HEART BLOCK ASSOCIATED WITH HIGH BLOOD PRESSURE

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The following two cases present several instructive phases of cardiovascular disturbance which will be discussed in detail following the case histories.

REPORT OF CASES

CASE 1.—Mrs. G., aged 60; white; housework; born in Ireland; admitted Sept. 28, 1913; discharged Nov. 8, 1913. The patient was admitted with the diagnosis of acute appendicitis. Appendectomy was performed a few hours after admission to the ward and the appendiceal region drained. Recovery was slow and when the patient was discharged there was a persistent, discharging sinus. While in the surgical ward note was made, "heart beats with a very slow rhythm." The patient was discharged from the surgical service Nov. 8, 1913, and readmitted to the medical service Dec. 30, 1913, on account of dizziness, headache and faintness. She had complained of the buzzing in the head for the previous three months with headaches and dizziness, had had frequent dizzy spells, and on two occasions in the previous month she nearly fell to the floor owing to dizziness, but had never lost consciousness. She had had scanty urination with no pain or burning, occasional throbbing in the head, much shortness of breath, palpitation of the heart and slight swelling of the feet but no cough, no vomiting, some nausea and belching of gas, some general abdominal pain. The bowels were constipated. The patient had never fainted.

Examination of the heart showed a slight enlargement down and to the left. The aortic second sound was accentuated; there was a soft blowing systolic murmur at the apex.

The urine excreted while in the hospital varied between 700 and 900 c.c. in the twenty-four hours. The specific gravity in numerous examinations was always high. Traces of albumin and hyaline casts were regularly found. Phenolsulphonephthalein excretion the first hour was 10 per cent., second hour, 7 per cent. The temperature was always normal; the pulse rate varied between 32 and 68 per minute. The blood pressure estimations were as follows:

12/30 ..................................270 - 150? Pulse rate about 40.
1/1 ..................................230 - ?
1/4 ..................................230 - 90
1/7 ..................................204 - 118
1/10 ..................................194 - 78
1/12 ..................................182 - 76

Mrs. G. was seen from time to time after her discharge. She was last visited in November, 1916. At that time the pulse rate was extremely slow (36) and it had been continuously since leaving the hospital. On account of an ignorant prejudice of her husband against mechanical medical instruments, permission had been refused for any type of examination, and I have been unable to take the pressure or make tracings. The same essential conditions, however, were apparently present as when in the hospital. Cardiac compensation was good, but there

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had evidently developed a certain amount of cerebral softening, as the mentality was decidedly feeble.

Case 2.—Mrs. W., aged 65; white; occupation, housewife; admitted Sept. 26, 1913; discharged Nov. 7, 1913.

This patient was seen in the medical dispensary, where, in the course of the routine examination, it was found that she had a blood pressure of 270 systolic, 140 diastolic. On account of the cardiovascular condition she was referred to the ward where the following notes were made:

"Shortness of breath on exertion; weakness; cardiac palpitation. Began to have shortness of breath, weakness, and to feel the heart beat about the end of May; cannot do any work or exercise; walking, going upstairs and all such efforts bring about these symptoms; the patient also gets, at times, shooting pains on the left side of the thorax which radiate to the precordia, and to the left shoulder and down the left arm. The symptoms have gradually been getting worse.

"The patient has had general good health, except for the latter part of her life. Had measles, whooping cough, mumps and diphtheria in childhood; was then in good health until four years prior to admission, when she had nervous prostration lasting about two months; began to feel cardiac palpitations after this. These were soon followed by inflammatory (?) rheumatism, polyarticular, lasting about six weeks; the pain reappearing at intervals up to the present. She has had frequent attacks of sore throat, though of mild character.

"The patient's mother died of stroke of paralysis at 53; father died with considerable anasarca at 63; had nine brothers and sisters, eight of whom are dead.

"Married forty-five years; did her own housework till husband's death thirty years prior; after that did outside general housework. Had three children; no miscarriages. Denied the use of alcohol and tobacco.

"Physical Examination.—The patient is an old female, well built and preserved. The physical findings are negative except for those of the cardiovascular system.

"Heart: Apex beat; visible in fifth interspace one-half inch to left of the midclavicular line, very irregular and slow in rhythm, and the beat is moderately strong; normal area of dulness; sounds weaker than normal and irregular; no valvular murmurs heard; peripheral arteries show slight thickening; ophthalmoscopic examination showed slight sclerosis of retinal vessels.

"September 28: No apparent change since admittance. Feels comfortable while in bed without exertion. Pulse tracing shows heart block.

"October 1: Slightest exertion seems to stir up the heart to beat more rapidly. Has no difficulty while remaining still.

"October, 7: Blood pressure, 165-75; weight about the same.

"October 31: Nothing indicating extra systoles or blocked beats. Temperature, pulse and respiration normal. General condition good.

