The legs had never swollen. She complained of palpitation in right side of chest. Feeble and poorly developed but intelligence normal. Very cyanotic. She had a parenchymatous goitre. Apex beat in 4th space in right nipple line. Pulse rate varied between 63 and 80, and was regular but for extra systoles. Systolic thrill. Heart enlarged to left. Sharp first sound, and an intense harsh systolic murmur audible best over the sternum, and conducted up the carotid arteries. Second sounds feeble. X-rays showed the heart on the right. The aorta, stomach and liver were also found to be transposed. The electrocardiogram showed right-sided preponderance. Atropine did not affect the ventricular rate but ocular compression had a very peculiar effect, as follows :—

						<i>Auricular rate.</i>	<i>Ventricular rate.</i>
Normal	..	..	..	..	..	100	.. 66
Weak compression	..	..	..	..	..	112	.. 80
Strong compression	..	..	..	..	..	55	.. 80

Thus weak compression increased the rate of all the chambers, but the auricles were greatly slowed and the ventricles unaffected by strong compression. Red blood cells 6,700,000 per c.mm. and Hb. 130%. White cells 11,000 per c.mm. W.R. negative.

CASE 23. (Gibson.) Female, aged 12. Complained of loss of weight and appetite. Usually enjoyed good health but was not as active as her playmates. Suggestion of cyanosis. Pulse slow and regular—rate 50. Heart slightly enlarged to left. Basal systolic murmur to left of sternum. Complete block by electrocardiogram. Regarded as probably a patent interventricular septum.

CASE 24. (Romberg and White.) Female, aged 9 months. Brought up for constant crying and excessive perspiration. Full time. The midwife thought she would die at birth. Cyanotic then ; now only when she cries. Fairly well developed and nourished. Pulse rate 60 to 70 ; frequent arrhythmia. No thrill. Heart not enlarged to percussion. Loud systolic murmur in lower præcordia. Suspicion of a diastolic bruit at apex and towards sternum. Complete block shown by electrocardiogram ; auricular rate 160, ventricular 70. No œdema nor clubbing. Liver and spleen not felt. A month later a long diastolic murmur was heard. Diagnosis :—probably a patent interventricular septum and possibly a patent ductus arteriosus.

CASE 25. (Leconte.) Female, aged 3. Diphtheria and whooping cough at one year. Broncho-pneumonia at 2, when her pulse rate had never exceeded 80. No dyspnoea, cyanosis nor fits. Child was pale but normally developed. Pulse rate 48. Systolic murmur loudest in pulmonary area. Diastolic murmur and reduplication of pulmonary second sound. Complete block and right-sided preponderance shown by electrocardiogram. Leconte diagnosed mitral stenosis and patent interventricular septum, and congenital rupture of the bundle of His. He wondered whether the hole in the septum had been enlarged during the diphtheria.

CASE 26. (Wilson & Grant.) Female, aged 14 months. Persistent vomiting. Frail and blue from birth. Bronchitis frequently. One sister with congenital dislocation of the hip. Weight 12½ lbs. No signs of syphilis. General cyanosis, particularly of the cheeks, lips, fingers

and toes. Distended venules in cheeks and limbs. Nose bulbous and fingers and toes clubbed. Pulse rate 66. Heart enlarged on both sides to percussion, and seen to be very big and globular when screened. Rough systolic murmur and thrill especially at base. Liver enlarged, its edge being in right iliac fossa. Slight œdema of the legs. Scattered rhonchi over both lungs but no signs of effusion or consolidation. Red blood cells, 1,111,000 per c.mm., and Hb. 128% ; white cells 27,800 per c.mm. (If these figures are correct, each red cell contained more than 5 times its normal amount of hæmoglobin.) Wassermann reaction of blood negative. Electrocardiogram : two to one heart block. P-wave upright in all leads and very large in lead II. Ventricular complexes normal, Q-wave being present in leads II and III. The extra auricular waves were superimposed on the preceding T-wave. Diagnosis : congenital pulmonary stenosis and patent interventricular septum. For a fortnight the child improved ; the vomiting ceased, the œdema and liver enlargement disappeared, and the weight increased. Pulse rate usually varied between 60 and 70, and never exceeded 100. Thereafter the vomiting returned, with diarrhœa ; the signs of heart failure again appeared, with intense cyanosis, and the child died suddenly.

