Pseudo-Atrial Flutter

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It has long been recognized that the electrocardiogram can be distorted by artifacts which may lead to errors in interpretation. These artifacts can be electrical or mechanical.1 Among the common types of artifacts are skeletal muscle tremors which at times may reach such a magnitude and rhythmicity as to resemble atrial flutter waves.2 The two frequently encountered clinical states that produce prominent muscle tremors are parkinsonism and hyperthyroidism. In most instances, they are easy to recognize and diagnose, because the resulting baseline oscillations are usually irregular in shape, size and rhythm. However, we recently encountered a patient, with spastic right hemiplegia and intermittent clonic contractions of his right arm, whose electrocardiograms showed “pseudo-atrial flutter” waves. The latter were so coarse, regular and rhythmic as to be almost indistinguishable from those of true atrial flutter waves. In fact, on two separate occasions the patient nearly received transcutaneous direct-current countershocks had the nature of these artifacts not been recognized in time.

CASE REPORT

The patient was a 60-year-old man with right hemiplegia consequent to a stroke suffered three years earlier. He entered the Brooklyn Veterans Administration Hospital because of chest pain, hypotension and progressive mental confusion. Electrocardiogram on admission (Fig 1A) showed complete A-V block and acute inferior myocardial infarction. While a pacemaker catheter was being inserted percutaneously through the right femoral vein,3 the patient developed ventricular fibrillation which was promptly terminated by one precordial direct-current countershock. However, instead of sinus P-waves, coarse rhythmic oscillations at a frequency of 400 per minute were present in both the bipolar precordial leads which were connected for purpose of R-wave synchronized D-C shock, and

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FIGURE 1. A, simultaneous electrocardiographic lead I, bipolar precordial lead (BPL) and lead II taken before the onset of ventricular fibrillation, showing complete A-V block with an atrial rate of 120 per minute and ventricular rate of 66 per minute and evidence of acute inferior myocardial infarction. B and C, continuous tracings of bipolar precordial lead (BPL) and lead II obtained immediately following ventricular defibrillation. Note the occasional sinus P-waves identifiable in lead II of B among the “pseudo-atrial flutter” (f) waves. While the P-P interval in B is practically identical to those in A and C, the ventricular rate in B is 66 per minute which is the same as in A and C. Note the gradual decrease in amplitude and rate of the “pseudo-atrial flutter” waves from the beginning of C until their complete disappearance and replacement by clearly discernible sinus P-waves toward the middle of C.
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**Figure 2.** A and B, continuous tracings of bipolar precordial lead. Sinus rhythm with a P-R interval of 0.18 sec and ventricular rate of 80 per minute is present at the start of A. Upon the application of carotid sinus pressure which results in sinus bradycardia toward the middle of A, demand ventricular pacing ensues at a preset rate of 85 per minute. Upon the release of carotid sinus pressure at the start of B, sino-atrial rhythm returns after the third beat followed by a paroxysm of "pseudo-atrial flutter" (f) waves. Note that the ventricular rate remains at 80 per minute in spite of "pseudo-atrial flutter" which has a rate of 400 per minute. C, taken soon after B, shows disappearance of the "pseudo-atrial flutter" waves and reappearance of sinoatrial mechanism at an identical ventricular rate as the non-paced beats in A and B.

lead II (Fig 1B). These oscillations were thought to represent atrial flutter waves with a 6:1 A-V block and a ventricular rate of 66 per minute. While a second precordial direct-current countershock was being considered, the oscillations gradually decreased in amplitude and also somewhat in rate till finally they vanished and were replaced by sinus P-waves (Fig 1C). It was also noted that occasional sinus P-waves were identifiable among the "pseudo-atrial flutter" waves (Fig 1B). They, like the ventricular rate, occurred at the same frequency during the "pseudo-atrial flutter" as before and after its onset.

The electrode catheter was placed in the apex of the right ventricle and demand pacing was accomplished successfully. Soon after establishment of a stable ventricular rhythm the patient's clinical status showed marked improvement. Over the next 96 hours the A-V block progressively decreased from third-degree through second-degree and Wenckebach periods to first-degree until finally normal A-V conduction returned with a P-R interval of 0.18 sec (Fig 2A).

