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### Which one is more important in traumatic brain injury: Hypotension or hypoxia?



I have greatly enjoyed reading the recently published article by Seo et al. [1]. In this study, the authors examined the association between hypoxia level and outcomes according to shock status in traumatic brain injury (TBI) patients. They found that the mortality rates were 49.4% in severe hypoxia, 30.7% in mild hypoxia, 18.5% in normoxia. Mortality rates were 47.1% in TBI patients with shock status and 20.5% in non-shock TBI patients. There was a trend toward worsened outcomes with mild and severe hypoxia in patient with and without shock, however, the only met statistical significance for patients with both severe hypoxia and non-shock status. These results suggested that, in patients already suffering hypotension, hypoxia did not add any significant effect.

Previous studies have revealed that hypotension (systolic blood pressure < 90 mmHg) and hypoxemia ( $P_aO_2 < 60$  mmHg) are important prognostic factors and should be avoided in patients with TBI [2]. However, there are no data in the literature which one is the more important factor (hypoxia or hypotension) influencing the prognosis. The study by Seo et al. gives rise to thought that hypotension is more important than hypoxia. However, in another study, Spaite and colleagues showed that had mortality rate was 20.7% in hypotensive patients, and 28.1% in hypoxic patients suggesting hypoxia as more important risk factor for TBI patients [3]. Therefore I think that further studies are needed to reveal whether hypotension or hypoxia is more important in patients with TBI.

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### Diagnostic considerations in detecting apical hypertrophic cardiomyopathy while utilizing point-of-care ultrasound



The emergency department (ED) clinical approach to patients with unexplained syncope has ushered in an era of advances in the point-of-care ultrasound (POCUS) practice. Cardiac ultrasound (US) is a key application that is often utilized in young patients with syncope when screening for structural abnormalities such as hypertrophic cardiomyopathy (HCM). The application of the cardiac US for this screening,

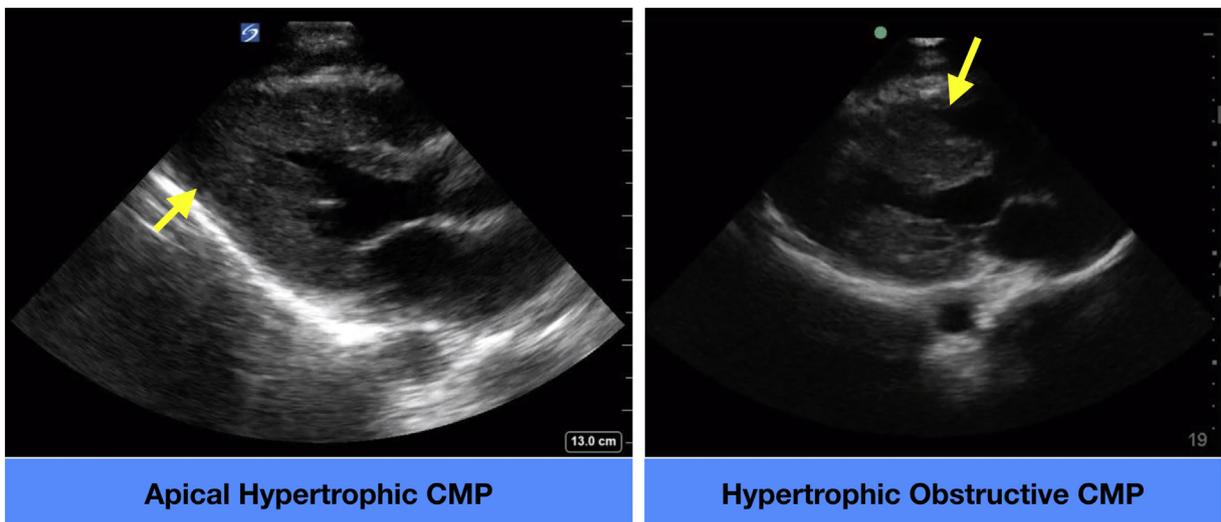
however, can be hindered by phenotypic variability of hereditary HCM. The apical variant of HCM constitutes a minority of all cases (<3%) and is generally associated with a benign prognosis. We describe a 28-year-old woman and former collegiate middle-distance/endurance runner who presented to the emergency department (ED) with syncope. Earlier in the day, she was one mile into a planned ten-mile run, when she reached an intersection requiring her to stop. Upon stopping abruptly, she experienced a feeling of warmth, followed by shortness of breath, blurred vision and subsequent loss of consciousness. The patient had syncopized several times before in her life—all in the setting of abruptly stopping after a warm-up exercise. She denied any other complaints and had no other relevant past medical history. Family history was notable for myocardial infarction (MI) in the patient’s father while he was in his 50s, as well as sudden cardiac death of her paternal grandfather while he was also in his 50s. Upon arrival to the ED, blood pressure was 93/59 mm Hg; heart rate, 59 beats per minute; oxygen saturation, 99% on room air, and temperature 98.4 F. On examination, the patient was well appearing, alert, oriented and in no acute distress. Orthostasis was not present. Breath sounds were clear and equal bilaterally. Cardiac examination demonstrated regular rhythm without murmur, rub or gallop. Distal pulses were intact and jugular venous distension was absent.

