

# Tachy-Brady Like Phenomena and Autonomic Instability in Critically Ill SARS-CoV2 Patients: A Case Series

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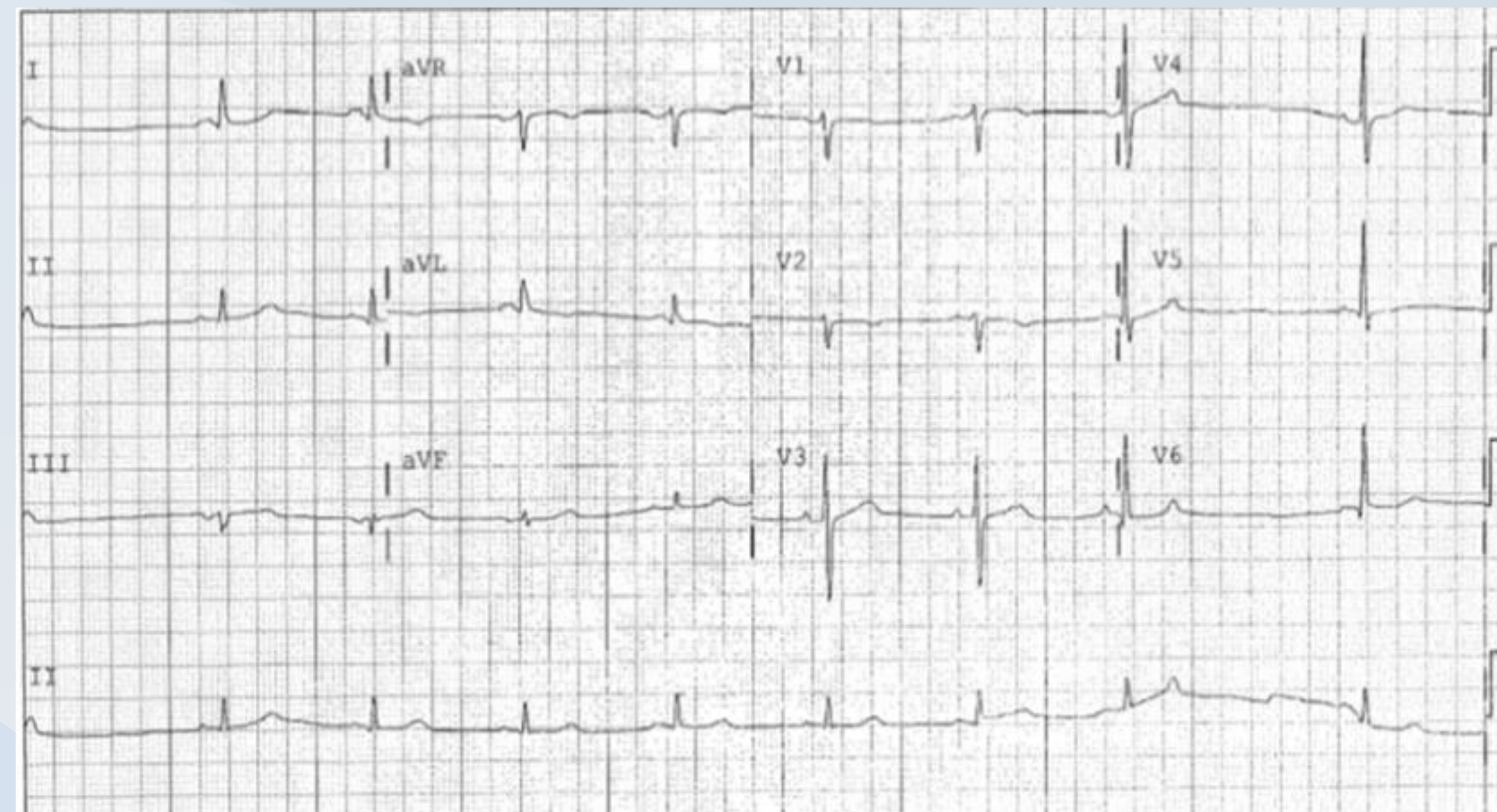
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## INTRODUCTION

