

COMPLETE HEART BLOCK IN AN EXPERIENCED PILOT

BY

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The purpose of this communication is to report the finding of complete heart block, probably of congenital origin, in an experienced pilot. A normal G tolerance, a normal response to both strenuous exercise and anoxia, together with a lack of referable symptoms while in the air indicated his capacity to perform flying duties efficiently within the limits to which he was exposed.

In 1945, an apparently healthy flying instructor, aged 23, was referred for special investigation because of bradycardia and an apical systolic murmur found at the time of his annual aircrew medical examination. Apart from admitting some recent lack of energy he had been in good health since entering the service. He was one of three normal children, born of healthy Canadian parents. He had been active in sport, and had fainted but once, following an injury. His tonsils and adenoids had been removed for nasal obstruction when he was 8 years old. He had not been subject to sore throats and there was no history suggestive of rheumatic fever. His mother first became aware of his slow pulse when he was 10 years old. In 1941, at the age of 19, he was passed medically fit for aircrew duties with the Royal Canadian Air Force. Before this, he had worked for a short period in an explosives factory, blending cordite.

Early in his airforce career, he was a member of an experimental group used in a study of the cardiovascular response to strenuous exercise. Because of his slow pulse and freedom from distress after exercise he was thought to possess better than average physical fitness. He had flown a total of 1850 hours, of which 350 hours were logged in the past twelve months. He had never blacked out, although his vision became dimmed on rare occasions when performing tight manoeuvres. The heart rates documented during his four years of service varied from 46 to 56 beats a minute. No heart murmurs were reported.

Physical examination showed a healthy well-built young man—height 66 inches, weight 139 pounds, pulse rate 48 beats a minute, blood pressure 130/68 mm. Hg. There was a faint systolic murmur at the apex transmitted to the midline which was best heard in the left lateral position and varied with respiration. In the supine position, a pulmonary systolic murmur was also heard. The liver edge was just palpable. Physical examination was otherwise normal. Blood sedimentation rate 7 mm. (Westergren). The heart was normal in size and contour by X-ray. The electrocardiogram showed complete heart block and was substantially the same as one taken ten years later (Fig. 3). Because of the early age at which bradycardia was recognized, the relatively rapid ventricular rate (Campbell, 1943) and the absence of other cause, it was concluded that his heart block was probably of congenital origin.

Further cardiograms were recorded with the pilot in various positions, after exercise and following the subcutaneous injection of atropine sulphate, 1/50th of a grain. In no instance did the ventricular rate exceed 60 beats a minute while the atrial rate, 83 at rest, rose to 115 on exercising fifty minutes after the injection of atropine (Fig. 1A).

In 1945, the subject was exposed to a simulated altitude of 22,000 feet in a standard R.C.A.F.

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decompression chamber. Cardiograms were taken periodically throughout the experiment. An altitude of 19,000 feet was reached at +15 minutes at which time he became pale, broke into a sweat, and complained of feeling faint. The pulse was hardly detectable and very slow. Oxygen (100%) was given by mask and recovery was immediate. The mask was removed at +23



FIG. 1.—(A) Lead II. Following exercise, 50 minutes after the subcutaneous injection of atropine sulphate gr 1/50. Note apparent 2 : 1 relationship of atrial and ventricular complexes. The P wave following the QRS complex is superimposed upon the T wave.

(B) to (E) Lead II taken during decompression run:

- (B) + 9 minutes. Before vaso-vagal attack.
- (C) +16 minutes. During vaso-vagal attack.
- (D) +18 minutes. After vaso-vagal attack. Breathing pure oxygen.
- (E) +33 minutes. Atrial and ventricular rates are nearly identical. Each P wave is superimposed upon the preceding T wave.

minutes and an altitude of 22,000 was reached at +32 minutes. At +42½ minutes oxygen was administered again because of rapidly developing anoxia, by which time he had remained approximately 19 minutes at or above a simulated altitude of 19,000 feet. This was considered to be a normal response. Samples of lead II recorded before, during, and after the episode of near syncope are shown in Figure 1B, C, and D. Coincident with the attack there was an abrupt slowing of both the atrial and ventricular rhythms. This slowing taken in conjunction with the clinical picture suggests that the episode was of vaso-vagal origin. On reaching 22,000 feet (+32 minutes), the atrial and ventricular rates were both recorded at approximately 80 beats a minute (Fig. 1E).