"November 7: Condition about the same as last note; no change found. Appears in good condition and has not felt uncomfortable for several weeks."

Laboratory Reports.—Blood: red blood count, 3,630,000; white blood count, 12,100; hemoglobin 62 per cent.; Wassermann, negative.

Urine: In twenty-seven examinations, traces of albumin were found at times, as well as occasional hyaline casts. Excretion varied between 500 and 1,800 c.c.; sp. gr., 1.010 to 1.032.

After four or five days in bed the pulse increased from an average of 48 to 60 beats per minute, to 80 to 100. The blood pressure while in bed during the latter part of the patient's stay in the ward was never over 165 systolic, 125 diastolic. According to the method of Stone her heart showed an overload of 47. The pulse work (Sahli) varied from 7.1 mm. Hg lying down to 14.96 mm. Hg while sitting up. During the interval following discharge from
the hospital Mrs. W. was in fairly good condition. She was able to do a fair amount of housework without much discomfort. I saw her from time to time and noted that heart rate fell shortly after leaving the hospital and remained persistently low afterward. At my request she was readmitted to the hospital in October, 1916, for further observation. At that time the following notes were made:

"The patient has been suffering from shortness of breath for several years. She becomes very short of breath on slight exertion; walking across the room often forces her to sit down to rest. She has attacks of precordial pain which are brought on by slight exertion. The pain radiates to the shoulder and arm. She is unable to lie flat, but always sleeps propped up by several pillows. At times she has swelling of the ankles. She has had a cough for several years. Each morning she has a coughing spell lasting five or ten minutes. She is bothered very little the rest of the day by the cough. Phlegm accompanies the cough. She has no cardiac palpitation, but, on the contrary, she says at times her heart seems to beat hardly at all. The bowels are regular; appetite good; occasionally has attacks of nausea and vomiting just after eating; no trouble at urination; no nycturia. At times she has a little bearing down pain after urination.

"At the base of the lungs there are fine, moist râles. Heart sounds are clear but very slow and irregular. With the stethoscope on the point of maximum impulse the heart sounds do not correspond with the pulsation over the jugular vein, they being very much faster than the apical pulsations. All the beats heard at the apex come through to the radial. There is a soft systolic murmur over the mitral area. No murmur is heard at the aortic or pulmonary area; heart border—above, third rib; to the left, 1½ in. outside of midclavicular line; to the right, sternal line; aortic second sound accentuated; slight sclerosis of radials; blood pressure, 220-75.

"October, 12: Patient's general condition is good. She has no pain. She walks around the ward a little each day.

"October 17: Condition about the same. She complains of pain about the heart. Blood pressure, 230-65."

Laboratory Reports.—Urine in five examinations showed no casts; a very faint trace of albumin twice; sp. gr., between 1.019 and 1.029.

Blood: Hemoglobin, 60 per cent.; red blood count, 3,980,000; white blood count, 9,900.

The temperature was continuously normal; respirations varied between 24 and 16; the pulse rate was uniformly between 38 and 48, once or twice going over the latter figure.

GRAPHIC EVIDENCES OF HEART-BLOCK

The polygraphic tracings of Mrs. G. will first be discussed. G-II, taken on admission to the ward, shows a 3:1 block. As G-III shows more clearly than G-II the sequence of events, and as the two tracings illustrate the same condition, the former alone will be discussed. This tracing was taken preliminary to an injection of atropin. It can be noted in the tracing of the radial artery that each ventricular systole for the most part occurs two seconds after the preceding one. Occasionally, however, an impulse comes through from the auricle which causes a corresponding contraction of the ventricle. These ventricular systoles, when they occur, are 1.33 seconds apart and are due to the temporary change of a 3:1 to a 2:1 block. The jugular tracing shows,
Fig. 2—Tracings of Mrs. W., taken in Presbyterian Hospital.
except where the extra ventricular beats occur, a regularly recurring series of \( a \) waves which are 0.66 of a second apart. The first \( a \) wave is not followed by ventricular response; the second \( a \) wave is followed by the usual \( c \) and \( v \) waves; a third \( a \) wave occurs synchronously with the \( v \) wave. The evidence that such synchronism takes place is based on the fact that the distance each \( a \) wave is from the next is equivalent to the distance from \( a \) to \( v \) to \( a \); furthermore, the time interval between each ventricular response is two seconds, excepting where the additional beats appear, due to change in the rate of block to 2:1, when it is 1.33 seconds. If the rhythm were a 2:1 rhythm rather than a 3:1 rhythm, then the interval of this additional ventricular response would be exactly one second. Likewise, it would seem to be unlikely that a 2:1 rhythm is present, because the auricle would then contract twice at a rate of approximately 90 per minute, after which there would be a pause followed by another series of two beats. Such a condition is possible but improbable, and necessitates the assumption of a 2:3 sino-auricular block, as well. The auricles may contract irregularly when there is abnormal vagus irritability; for example, sinus arrhythmia, when extra systoles arise in the sinus or the auricle, when there is sino-auricular block and when there is auricular fibrillation; but none of these conditions is suggested in the tracing. Sinus arrhythmia, sinus or auricular extra systoles and auricular fibrillation can be ruled out at once, although a sino-auricular block might possibly be present; but as such a condition is rare, it seems fair to hold that the third auricular contraction occurs at the same time as the ventricular.

