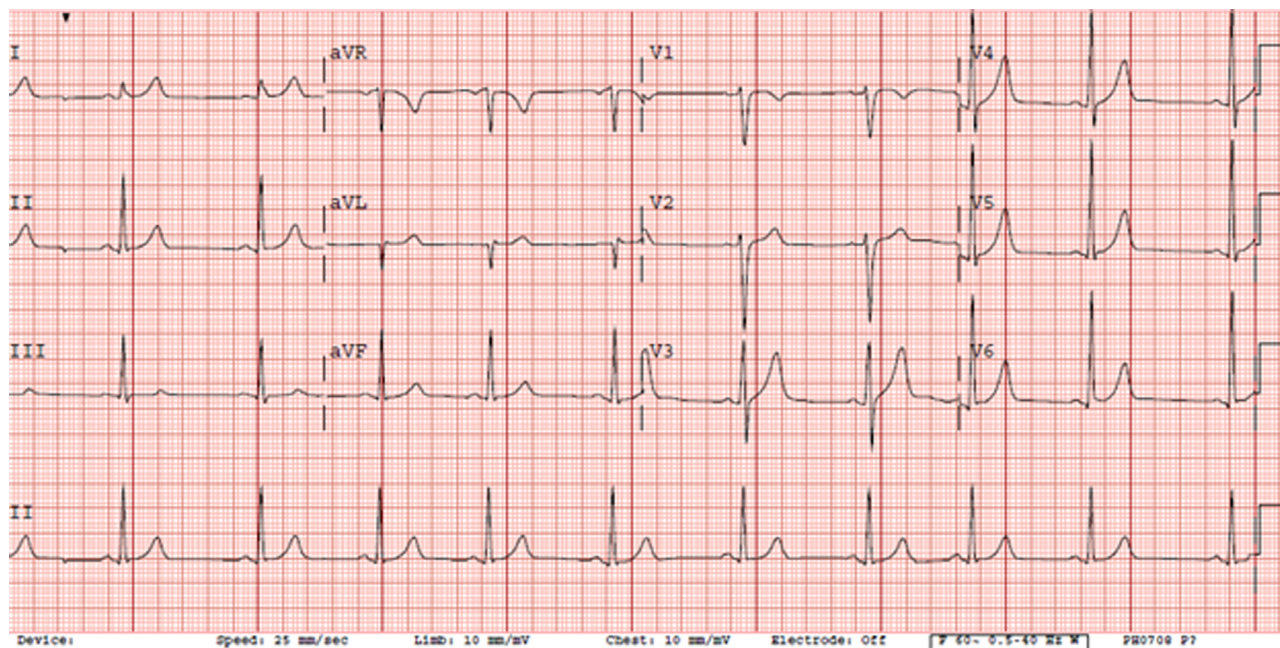


# Recurrent commotio cordis: Déjà vu



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**Figure 1** A 12-lead electrocardiogram of the patient after the second episode of chest trauma-induced syncope.

## Introduction

Commotio cordis is defined as sudden cardiac death caused by a chest blow to the anterior chest. Events are predominantly reported in youth sports of baseball, lacrosse and hockey when the ball or puck strikes the chest. But commotio cordis can occur with other relatively innocent blows to the anterior chest wall, including fists, elbows and other objects. The mechanism of commotio cordis is ventricular fibrillation induced by an appropriately timed strike on the upslope of the T-wave.

## Case study

While practicing for baseball, a 14-year-old male was hit in the mid-sternum by a tennis ball filled with coins. This tennis

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ball was estimated to be 3 times the weight of a standard baseball, and was being thrown for strength training. He immediately lost his breath and went down to a knee. Within a time frame of a few seconds he lost consciousness. The coach observed him to be unresponsive and turn blue. As cardiopulmonary resuscitation was beginning, he awoke. He complained of lightheadedness for 15 to 20 additional seconds and then felt completely back to normal. An evaluation in the emergency department showed a normal physical examination, blood pressure, electrocardiogram (Figure 1), and echocardiogram.

A year earlier, a similar event had occurred. In school, the patient was punched in the mid-chest by a friend. The force of the blow was not extreme. As the event on the baseball field, he gasped for breath and then lost consciousness. He fell and hit his head on a desk. According to his classmates, he was not breathing and was “white as a ghost.” After 10 to 15 seconds, he awoke and had lightheadedness for another minute. He did not seek medical attention for this episode.