Electrocardiogram (ECG) was notable for sinus bradycardia with deep and symmetric T-wave inversions in leads I, II, III, aVF, V3–6, as well as ST depressions in leads V3–6. The R waves were also very prominent in leads II, III, aVF, V3–6. These findings were consistent with left ventricular hypertrophy (LVH) with strain pattern. Point-of-care cardiac ultrasound (US) demonstrated no pericardial effusion and grossly normal ejection fraction without obvious evidence of segmental subaortic septal hypertrophy or LV outflow obstruction.

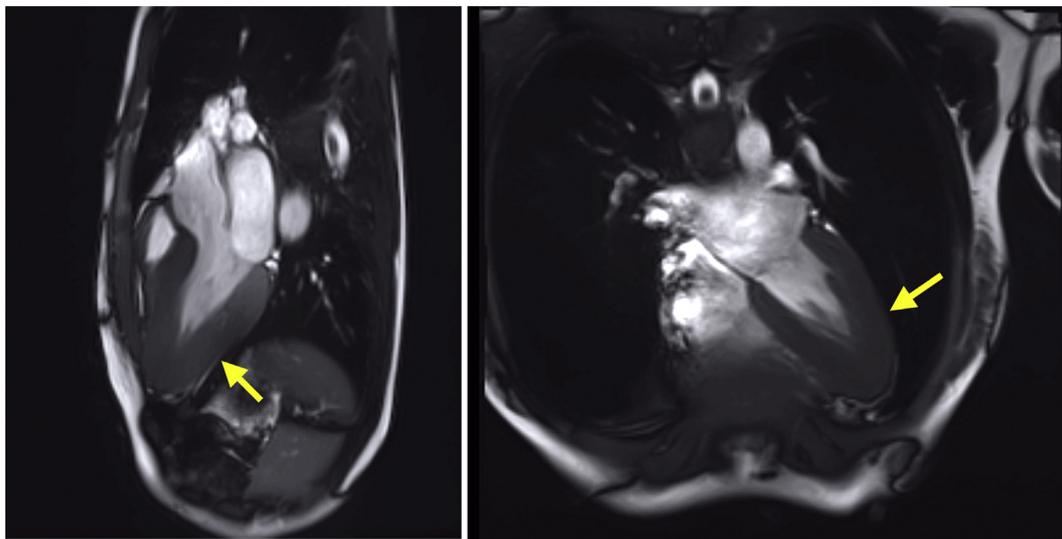
Inpatient workup included a comprehensive echocardiogram, which demonstrated near-oblivation of the left ventricular apical cavity at end systole and prominent LVH in the apex measuring 11–13 mm, with a “spade” shaped left ventricle, without evidence of outflow obstruction (Fig. 1). The patient underwent stress testing with an exercise capacity of 17 metabolic equivalents of task, with an ECG that was non-diagnostic for ischemia secondary to a baseline LVH, but showing no arrhythmias or ectopic beats during exercise or recovery. The patient also underwent cardiac magnetic resonance imaging at rest, including conventional structural and functional imaging, which showed concentric thickening of the apical segments of the left ventricle (LV) with maximal wall thickness of 17 mm and associated mid-myocardial late gadolinium enhancement involving 6% of the left ventricular myocardium—establishing the diagnosis of apical HCM (AHCM) (Fig. 2).

