Two patients with wandering cardiac pacemakers are discussed to illustrate techniques for deciphering the pathophysiology and clinical significance of such intermittent supraventricular arrhythmias.
<table>
<thead>
<tr>
<th>KEY WORDS</th>
<th>LINK A</th>
<th>LINK B</th>
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<td></td>
<td>ROLE</td>
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<td>Supraventricular arrhythmias</td>
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<td>Electrocardiographic dysrhythmia</td>
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Case Report: Evaluation of Asymptomatic Flying Personnel With Intermittent Supraventricular Arrhythmias

A 45-year-old pilot was referred to the USAF School of Aerospace Medicine for evaluation of cardiac arrhythmia. Five weeks previously he had noted the onset of "skipping heart beats." These occurred one to five times a minute while he was in the recumbent position, would persist for several hours, and were associated with a pressure sensation in the suprasternal notch. Jogging in place would occasionally eliminate them. Concern about the significance of his symptoms prompted medical evaluation. There was no history of rheumatic fever, diphtheria, parasitic infection, heavy metal exposure, or chest trauma. At age 30 he had had a three-day episode of right chest pleurisy which was not associated with hemoptysis. He had always been physically active and never noted chest pain or discomfort during his athletic activities. The remainder of his history was noncontributory.

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sition, his cardiac pacemaker site shifted from a normal sinus focus, at a rate of 84 per minute, to an intermittent normal sinus or low atrial rhythm, at a rate of 68 per minute. The P-R interval under either case was 0.12 second. With right carotid massage the slower atrial focus became the dominant pacemaker at a rate of approximately 56. Within 20 sec. of discontinuing a right carotid massage the patient reverted to a normal sinus pacemaker. During phase II of a standard 10-sec. Valsalva maneuver, the atrial-bradyarrhythmia reverted to a normal sinus rhythm which was again at a rate of approximately 80 per minute. Simple exercise such as 3-sec. hand-squeezing promoted the appearance of the more normal sinus rhythm (Figure 2) in contrast to the same height and for the same duration, however, had no effect on the pacemaker focus, the slower atrial focus persisting unmodified. Following 300 mcg. of sublingual nitroglycerin, the patient's cardiac pacemaker focus reverted from the slower atrial rhythm to the sinus focus at a rate of approximately 95 beats per minute.

Fig. 1. Baseline electrocardiogram for Patent No. 1. Note particularly the P-vector in leads II, III, and aVF.
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This rhythm persisted despite alternate right or left carotid massage.

COMMENTS

One could postulate that the patient's normal vagal tone tended to slow his sinus node so that some more excitable atrial focus, probably in the region of the coronary sinus along the right atrial wall, emerged as the dominant pacemaker. Inasmuch as the aberrant pacemaker complex had a P-R interval greater than 0.12 sec., a nonendocardial site would be the most likely choice. Thus, increased vagal activity to the S-A node, as with carotid massage, produced or promulgated the low atrial focus. Stimuli decreasing vagal tone to the sinus node (promoting sinus tachycardia through peripheral vascular dilatation, such as with sublingual nitroglycerin, allowed the normal sinus pacemaker to assume a more rapid rate and overdrive the lower atrial focus.

There did not appear to be a pacemaker focus shift on the basis of altered venous return. If this had been the case, passive leg-raising should have promoted the same change in pacemaker focus that active leg-raising produced. The rate of passive leg-raising was not sufficient to cause an abrupt augmentation of venous return.

J.C.

RESPONSE TO ISOetrics EXERCISE

Fig. 2. Lewis Lead electrocardiogram of Patient No. 1 during a 4-sec. isometric hand-squeezing exercise and recovery. Note that the fourth P-wave after the onset of exercise is upright and remains so for approximately 13 sec. and then returns to the pre-exercise configuration.

J.C.