In an attempt to test if the demand pacemaker was functioning properly, carotid sinus pressure was applied. When the sinus rate was slowed down from 80 per minute, ventricular demand pacemaker, which was preset at 65 per minute, assumed complete control of the cardiac rhythm (Fig 2A). When the carotid sinus pressure was released, sinus rhythm returned. However, coarse regular oscillations at a frequency of 400 per minute were again noted (Fig 2B). While cardioversion was again being considered, but temporarily withheld in view of the prior experience, these "pseudo-atrial flutter" waves again disappeared spontaneously (Fig 2C). That these were indeed "pseudoflutter" waves of extracardiac origin due to spasmodic contractions of the right pectoral muscle group was subsequently confirmed by simultaneous recording of lead V1 with II, in which the "pseudo-atrial flutter" waves were prominently displayed only in the former, but not in the latter lead (Fig 3).

**DISCUSSION**

The recognition of "pseudo-atrial flutter" may be
difficult and at times almost impossible. However, its diagnosis can usually be conclusively established by following one or more of these criteria: 1) the baseline oscillations are usually not very regular in rhythm; 2) the deflections are usually not even in size and shape, being sharp at times and blunt at other times; 3) the amplitude and frequency usually wax and wane, producing a concertina-like appearance; 4) their occurrence is usually intermittent; 5) the ventricular rate remains regular and unaffected by the onset of “pseudo-atrial flutter;” 6) whereas both the rhythm of the “pseudo-atrial flutter” waves and the ventricular rhythm are regular, there is no constant relationship between the last oscillation and the next ventricular complex as in the case of a fixed degree of A-V block in true atrial flutter; 7) careful inspection will usually identify sinus P-waves among the “pseudo-atrial flutter” waves; 8) “pseudo-atrial flutter” waves are usually localized to one or two leads with normal sinus P-waves present in the other leads. While “pseudo-atrial flutter” waves usually originate in the right arm and are therefore most pronounced in leads I and II, true atrial flutter waves are usually best seen in leads II and III; 9) “pseudo-atrial flutter” waves should be traceable to the particular group of skeletal muscles responsible for the tremors which are usually grossly visible.

The purpose of this communication is to call attention to this unusual electrocardiographic artifact which, if unrecognized, might lead to diagnostic difficulty followed by erroneous therapeutic decisions that might be detrimental to the patient. Indeed, on two separate occasions, our patient would have received unnecessary and potentially dangerous electrical countershocks had the true nature of the “pseudo-atrial flutter” waves been not recognized in time.

With the routine use of bipolar precordial lead system for monitoring cardiac patients in the modern coronary care units, this potential source of artifact should always be kept in mind. Our patient manifested “pseudo-atrial flutter” waves in both the bipolar precordial lead and limb lead II, because the involuntary muscle tremors affected the right arm, particularly the pectoral muscle group.

It is interesting to note that the first paroxysm of “pseudo-atrial flutter” in our patient followed a precordial direct-current countershock of 200 wattseconds delivered for ventricular defibrillation and his second paroxysm occurred after the application of carotid sinus pressure. Both of these maneuvers apparently constitute a powerful stimulus to evoke an exaggerated response with resultant intensification of the clonic tremors of his hemiplegic right arm, particularly the right pectoral muscles, to result in appearance of these “pseudo-atrial flutter” waves which might otherwise be unnoticeable in his electrocardiogram.

**References**


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**ANNOUNCEMENT**

The Department of Otolaryngology of the Eye and Ear Infirmary, University of Illinois Hospital and the College of Medicine of the University of Illinois at the Medical Center, will conduct a Postgraduate Course in Laryngology and Bronchoesophagology, April 6-17, 1970. The course is limited to 15 physicians and will be under the direction of Paul H. Hollinger, M.D. The course will take place mainly at the Eye and Ear Infirmary, 1855 West Taylor Street, Chicago, and will include visits to a number of other Chicago hospitals. Instruction will be provided by means of animal demonstrations and practice in bronchoscopy and esophagoscopy, diagnostic and surgical clinics, as well as didactic lectures. Interested registrants should write: Department of Otolaryngology, College of Medicine, University of Illinois at the Medical Center, PO Box 6998, Chicago, Illinois 60680.