On the afternoon of the same day the subject was placed in the human centrifuge and exposed to 2, 3, 4, and 4.5 G positive, each for a period of five seconds. The procedure followed was that developed by the accelerator section of the R.C.A.F. Institute of Aviation Medicine (Rose, 1942; Rose *et al.*, 1942; Kennedy *et al.*, 1943). In brief, a standard run is one in which the candidate is exposed for five seconds to a predetermined acceleration expressed in terms of the acceleration of gravity (G) at heart level. Thus a standard 3 G run is one in which the acceleration, applied for

five seconds at heart level, equals three times that of gravity. The total run requires 23 seconds to complete. An initial four-second period is required to reach 1.5 G, during the second 5 seconds the acceleration is built up to the predetermined G. This G is maintained for the next five seconds, following which five seconds are required to return to 1.5 G, and approximately four seconds more to bring the subject to rest.

The blackout threshold of the average healthy young pilot varies between 4 and 6 G with a range of 3 to 9 G. The normal pulse response is characteristic. Immediately preceding the run the pulse rate averages 95, the range being from 90 to 130 beats a minute. This tachycardia is thought to be emotional in origin. With the onset of acceleration there is an immediate and rapid increase in the heart rate which persists for 6 to 10 seconds after full G is attained and may thus persist for one to five seconds after the G has begun to decline. The maximum pulse rate varies between 120 to 196 beats a minute. Immediately following the run, the pulse slows usually to a level below that which was present before the run. This recovery phase is commonly characterized by one or more diminishing bursts of tachycardia.

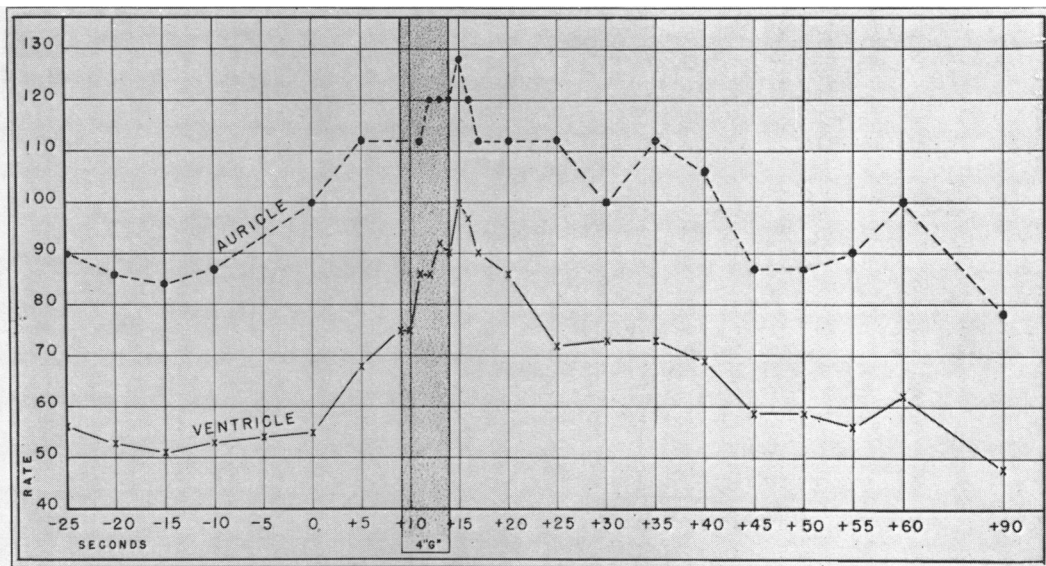


FIG. 2.—Response of auricular and ventricular pace makers to an acceleration of 4.5 G.

The present subject blacked out at $4\frac{1}{2}$ G which is considered to be within the normal range of response. A continuous cardiogram showed that complete atrio-ventricular dissociation persisted throughout each run. The rates were calculated by measuring the distance between identical points on the three complexes (both atrial and ventricular) which most closely coincided with the moment of time being considered. The atrial and ventricular rates in response to the 4 G run are shown graphically in Fig. 2. The atria and ventricles responded in a similar manner, although their respective rates differed. The maximum atrial rate of 128 occurred one second following the five-second exposure to 4 G. It then fell off but was interrupted in the usual manner by secondary rises. The curve of the ventricular response was similar to that of the atria but at a slower rate. The maximum ventricular rate of 100 occurred at the same moment as the maximum auricular rate and the secondary rises were also coincident.