PASSIVE & ACTIVE LEG RAISING

Fig. 3. Lewis Lead electrocardiogram of Patient No. 1 during passive and active leg-raising while in the recumbent position. Note that passive leg-raising does not alter the pacemaker site, although it does lead to a slight acceleration of the ectopic pacemaker. Active leg-raising, which is a mild form of exercise, produces a shift in pacemaker site and an acceleration of the heart rate similar to that illustrated in Figure 2.
and thereby an acute atrial distention. Such a maneuver might have elicited the so-called Bezold-Jarisch reflex or reflex bradycardia secondary to acute atrial distention.\(^2\)

Inasmuch as patient J.C. was able to accelerate his cardiac rate by a normal sinus mechanism when stimulated to do so by exercise, peripheral vasodilation, or vagal inhibition, and because his atrial escape beats appeared only during a relative sinus bradycardia, his dysrhythmias were considered to be normal variants, requiring no restriction in physical activity.

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**Fig. 4** Baseline electrocardiogram of Patient No. 2. Note the absence of P-waves in lead aVL, and the last complex in aVR. In these same segments the QRS is deformed. See text for further discussion.
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Case No. 2: J.E.

This 36-year-old pilot was referred for evaluation because an annual electrocardiogram was interpreted as showing A-V dissociation with A-V block and abnormal intra-ventricular conduction. At the age of 8 years he had undergone a tonsillectomy and adenoidectomy under general anesthesia. At the age of 18 he had had German measles and been hospitalized at his college dispensary for two nights. That same year he had undergone a herniorrhaphy under spinal anesthesia without complication. At present all of the points in aVr and all of the im points in aVI are broader than those in aVE. Chest examination was unremarkable. The cardiac point of maximal impulse was normal in position and configuration. The first heart sound varied in intensity and the second heart sound was physiologically split. There were no pathologic extra-cardiac sounds, murmurs or gallops. The remainder of his physical examination was unremarkable. All routine laboratory blood studies and roentgenograms were normal.

His routine electrocardiogram (Figure 4) was quite remarkable, however, demonstrating a unique arrhythmia. Curvilinear examination might have failed to disclose it. The last QRS complex in aVr and all of them in aVI are broader than those complexes in the other leads and also show initial notches or slurring. No discernible P-waves precede these QRS complexes and their repolarization is distorted. Except for this transient abnormality the electrocardiogram is quite normal. To decipher this problem further, relatively simple electrocardiographic studies were performed.

Once again, with Lewis and Wilson leads, the patient in the recumbent position performed a standard 10-sec Valsalva maneuver. Phases III and IV (the recovery phases) from his electrocardiogram are shown in Figure 5. The tracing demonstrates a His bundle or peripheral conduction tissue (formerly referred to as a "low nodal") pacemaker rhythm. The onset of the QRS complex is difficult to determine inasmuch as the P-wave coincides with the QRS onset in several complexes. Compare beats 1, 2, and 3 where there is no fusion with beats 4, 7, and 12. Complex 9 produces retrograde depolarization of the atrium manifest by the inverted P-wave in the S-T segment. The QRS duration of the infra A-V nodal beats at their longest is equal to or less than 0.12 sec. and their configuration is quite similar to the beats normally induced by the sinus pacemaker. The nodal rhythm is 78 per minute while the sinus rate varies between 72 and 90 per minute. Thus, when the sinus rate slows below the nodal rate, the nodal focus supervenes as the dominant pacemaker. This competition for pacemaker dominance was also brought out during recovery from mild exercise (Figure 6). The nodal rate persisted at approximately 80 per minute. Exercise however, decreased vagal tone on the S-A node with a resulting acceleration of its rate. As the patient recovered from the exercise, vagal...
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tone slowly increased so that the two pacemaker sites were synchronous—the first complex designated with an asterisk, and the third complex in the second portion of the rhythm strip—the infra A-V nodal focus controlled when the sinus rate slowed to about 70. and then was deposited when the sinus sped to 95 at the end of the rhythm strip.