At autopsy all the organs were found to be congested. The kidneys were fused on the right side, but with separate pelves and ureters. There was hydronephrosis of the left element, with constriction of the pelvi-ureteral junction. The heart weighed 2 oz., and was globular. The aorta was very wide and flattened antero-posteriorly. It gradually narrowed as it descended. The pulmonary artery was much reduced in calibre and indeed was quite cut off from the ventricular cavity. At its junction with the ductus arteriosus, which had an external diameter of 3 mm., it measured 5 mm. across. The right auricle was large, and the venæ cavæ dilated, but the left auricle was very small. The ventricle was a common one, with no definite septum. Its wall was thick, and fibrous in the right upper portion. A large aortic orifice was placed centrally. The tricuspid valve was deformed but not stenosed. The mitral valve was normal. A rounded muscular prominence running down the posterior wall alone represented the septum. The endocardium over it was thickened and below the former lay the left branch of the bundle of His. The A-V. node was well developed, but the conducting fibres coming from it were interrupted and broken up by the fibrous tissue of the central fibrous body, only to re-unite and divide normally into right and left branches lower down. None of the fibrous tissue seemed inflammatory in origin.

Note.—It is remarkable that there was a normal Q-wave in the electrocardiogram, for this wave is supposed to represent the spread of the impulse of contraction through the septum, which in this case was absent. Possibly a large mass of muscle in the area of the conus arteriosus, when stimulated in an upward direction, might have given rise to the Q-wave, but it was not related specially to the conduction system to allow of its early excitation. Doubtless the fibrous tissue which broke up the A-V. node produced the partial heart block.

CASE 27. (McIntosh.) Female, aged 7 months. Good family history. Full time. Pneumonia at 3 months. Much indigestion after weaning. She had a persistent cough and slept badly. At 7 months she weighed only 7½ lbs. and was poorly developed, under-nourished, weak and fretful ; pale but not cyanotic. The cervical, inguinal and axillary glands were enlarged. Pulse rate 57 to 64. No thrill. Heart a little enlarged to left. Prolonged systolic murmur, loudest at apex. Complete block shown by electrocardiogram ; auricular rate 136, ventricular 57 ; right-sided preponderance. T-wave upright in all leads. Liver 2 cm. below costal margin ; spleen just felt. No clubbing. Red blood cells, 3,600,000 per c.mm. ; white cells 12,850 ; Hb. 60%. Wassermann reaction and von Pirquet negative. She died a month after admission to hospital, but there was no autopsy.

CASE 28. (Aldrich.) Female, age not given. She played games actively and was fond of swimming. No dyspnoea and very slight cyanosis. Pulse rate 45. Congenital heart disease ; a 3 to 1 block proved by electrocardiogram. One day she dropped down dead in the water before her swim. No autopsy.

CASE 29. (Davis.) Age and sex not given. Child did not gain weight normally. Neither cyanosis nor clubbing. Author thought there was a patent interventricular septum. Complete heart block shown by electrocardiogram ; auricular rate 125, ventricular 45. Atropine did not accelerate the pulse rate.