The next tracing, G-IV, illustrates a tracing taken ten minutes after the hypodermic injection of 0.2 mg. atropin sulphate. The same sequence of events takes place, except that the auricular, and consequently the ventricular, rate is more rapid as a result of the suppression of the inhibitory effect of the vagus by the atropin.

G-V is a tracing taken twenty minutes later. Again the same sequence of events occurs. In both the tracings taken after atropin was injected, there is no essential change in the character of the block, showing it to be organic rather than functional in type.

The graphic studies of Mrs. W. include not only polygrams, but also electrocardiograms. The tracing, W-I, taken in 1913, is presented as a long strip in order to show towards the end of the tracing a few contractions that arise as a result of impulses coming through without blocking in the bundle of His. Here the phlebogram shows a normal tracing. Elsewhere an additional \( a \) wave is shown which is followed by an extra wave (\( n \)) the genesis of which I have been unable to determine and which may be an artefact due to instrumental “fling.” The tracing then shows a partial (2:1) block with occasional periods when
beats get through normally. It is interesting to note the differences in size of the arteriogram waves where there is block and where the block has disappeared. The waves are almost twice as large when the rate is slow as compared with the heart beats when twice as rapid, demonstrating that when all other factors are equal, the adjustment of levers

![Fig. 3.—Electrocardiogram of Mrs. W., taken Sept. 14, 1916. Leads I and III.](image)

Fig. 3.—Electrocardiogram of Mrs. W., taken Sept. 14, 1916. Leads I and III.

being unchanged, a high pressure, presumably, causes large graphic arterial waves. Tracing W-III was taken two days after admission to the ward and following an injection of 0.3 mg. of atropin. There is no essential or minor change in the tracing as compared with the part of W-I that shows 2:1 block. Tracing W-II was taken two weeks

![Fig. 4.—Electrocardiogram of Mrs. W., taken Oct. 10, 1916.](image)

Fig. 4.—Electrocardiogram of Mrs. W., taken Oct. 10, 1916.
after rest in bed. Here a practically normal tracing is shown, except that there is possibly a slight prolongation of the a-c interval. The first electrocardiogram, Lead 1, taken September, 1916, shows partial heart block. The ventricular complex occurs every 1.32 seconds, the

auricular every 0.66 second, in the periods of 2:1 block. The P waves show a complex relationship to the R S T group, caused by the fact that the partial block varies at irregular intervals between a 2:1 and a 3:2 grade. The short cycles represent the 3:2 block, the longer cycles

Fig 5.—Electrocardiogram of Mrs. W., taken Oct. 12, 1916.

Fig. 6.—Electrocardiogram of Mrs. W., taken Oct. 23, 1916. Lead III not entirely clean and hence not reproduced.
the 2:1 block. The P-R intervals are greatly prolonged and vary according to the degree of refractility that exists at the moment in the junctional tissues. Tracings 260 A and B were taken one month later. Here the ventricular rate is 1.45 seconds, or approximately 42 per minute. Here again there is partial dissociation, with the block varying between a 2:1 and a 3:2 rhythm with a P-R interval that is 0.84 second long at times (see 260-B, Lead II). Tracings 264 and 267, taken thirteen and fifteen days later, respectively, show a clear 2:1 block with a normal P-R interval; 260-A shows a 2:1 rhythm, where every other P falls exactly or almost on the R of the preceding cycle. In Lead II the cycles are constant. In Lead III in the first cycle P is

Fig. 7.—Electrocardiogram of Mrs. W., taken Oct. 25, 1916.

0.19 of a second from R, but with each cycle, gets nearer, then merges (note the higher R wave), then appears on the down stroke; conductivity improves gradually until the last cycle shows no block. In Lead I, the second P hovers about R, at first receding from it, then approaching, till in the last cycle it is merged.