To further substantiate the physiologic explanation proposed, the patient received 0.3 mg edrophonium chloride intravenously during electrocardiographic monitoring. His response is illustrated in Figure 7. Edrophonium chloride, a cholinesterase inhibitor, potentiates existing or provoked vagal activity. Thus, there was a slowing of both pacemaker sites with exaggeration of the effect from carotid sinus massage. The sinusal rate slowed to approximately 63 per minute, which allowed the infra A-V nodal focus to intermittently escape at a rate of approximately 65 per minute.

The physiologic variables in this patient, therefore, are similar to those in Case No. 1. Inasmuch as no cardiovascular abnormalities were noted, both patients were recommended for continuation of flying duties.

DISCUSSION

The clinical significance of supraventricular arrhythmias is variable. It is strategic that we be able to distinguish the pathologic arrhythmias which could jeopard—

Fig. 6. Wandering pacemaker with fusion beats (designated with *; asterisk) and dominant low nodal rhythm. See text for discussion.

Fig. 7. Sinus bradycardia with wandering pacemaker accentuated with edrophonium chloride and carotid sinus massage in Patient No. 2.
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dize a pilot's life from physiologic variant—arrhythmias with no morbid potential. Certain arrhythmias such as atrial fibrillation, atrial flutter, atrial arrest, and third-degree heart block have been diagnosed arbitrarily as pathologic, either on the basis that they result from some underlying disease or that the arrhythmia itself might compromise cardiovascular function, particularly in a pilot under stress. Other arrhythmias such as sinus arrest, first-degree A-V block, occasional uniform premature ventricular depolarizations, and wandering supraventricular pacemakers, may result from normal physiologic mechanisms without ominous portent. To misinterpret these is to risk not only the unnecessary grounding of healthy personnel but also the production of cardiac neuroses. The two cases herein presented had been diagnosed prior to USAFSAM evaluation as having atrial arrest, third-degree A-V heart block, and intermittent bundle-branch block. In actuality, they both represent wandering pacemakers, the first shifting from sino-atrial node to the atrium, the second shifting from sino-atrial node to the infra A-V nodal region. Monitoring the patient during minor physical or pharmacologic maneuvers frequently will allow more specific differentiation of the rhythm's etiology. Table I summarizes several such maneuvers.

A brief commentary on each of the agents may serve to expedite their clinical application. Edrophonium chloride (Tensilon) potentiates the effect of available acetylcholine. Patients may manifest a variable sensitiv