CASE 30. (Shapiro.) Female, aged 9. A typical blue baby from birth. Always below weight and under-nourished. Tired easily and became very blue when she played games, as she did habitually. Persistent dry cough and insomnia. Whooping cough at 6 months, measles at 2 years, influenza at 3, and pneumonia at 4. Deep cyanosis of face and all mucuous membranes. Fingers and toes clubbed. Eyes prominent and sclerotics blue. Marked pulsation of veins of neck, which was double the rate of the radial artery, 88 to 44. Bulging of the præcordia. Basal systolic thrill. Heart considerably enlarged, especially to left. Sounds slow, regular and of good quality. Prolonged harsh systolic murmur, loudest in pulmonary area. Moist râles at bases of both lungs. Liver and spleen not felt. No œdema. Tonsils and cervical lymph glands enlarged, and copious thick discharge from left ear. Provisional diagnosis : Pulmonary stenosis. By X-rays heart was seen to be enlarged to right and left. Electrocardiogram : auricular rate 130, ventricular 65. P-R. interval .28 sec. (normal .18 sec.). In lead I, P-wave was exaggerated and T-wave inverted. P-wave highest in lead II, where it was almost as high as R-wave : right-sided preponderance, and 2 to 1 block.

CASE 31. (Nissé.) Male, aged 7 years. Had had measles and whooping cough ; bronchitis every winter. Broncho-pneumonia when nearly 2. Complained of occasional palpitation at night. No syncope. Well developed, and bright mentally. No cyanosis and no clubbing. Pulse rate regular at 48. Heart a little enlarged on both sides. No thrills. Harsh systolic murmur heard all over præcordia, especially in pulmonary area. B.P. 90 mm. systolic, 60 mm. diastolic. Electrocardiogram : complete block at one time (auricular rate 92, ventricular 45) ; 2 to 1 block at another time.

DISCUSSION.

An Analysis of these Cases (see Table) shows them to be fairly equally divided as to sex, there being 14 cases of boys and 16 girls. All became evident in childhood, but at different periods. It is striking that many were not diagnosed for a considerable number of years. Indeed the average age for the slow pulse to be first noted is $6\frac{1}{2}$ years. Nor were the fainting attacks, if present, found in the earliest years. Cardiac enlargement was, of course, fairly constant. Naturally the most frequent lesion was patent interventricular septum, but it is remarkable that in the only case where an autopsy is recorded, although there was no septum, yet the block was not due to its absence, but to fibrous tissue in the A-V bundle. This is hinted at in another case when heart block is complete except during complete expiration, when the position of the heart is at its highest, and consequently tension on it at its lowest. For it is not partial interruption of the bundle, but pressure thereon, that produces heart block. Some of the hearts were low in position, reaching to the 6th space, and therefore stretched.

The slowest pulse rate recorded varied from 20 to 72, and averaged 47. Had the observations been taken on adults, the average would have been lower. Cyanosis was observed less frequently than one would expect, seeing that not only the circulation, but also the cardiac rhythm, was disturbed.

There were 23 cases of complete heart block, and 3 cases of a 2 to 1 partial block. One case showed a 3 to 1 block ; one varied between a complete and a 2 to 1 block ; one case had a block which at times was partial (2 to 1 and 3 to 1) and at other times less. Another had complete block except at full expiration, when there was none. One showed delayed conduction, with a lesion in the left branch of the bundle of His.