Because of freedom from subjective symptoms while flying, a normal response to anoxia and to positive G, and a failure to demonstrate any functional limitation, the pilot was returned to full flying duties. He continued to serve as a flying instructor until later that year, when at the age of 23 he was retired to civilian life with an unrestricted medical category.

He re-applied to the R.C.A.F. in 1950. In view of the favourable results of the 1945 investigation and the absence of abnormal findings on examination he was classified fit for full flying duties. Following a refresher course he was posted as an instructor to an elementary flying school and was serving in this capacity when seen by one of the authors in 1951. Apart from a short systolic

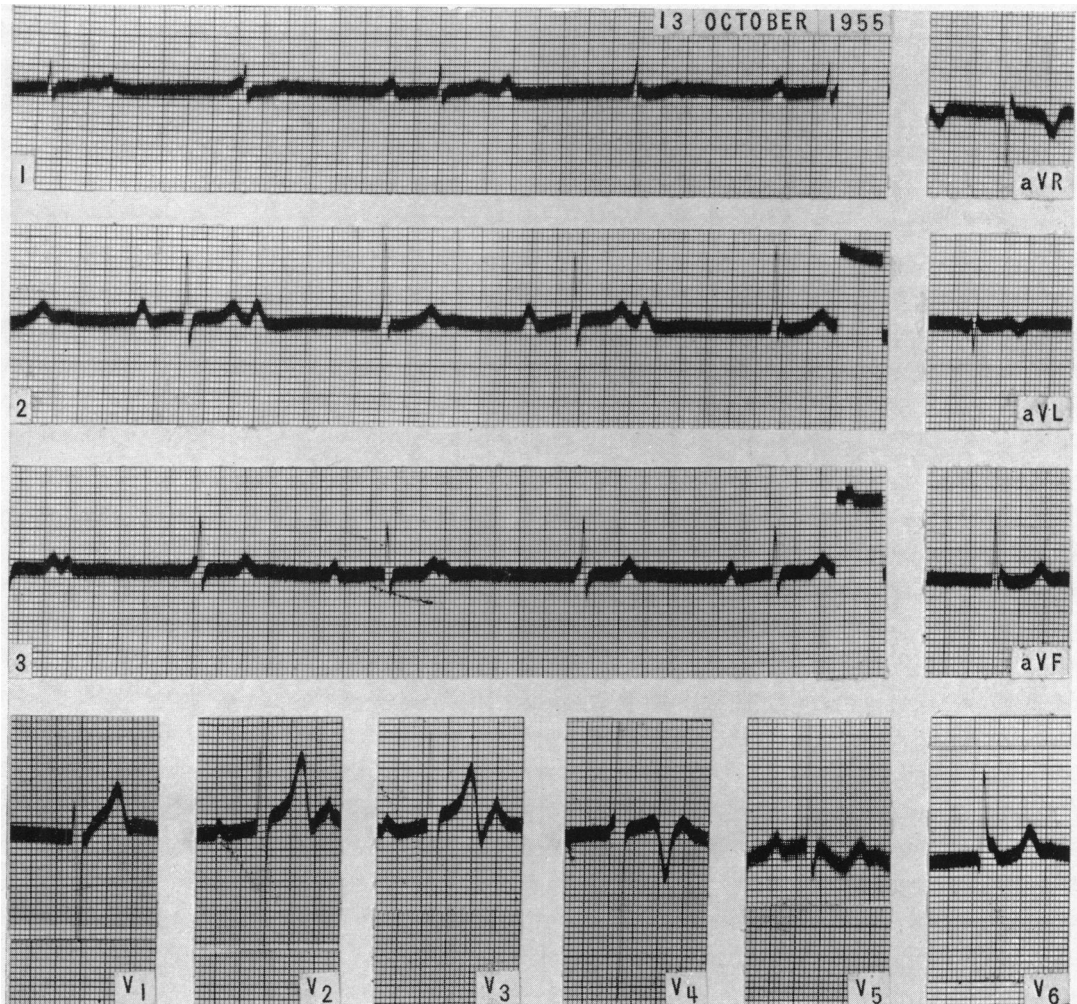


FIG. 3.—Electrocardiogram in 1955, showing complete A-V dissociation and abnormal T waves in V3-V5.

murmur at the apex, examination was normal. The cardiogram was unchanged from that recorded in 1945. He was now married and had two children: neither of them showed signs of heart disease and their electrocardiograms were normal.