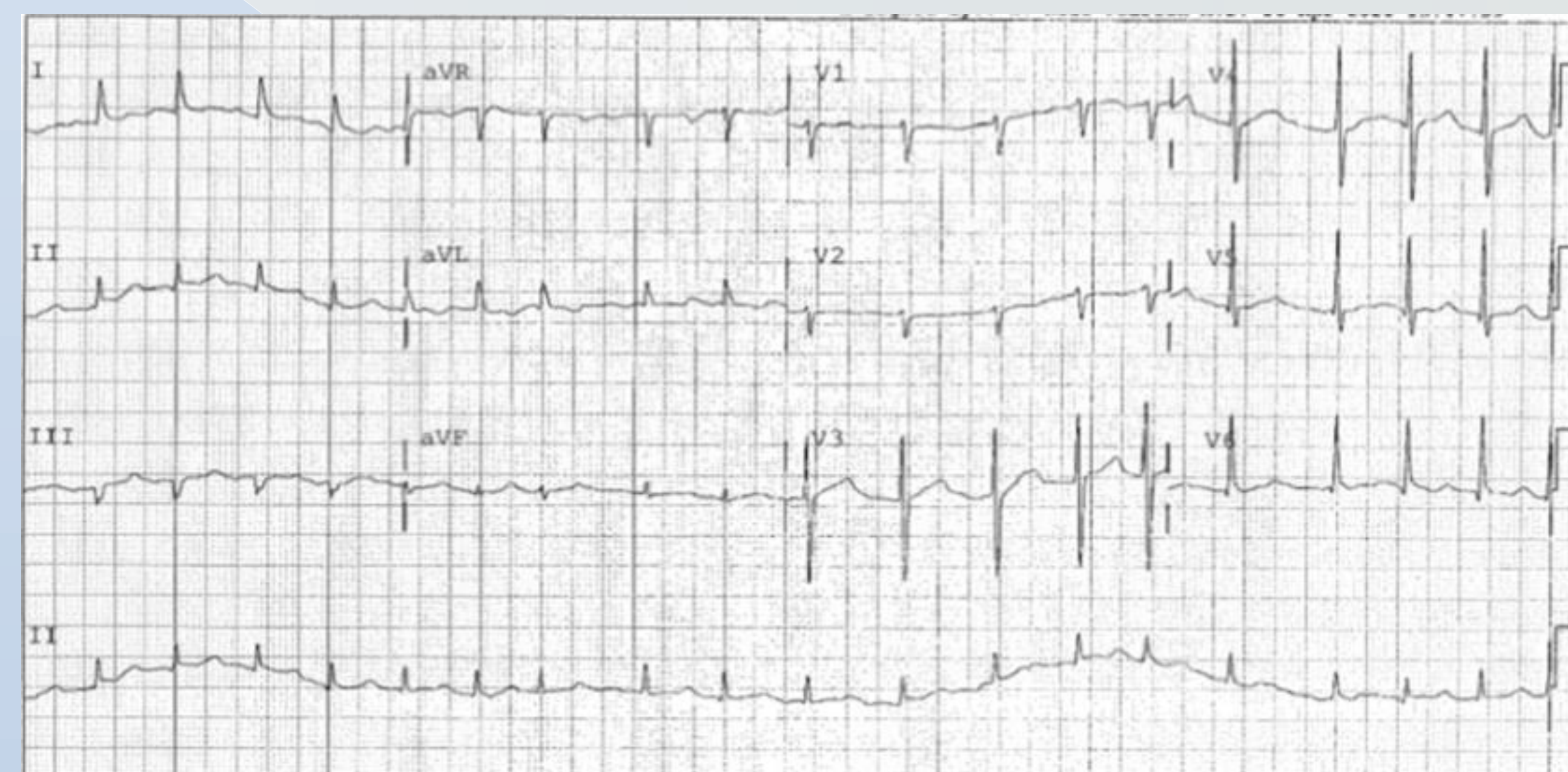
- SARS-COV2 can cause a variety of organ damage including the heart: dysrhythmias
- No reports so far of tachy-brady like phenomena as form of arrhythmia manifestation

## CASE DESCRIPTION

- Case 1: 70 year-old man with history of CAD post-stent, AAA post-repair, atrial fibrillation and hypertension
- Presented with fever and acute respiratory failure
- Rapid atrial fibrillation with intermittent sinus bradycardia-sinus rhythm was recorded on day 1-19
- Treated primarily with amiodarone due to hypotensive and bradycardic episodes
- Echocardiogram showed an ejection fraction (EF) of 60% and troponin mildly elevated (0.20)
- Discontinuing sedation did not alter the bradycardia
- Eventually discharged to long term facility
- Case 2: 80 year-old man with history of aortic insufficiency and hypertension
- Presented with acute respiratory failure
- Day 1 he had alternating sinus tachycardia and bradycardia thought to be related to respiratory distress
- Discontinuation of fentanyl did not resolve bradycardia
- Patient had new-onset rapid atrial fibrillation with conversion to sinus rhythm and sinus bradycardia after diltiazem use on day 5
- Troponin not elevated (0.05)
- Eventually passed away after compassionate extubation



Patient 1. SA node exit block (above) and rapid a-fib (below)



Patient 2. sinus bradycardia (above) and rapid a-fib (below)



## DISCUSSION

- Myocarditis is a possible mechanism but a difficult diagnosis to establish without meeting specific biopsy-based criteria.
- Another possible mechanism is the angiotensin-converting enzyme-2 (ACE-2)-mediated cellular entry of SARS-CoV-2, which downregulates ACE-2 expression. This results in reduced degradation of angiotensin II
- In the presence of a hyper-inflammatory state, hypoxia, and oxidative stress, cardiac arrhythmias can develop.
- In both our patients, arrhythmias occurred in the setting of hyperinflammation as evidenced by increased inflammatory markers that could have caused cardiac stress, injury, or myocarditis resulting in increased troponin levels.
- In hyperinflammatory phase or cytokine storm, IL-6 seems to play as the central mediator of cytokine storm where it organizes the cascade of inflammation

## REFERENCES

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