Author.	Sex	Age of proof of lesion.	Age when slow pulse observed.	Congenital Malformation.	Cardiac enlargement.	Grade of block.	Slowest pulse rate.	Cyanosis.	Syncope.	Year.
Writer's case ..	M	6 yrs.	5 yrs.	? Patent interventricular septum	+	C	48	0	0	1928
Morquio ..	M	8 "	4 "	None	0	? C	50	0	++	1901
Scoseria ..	M	5 "	3 "	None	+	? C	50	0	++	
Von Stark ..	M	4 "	4 "	?	+	? C	40	0	+	
Van den Heuvel	F	5 "	4 "	?	?	? C	28	?	+	1903
	F	23 "	15 "	Patent interventricular septum	?	C	34	(+)	+	1908
Fulton, Judson & Norris ..	M	1½ "	1 "	Patent interventricular septum	(+)	C	40	0	0	1910
Zahorsky ..	F	15 mths.	1 mth.	? Patent interventricular septum	+	C	54	+	2	1915
D'Espide & Cottin	M	8 yrs.	8 yrs.	?	(+)	C	20	0	0	1915
Whipham ..	F	2 "	2 "	Patent interventricular septum	+	C	40	0	0	
	F	12 "	6 "	Patent interventricular septum	+	C	40	0	0	1915
Bass ..	M	15 "	13 "	?	+	C	40	0	0	1918
Gorter ..	M	6 mths.	6 mths.	?	?	C	72	(+)	0	1919
Josué ..	F	18 yrs.	18 yrs.	Pulmonary Stenosis and patent interventricular septum	++	delayed conduction	?	++	0	1919
Rosenson ..	F	10 "	7 "	Opening from aorta to pulmonary artery ..	++	C	40	0	0	1920
Carter & Howland	F	6 "	6 "	Patent interventricular septum	(+)	C	37	0	(+)	1920
Smith ..	M	20 "	20 "	?	(+)	C & O	43	0	early +	1920
White, Eustis, & Kerr ..	F	4 days	before birth	Patent ductus arteriosus	++	2:1 & less	70	(+)	0	1921
Willius ..	M	6 yrs.	5½ yrs.	?	+	C	45	(+)	0	
	M	20 "	20 "	Pulmonary stenosis and patent ductus Botalli	+	C	63	early	0	1921
Barbier, Lebée & Mouquin ..	M	15 "	15 "	Patent interventricular septum	+	C	30	0	0	1922
Meyer ..	F	18 "	?	Pulmonary stenosis and patent interventricular septum	++	C	63	++	0	1923
Gibson ..	F	12 "	12 "	? Patent interventricular septum	(+)	C	50	trace	0	1923
Romberg & White	F	9 mths.	9 mths.	Patent interventricular septum	0	C	60	(+)	0	1924
Leconte ..	F	3 yrs.	2 yrs.	Patent interventricular septum and mitral stenosis	?	C	48	0	0	1924
Wilson & Grant ..	F	14 mths.	14 mths.	No interventricular septum	+	2:1	66	++	0	1926
McIntosh ..	F	7 "	7 "	?	(+)	C	57	0	0	1926
Aldrich ..	F	?	?	?	?	3:1	45	(+)	0	1927
Davis ..	?	?	?	Patent Interventricular septum	?	C	45	0	0	1927
Shapiro ..	F	9 yrs.	8 yrs.	? Pulmonary stenosis ..	++	2:1	44	++	0	1927
Nissé ..	M	7 "	7 "	?	+	C & 2:1	45	0	0	1928

The effect of atropine, mentioned in seven of the cases, was to accelerate the ventricles in one; in the others the rate was unchanged. In one case the auricles were retarded.

Where a blood count was reported, there was usually a polycythæmia (5 cases out of 8) but the hæmoglobin was 100 per cent. in one case, increased in 3, and decreased in 5.

The prognosis is naturally of great importance. One case died on entering the water to swim, though he had previously swum frequently. Others had relatively sudden heart failures and died in a few days or weeks. But it is remarkable that some infants lost their heart block, while the lesion in other children was not noticed until they were in their teens, and some even played games and led an ordinary life. Doubtless in every case where the heart block and cardiac murmurs and enlargement persist great caution should be enjoined, and no games or violent exertion permitted. But from the point of view of the child, when there are no symptoms, an ordinary life, even though a slightly dangerous one, seems preferable to an existence hedged about with restrictions. Every case, however, must be judged on its own merits, and considerations such as the size of the heart, the ventricular rate, the degree of heart block, and the response to exercise, be weighed and judgement delivered accordingly.

CONCLUSIONS.

1. In certain rare instances of congenital heart disease there may be heart block of congenital origin.
2. This heart block is usually associated with an anomaly of the inter-ventricular septum.
3. It is probably due, not so much a hole in the septum, as to the presence of fibrous tissue causing pressure on the bundle of His.
4. Where there is a hole in the septum, the bundle may run on the edge thereof, and be liable to damage by inflammation, causing partial destruction of the septum, in rheumatic fever, syphilis and possibly diphtheria.
5. Where patients with this disease lead comparatively normal lives they are liable to sudden death.

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