The other remarkable observation easily answers these 2 questions: “No”. This observation is that this rapid electrical activity is present in varying degrees in the 12 leads of this ECG. The largest amplitudes are in the limb leads, with much lower voltages in the chest leads; and in lead II there is almost nothing (Figure 2). If we were analyzing a single lead ECG monitor with lead II alone, we would have little hesitation in saying that there is probable sinus rhythm at around 75 bpm, with a normal PR interval of 180ms (4 ½ small blocks) and a normal 1:1 AV relationship. Normal atria cannot sustain 2 different rhythms or activities simultaneously. If there is sinus rhythm, there cannot in the same atria be atrial fibrillation or flutter.

Thus, the fast electrical activity is clearly unrelated to the QRS and P waves and independent of atrial and ventricular action. (This patient had never had previous cardiac surgery. If she had had a heart transplant, it is possible that the new heart could be in one rhythm, e.g. sinus, and the remnant of the recipient old heart’s atria could generate another rhythm, such as an atrial tachycardia. This does occur but is rare and is difficult to spot because the remnant atria cannot generate such high voltage activity as is seen in this patient.)

**Diagnosis and discussion**

The ECG diagnosis is, therefore, 4 and 5, i.e. not atrial fibrillation / flutter or heart block but sinus rhythm with other electrical activity.

**General Comments**

Before deciding on the correct treatment, an ECG diagnosis needs to be made. A quick glance at the ECG may suggest possible diagnoses that may need to be considered:

1. Atrial flutter
2. Atrial fibrillation
3. Complete heart block (with a junctional escape rhythm)
4. Sinus rhythm
5. Other electrical activity

**Observations**

There are 2 most striking observations on examining this ECG. The first is the evidence of rapid electrical activity. This activity does not involve the QRS complexes which are narrow (i.e. using a normal ventricular conduction system), regular and at 84 per minute. This may result in the suspicion that the rapid activity represents some kind of supraventricular tachycardia. This rapid activity is almost but not absolutely regular as well seen in the middle of the aVL recording with “cycle length” intervals varying from 200ms (5 small blocks: equivalent to a rate of 300 per minute) to 160ms (4 small blocks: equivalent to a rate of 375 per minute) and with more-or-less monomorphic appearance (Figure 1). Could this therefore be some kind of atrial flutter, in which typically the atrial rate is around 250-350 per minute?

Analysis of the V1 rhythm strip shows that the rapid electrical activity is not associated with the ventricular activity, i.e. it appears the ventricles are independent of this activity. So is it complete heart block?

**FIGURE 1:** Lead aVL shows the slight irregularity of the “cycle length” of the rapid electrical activity varying from 4-5 small blocks or 160 to almost 200ms (equivalent to 300-375 per minute or around 5-6 per second).

**FIGURE 2:** Lead aVL shows the slight irregularity of the “cycle length” of the rapid electrical activity varying from 4-5 small blocks or 160 to almost 200ms (equivalent to 300-375 per minute or around 5-6 per second).
What is the differential diagnosis of this dissociated electrical activity that happens to be recorded on the same ECG as the underlying normally conducted sinus rhythm?

1. Intrinsic, i.e. in the patient
2. Extrinsic: outside electrical interference, e.g. electrical equipment:
   a. drip rate controllers,
   b. ripple bed mattresses. (This patient was ambulant and not connected to any electrical machinery that could generate these artifacts.)

Regarding the intrinsic electrical activity, only muscle can produce such relatively high voltage signals:

1. Cardiac muscle
2. Skeletal muscle myopotentials.

The “cycle length” of the electrical activity was noted above to be slightly variable, 4-5 little blocks, or 160-200ms, which translates to 5-6.25 Hz, i.e. around 5-6 actions per second. This is compatible with a muscle tremor, as in this case of Parkinson’s. This ECG was from such a patient who had an incidental ECG recording. Parkinsonian tremor is usually between 3 and 7 Hz. So this fits. The slight variability is also suggestive that this is not some electrical machine but rather from the patient.

Why are these skeletal myopotentials not obvious in the lead II and less in the lateral chest leads? There are 2 reasons:

- It is not unusual for Parkinson’s to be unequal on both sides or sometimes unilateral and for the upper limbs to be affected more than the lower limbs.
- Also the limbs are more affected than the trunk.

This patient had Parkinson’s predominantly on the right, hence the right sided limb leads have greatest tremor-related amplitude. The chest leads have much less because the limbs are more affected and the left lateral leads are further away from the right limbs. Lead II, which is the lead between the right arm and left leg, is probably also at right angles to the “axis” of the myopotentials which would also reduce the size.

**Conclusion**

The ECG shows normally conducted sinus rhythm with superimposed electrical / myopotential artifact arising from skeletal muscle tremor of Parkinson’s disease. No cardiac treatment is needed. Therefore, the correct answer is (c) levodopa, an anti-parkinsonian agent.