In 1952, he converted to jet aircraft and flew as an examining officer at the R.C.A.F. Central Flying School, completing some 75 hours on jets and 375 hours on other single and multi-engine aircraft. His most recent flying achievement was to complete an advanced jet fighter course.

In October, 1955, he was referred for re-examination. By this time he had completed a total of 3000 flying hours and on no occasion had experienced symptoms while flying that could be attributed to an inefficient cardiovascular system. His personality was well integrated and he

exhibited a keen interest in flying. He admitted that recently his heart seemed to pound more than it used to do when he exerted himself.

On examination he weighed 142 pounds, his pulse was 48 beats a minute, and his blood pressure 132/78 mm. No heart murmurs were heard. The heart was of normal size and shape by X-ray. The cardiogram (Fig. 3) again showed complete heart block, and cardiograms with the subject in different positions and following exercise were recorded in a manner similar to those taken in 1945. The maximum atrial and ventricular rates were 88 and 75 respectively. These values were not exceeded following the intravenous injection of 1/30th of a grain of atropine sulphate.

While this pilot had an excellent flying record, he had reached the age of 33 years and was engaged in a type of flying that offered but little chance of survival should he even momentarily lose consciousness. It was ruled therefore that he be restricted to flying conventional aircraft.

DISCUSSION

Throughout the period of observation all electrocardiograms showed complete heart block. On occasion, when the atrial rate was increased with (Fig. 1E) or without (Fig. 1A) a coincident increase in the ventricular rate the relationship of the atrial and ventricular complexes suggested that the impulse arising in the sinu-atrial node was conducted. An examination of those parts of the record that immediately precede and follow these particular sections shows clearly that the A-V association was apparent and not real. In response to certain specific stimuli the two rhythms reacted in a similar manner. An acceleration of both the atrial and ventricular rates occurred in response to anoxia and positive G. Marked slowing of both took place during the episode of near syncope which suggests that in addition to the S-A node the centre initiating ventricular activity was also subject to vagal inhibition. The ventricular complexes being of the supraventricular type this centre may well be located near the A-V node.

In the 1945 tracing the T wave in lead CF4 was inverted. Similarly in the 1955 tracing (Fig. 3) the T waves in leads V2-V5 were abnormal, although in V2 and 3 the second spike that follows the main T wave was atrial in origin. The reason for these T wave changes is not clear. How commonly they occur in congenital complete heart block is difficult to determine as illustrations in published case reports are selected to demonstrate changes in rhythm and seldom include præcordial leads. These were, however, normal in 6 of the 7 cases reported by Campbell and Thorne (1956).

Turner (1947) reported asymptomatic congenital complete heart block in a trained pilot which was identified after three years service, when he was referred because of bradycardia discovered during a routine physical examination. It was under similar circumstances that the present case was discovered. Campbell (1943) described a man with congenital complete heart block who was passed for flying duties with the Royal Air Force although he was not employed in this capacity.

Stokes-Adams attacks or paroxysmal tachycardia are uncommon in apparently healthy persons with congenital complete heart block (Campbell, 1943). On the other hand, Benjamin and White (1952) identified atrial flutter for the first time in a 55-year-old woman who had asymptomatic congenital complete heart block, and Jaleski and Morrison (1943) described a woman, aged 31, with congenital complete heart block who developed premature beats before delivery and who later under strain had fainting spells. Modern fighter aircraft are capable of reaching altitudes and accelerations that impose an exacting load upon the hæmodynamics of the pilot's cardiovascular system. While it is unlikely this pilot would develop an important disturbance in rhythm under these conditions, should such occur his chances of survival would be minimal.

SUMMARY

A pilot, aged 33, with congenital complete heart block was followed for 10 years. He flew over 3000 hours in conventional and jet aircraft, including advanced operational flying. He experienced no adverse symptoms while in the air and showed a normal G tolerance and a normal response to

anoxia. Certain observations concerning the response of the atrial and ventricular pace makers are included.

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