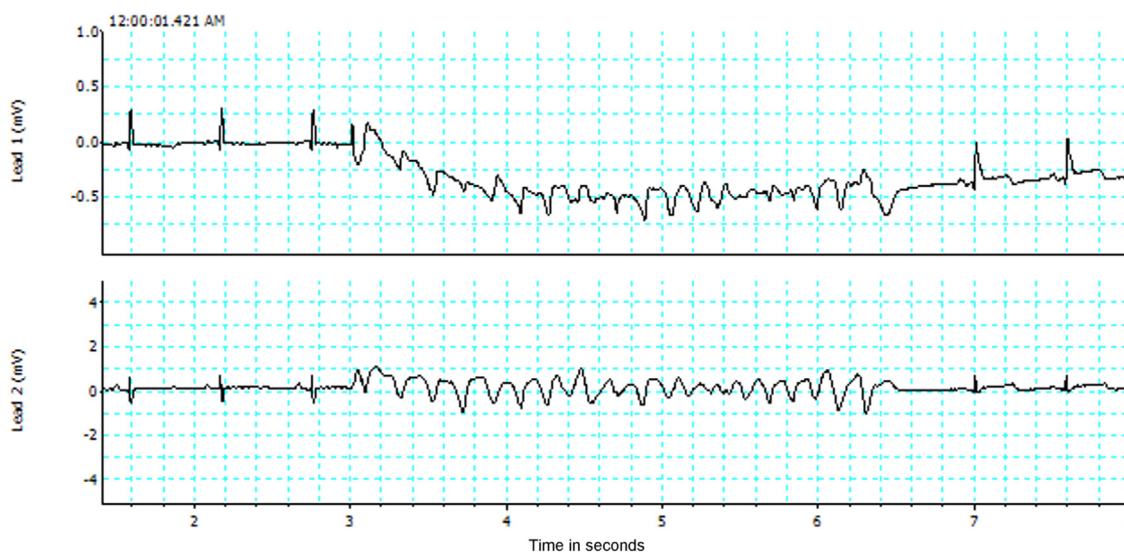
## KEY TEACHING POINTS

- Individuals who are struck in the chest and subsequently collapse should be suspected to be in ventricular fibrillation, and appropriate resuscitation should begin.
- Rare individuals may be uniquely susceptible to commotio cordis.
- Both nonsustained ventricular fibrillation and transient heart block are observed in an experimental model of commotio cordis, but transient heart block is typically seen with higher-velocity impacts that cause structural damage.

The patient had no other episodes of collapse or syncope. He had the normal childhood illnesses and no other medical

history. There was no family history of sudden cardiac death. The patient was healthy and active. Other than the 2 episodes, he had no abnormal cardiac symptoms. He was on no medication.

Given the circumstances of his syncope, it is likely that he had either a short episode of nonsustained ventricular fibrillation or transient heart block.<sup>1</sup> Both arrhythmias have been observed in our commotio cordis animal model.<sup>2,3</sup> However, transient heart block is predominantly observed with impact velocities of 50 mph or greater in our model; impact velocities this great frequently caused cardiac structural damage, including myocardial rupture and papillary muscle tears.<sup>4</sup> Thus, we believe it is more likely that the arrhythmia induced in this individual was transient ventricular fibrillation. In our model, up to 10% of impacts in the vulnerable time window cause nonsustained ventricular fibrillation (Figure 2).<sup>5,6</sup> In addition, and relevant to this case, in our commotio cordis animal model, individual susceptibility has been observed.<sup>7</sup> A small



**Figure 2** Nonsustained polymorphic ventricular tachycardia in an experimental model of commotio cordis. In this animal, a lacrosse ball striking a swine at 40 mph during the upslope of the T-wave caused ventricular fibrillation, which terminated after 3 seconds.

percentage of the swine are uniquely susceptible to ventricular fibrillation with chest wall impact.

In conclusion, given the circumstances of the 2 events in this individual, separated by 1 year, we suspect that nonsustained ventricular fibrillation caused his 2 collapses, and in addition, that he is an individual susceptible to commotio cordis.

## References

1. Maron BJ, Estes NA 3rd. Commotio cordis. *N Engl J Med* 2010;362:917–927.
2. Link MS, Wang PJ, Pandian NG, Bharati S, Udelson JE, Lee MY, Vecchiotti MA, VanderBrink BA, Mirra G, Maron BJ, Estes NA 3rd. An experimental model of sudden death due to low-energy chest-wall impact (commotio cordis). *N Engl J Med* 1998;338:1805–1811.
3. Link MS. Commotio cordis: ventricular fibrillation triggered by chest impact-induced abnormalities in repolarization. *Circ Arrhythm Electrophysiol* 2012;5:425–432.
4. Link MS, Maron BJ, Wang PJ, VanderBrink BA, Zhu W, Estes NA 3rd. Upper and lower limits of vulnerability to sudden arrhythmic death with chest-wall impact (commotio cordis). *J Am Coll Cardiol* 2003;41:99–104.
5. Link MS. Mechanically induced sudden death in chest wall impact (commotio cordis). *Prog Biophys Mol Biol* 2003;82:175–186.
6. Link MS, Estes NA 3rd. Mechanically induced ventricular fibrillation (commotio cordis). *Heart Rhythm* 2007;4:529–532.
7. Alsheikh-Ali AA, Madias C, Supran S, Link MS. Marked variability in susceptibility to ventricular fibrillation in an experimental commotio cordis model. *Circulation* 2010;122:2499–2504.