Patients with HCM exhibit a variable phenotype with LV hypertrophy being the main manifestation, and diastolic dysfunction and dynamic LV outflow tract obstruction as important pathophysiologic features. The diagnosis is confirmed when thickening  $\geq 15$  mm is noted anywhere on the LV wall during end diastole [1]. Wall thickening is frequently asymmetric, and most commonly involves the basal septum, just below the aortic valve, leading to LVOT obstruction. It is important to note, however, that our patient exhibits AHCM, a rare variant of HCM (<3%) involving solely the apex of the LV [2,3]. In AHCM, transthoracic echocardiography (TTE) will demonstrate hypertrophy of the LV apex and a spade-like left ventricular cavity during systole when aided by intravenous echo-contrast material [2,4]. Cardiac US that focuses solely on LV outflow and basal septal thickening conveys a possibility of false negative diagnosis. Careful assessment of the entire LV including the apex may preclude this pitfall.

Studies have generally indicated a benign prognosis for individuals with AHCM. Nevertheless, there have been case reports of patients with AHCM developing potentially serious arrhythmias including atrial fibrillation, supraventricular tachycardia and ventricular tachycardia [1–5]. Notably, all of these reports have been made in individuals over the age of 50. In the case of our relatively young and exceptionally healthy patient, it is unlikely syncope was secondary to outflow obstruction,



**Fig. 1.** Cardiac ultrasound image of our patient with AHCM (left) revealed LV apical hypertrophy that depicts a spade-shape feature of the LV. In the right image we present a cardiac ultrasound of a patient with hypertrophic CMP that shows subaortic focal hypertrophy.



**Fig. 2.** Supplementary Data: Coronal (Video1) and sagittal (Video 2) view of cardiac MRI with gadolinium enhancement demonstrates a spade-like feature of the left ventricle with focal apical hypertrophy, confirming the diagnosis of AHCM.

but rather an arrhythmia, although this was never confirmed during her hospitalization.

In 10% to 20% of patients with AHCM, an apical aneurysm has been reported, which is associated with a higher adverse outcome. In the majority of these cases, this finding is not visible on echocardiography. Therefore, a cardiac MRI should be considered in patients with suspected apical HCM to confirm the diagnosis and to ascertain the presence of aneurysms [6]. In our case, the MRI revealed an underdiagnosis in the severity of LV wall thickness, but no apical aneurysm.

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#### Conflicts of interest

None.

#### Appendix A. Supplementary data

Supplementary data to this article can be found online at <https://doi.org/10.1016/j.ajem.2019.01.057>.

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## Ultrasound and optic neuritis



Dear Editor,

We were really interested in the paper by Yee et al. concerning four patients with optic neuritis, diagnosed with ultrasound [1].

The possibility to detect optic nerve sheath diameter (ONSD) increase in case of optic neuritis or intracranial hypertension has been widely proven [2] but we would like to comment on the way the measurements have been taken in this case report.

We will avoid to comment on the use of B scan for such measurement, which is now well known to be subjective and not very reliable due to the so called blooming effect [3–7] and how better is the A scan technique for this purpose [8–13].

We would like to put our attention on the table that have been published, which proves how important is the skill and the knowledge of ocular and orbital anatomy to make safe and repeatable diagnoses [14,15].

In the introduction, the authors stated that “in order to measure the optic nerve sheath on-axis, the patients were instructed to look straight ahead and the ultrasound image view was verified to include the lens or iris; this was performed to prevent the possibility of measuring the optic nerve at an angle off midline”.

In the table shown in the paper, only in the left eye of Case #1 the lens is visible in the scan, proving, in contrast with what the authors have stated, that all the other pictures are off-axis.

Moreover, in our opinion, to include the lens in the image is correct but not sufficient, as the scanned plane should not only pass through the lens, but also through the optic nerve (ON) insertion. In this way, we are not only sure to be on axis, but we can also have reference points to measure the ONSD at the same 3 mm distance from the ON insertion.

Unfortunately, the ON insertion is only visible in the right eye of patient #3 and in the left eyes of patients #1 and #3. In patients #1 and #2, the images of the two eyes have been taken with different setting, making us wondering if the difference could have been due to the blooming effect. Furthermore, in patient #2, the distance where the measurements have been taken is not shown.

In addition, in patient #4, the distance has not been taken at the center of the optic nerve, but laterally and, as the posterior wall of the eye is concave, the distance is obviously inferior to the one that would have been measured if it was put correctly at the center of the optic nerve.

In conclusion, we would like to remark that echography could look simple but, as all the other techniques, it requires skill and knowledge of the anatomy to get trustworthy and reproducible results [16,17].

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