From the evidence here presented the two patients unquestionably had heart block. The first showed but little change in the character of the block at any time. The several tracings taken of patient, Mrs. W., showed variations from a normal tracing to different degrees of partial block, at times associated with an extremely prolonged P-R interval.¹

BLOOD PRESSURE FINDINGS

The most interesting determination in these two cases was the extremely high systolic pressure. In both of them the pressure during the block and before rest in bed was higher than the Nicholson sphygmomanometer can register. In reviewing the literature of heart block from 1904 to 1916, I was able to find cases of heart block associated with high systolic pressure reported by Bramwell (systolic pressure 235), Falconer (systolic pressure 230 during attacks), Gibbes and Dally (systolic pressure 240-Pachon), Gibson (pressure of 270), Gossage (pressure 200-220), Griffith and Kennedy (systolic pressure as high as 260), Grosh (pressure 225), Herrick (two cases, systolic pressure 240 and 200, respectively), Hoffman (two cases, pressure of 240 and 250, respectively), Hume (systolic pressure 230), Jellinek and Cooper (two cases with pressures over 200), Levine (systolic pressure 196), Purser and Davis (systolic pressure 214 during attack, 180 in the interval), Schwarzmann (systolic pressure 235), Souques and Routier (two cases, systolic pressure 250 and 220, respectively). In none of these cases did the systolic pressure approach closely the pressure in the two cases detailed above. The pressure of these two patients was first taken after they had been doing a certain amount of work. After rest in bed the pressure of both fell very materially; in Mrs. G's case about 100 mm. Hg; in Mrs. W's case somewhat more. When the block disappeared in the latter patient's case after rest in bed, the systolic pressure never exceeded 155, though the diastolic pressure remained high. These figures of 155-125 probably represent the true pressure without relation to the block, as they were found only when the block disappeared. Although the block returned shortly after the first stay of Mrs. W. in the ward, nevertheless, the possible dangers of the extremely high pressure having been fully explained to her, she so altered her manner of living that she took no violent, sudden or continuous exertion of any kind, and made no severe demand on the heart. Her pressure, even with the block present under these circumstances, never attained the heights reached on the first examination, which was made after resting a short time from a long walk.

Both of these patients were obviously cases of cardiosclerosis with associated fibrotic changes in the bundle of His. The first patient had apparently comparatively slight peripheral sclerosis, if we may judge from the low diastolic pressure. The second apparently had considerable peripheral resistance, as her diastolic pressure remained consistently high. In this patient the sclerotic changes evidently are progressing steadily, as the block no longer disappears after rest in bed.

2. References to the literature will be found alphabetically arranged at the end of the article.
HEART BLOCK

Probably one of the most instructive points demonstrated in the study of these two cases is the relationship of extremely high blood pressure to the cardiac strength and complete filling of the ventricle. The systolic pressure represents the strength of the heart beat, and is dependent largely on the diastolic filling of the ventricles and the completeness of systolic emptying. In these two patients the pressure was raised very markedly; there were the factors of complete filling of the ventricle as a result of the very slow ventricular rate which Henderson believes increases the amplitude of the systolic discharge, and also the fact that with each beat the ventricle was filled by the extra auricular beats with more blood than it would receive under ordinary circumstances, though Wiggers3 does not believe that auricular systoles aid materially in filling the ventricle. Furthermore, a ventricle in low tone has a greater systolic output than one of high tone, provided it is not fatigued and dilated, but is able to expel all the blood — to empty itself completely with each systole.

The complete filling of the ventricle had apparently a very distinct effect on the pressure, because on the one individual whose block disappeared, in spite of the cardiac hypertrophy, systolic pressure fell some 100 mm. Hg when the block was abolished. In other words, the very high systolic pressure apparently depended not so much on the strength of the heart muscle, but on the very complete filling of the ventricle with subsequent contraction on a large mass of blood. This has been shown by physiologic experiments; an increased systolic output causes a rise in systolic and diastolic pressure, provided the pulse rate and peripheral resistance remain constant. On a similar basis can be explained the extremely high pressures associated with aortic insufficiency. The left ventricle contains much more blood than normal, owing to the regurgitation of the blood backward into its chambers. As a result of this the ventricle is enabled to contract with the maximum amount of strength on a large amount of blood, resulting in a high systolic pressure. In both disorders, aortic regurgitation and heart block as here shown, there is, of course, the added factor of the cardiac hypertrophy, but in cardiac hypertrophy unassociated with these lesions, very high pressure is the exception, though relatively high values occur. It is by the forcing out of the larger volumes of blood with each ventricular contraction into the arterial tree that the pressure is raised so markedly, rather than by the ventricular hypertrophy per se. This latter condition is common where pure aortic obstruction exists, but the systolic pressure is usually only slightly elevated despite the hypertrophy, and the frequently associated arteriosclerotic changes. In

the tracing W-1 the difference in the size of the pulse waves when the ventricle is not so completely filled, is graphically shown.

**SUMMARY**

Two cases of heart block with extremely high systolic pressure are recorded. Evidence is offered to show that this high pressure is dependent more on increased blood mass discharged by the left ventricle than on the associated cardiac hypertrophy and peripheral sclerosis.

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