### TABLE I

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<tr>
<th>AGENT</th>
<th>MODE OF ACTIVITY</th>
<th>INTERMEDIARY RESPONSE OR EFFECT</th>
<th>CARDOVASCULAR RESPONSE</th>
<th>RHYTHM RESPONSE</th>
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<tr>
<td>(1) Edrophonium chloride</td>
<td>Anticholinesterase</td>
<td>Increased vagal effect</td>
<td>Slowing of the sinoatrial node; prolongation of atrial ventricular conduction; slight myocardial depression</td>
<td>Sinus bradycardia atrial and atrioventricular nodal escapes; first- and second-degree heart block</td>
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<td>(2) Carotid massage</td>
<td>Increased carotid sinus nerve activity</td>
<td>Increased vagal firing</td>
<td>and sino-atrial and atrial ventricular nodal arrest</td>
<td>and sino-atrial arrest with nodal escapes and ventricular escapes</td>
</tr>
<tr>
<td>(3) Phenylephrine (Neoephrine)</td>
<td>Increased peripheral resistance; increased blood pressure; with increased carotid sinus nerve activity</td>
<td></td>
<td>and sino-atrial and atrial ventricular nodal arrest</td>
<td></td>
</tr>
<tr>
<td>(4) Amyl nitrite</td>
<td>Decreased peripheral resistance with decreased blood pressure; decreased carotid sinus nerve activity (there may also be an increased venous tone and increased venous return)</td>
<td>Increased sympathetic nervous system tone</td>
<td>Acceleration of sino-atrial nodal escape; decreased A-V conduction time</td>
<td>Sinus tachycardia with suppression of other pacemaker sites</td>
</tr>
<tr>
<td>(5) Atropine sulfate</td>
<td>Blocks receptor site for acetylcholine</td>
<td>Decreased vagal effect</td>
<td>Acceleration of sino-atrial nodal escape; decreased A-V conduction time</td>
<td>Sinus tachycardia with suppression of other pacemaker sites</td>
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<td>(6) Passive leg-raising</td>
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<tr>
<td>(a) Gradual</td>
<td>Increased venous return</td>
<td>Increased right atrial filling</td>
<td>Baroreflex (?)</td>
<td>Slight increase in sinus rate</td>
</tr>
<tr>
<td>(b) Abrupt</td>
<td>Rapid venous return</td>
<td>Abrupt right atrial distension</td>
<td>Bezold-Jarisch reflex</td>
<td>Sino-atrial arrest</td>
</tr>
<tr>
<td>(7) Exercise</td>
<td>Many varied effects</td>
<td>Decreased vagal tone; increased adrenergic tone</td>
<td>As with Amyl nitrite, plus increased irritability</td>
<td>Sinus tachycardia and occasional premature atrial and/or ventricular depolarizations</td>
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may be similarly manipulated. It is worthwhile noting that right carotid stimulation usually has a dominant effect on the sino-atrial node, with only slight effect on the atrio-ventricular node, while left carotid stimulation produces a predominant effect on the atrio-ventricular node with lesser effects on the sino-atrial node. Individual variation in this regard is marked because of the dual innervation of both the SA and AV nodes. Nonetheless, left carotid massage will usually increase atrioventricular block more effectively than right carotid massage.

Phenylephrine (Neoephrine), being predominantly an alpha-adrenergic catecholamine, produces its major cardiac effect via baroreceptor reflexes and vagal cardiac suppression. The magnitude of the response to intravenous phenylephrine is thus related. Considerable caution should be exercised with this drug because of its marked potency. Few situations justify a dose greater than 0.1 mg. Sufficient drug usually is left adhering to the walls of a tuberculin syringe after the syringe is emptied to produce the desired physiologic effects by merely flushing the emptied syringe with the patient's venous blood after a venipuncture. The following procedure is recommended: (1) draw 0.2 cc. of 1% phenylephrine hydrochloride into a 1-cc. syringe; (2) remove needle and syringe from the drug vial and empty the syringe in a waste receptacle; (3) using this now emptied syringe and a new needle, perform venipuncture; (4) allow the syringe to fill with 0.8 cc. of blood; (5) inject the 0.8 cc. of blood back into the vein over a 5- to 10-sec. interval.

Amyl nitrite inhalation, as a diagnostic maneuver, has many attractive advantages. It is relatively innocuous, its effect is transient, and it decreases cardiac work. The patient should be cautioned, however, that he may experience a 1- to 2-min flushing headache and blurred vision.

Atropine sulfate, 0.4 mg. intravenously, will usually provide sufficient vagal blockade to allow deciphering of arrhythmias resulting from neurogenic reflexes. Occasionally, however, a dose of 2.0 mg. is required. The major side effects (which are usually just nuisances) are mydriasis, which may last for two to three hours, and obstipation. Passive and active leg-raising, as well as exercise, are readily employed as the most physiologic maneuvers available to assist arrhythmia analysis.

SUMMARY

Diagnosis and ceromedical evaluation of the significance of supraventricular arrhythmias may frequently be assessed in the Flight Surgeon's Office with the assistance of an electrocardiographic machine, simple physiologic maneuvers, and a few pharmacologic agents, which are herein tabulated and briefly discussed. Two cases are presented in which the rhythm interpretation was facilitated by such studies.

ACKNOWLEDGEMENT

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REFERENCES