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Carotid ultrasound is not helpful in simple syncope

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Clinical Context
51 year old Caucasian male who presented to the ED with complaints of loss of consciousness that was witnessed by his wife one day prior. The patient had finished kayaking and shortly after felt lightheaded and went to sit down. This was followed by a self-limited, syncopal episode lasting a few seconds, witnessed by his wife. He states he did not have any particular symptoms prior to this episode, including ringing in the ears, headache, throbbing or nausea. Pertinent negatives included neurological symptoms, tonic or clonic activity, bowel/bladder incontinence, tongue biting and rolling of the eyes. Denied any aggravating or alleviating causes. He had no prior similar episodes. He thought this may have been caused by hot weather and lack of proper fluid intake.

The patient was in good physical condition, ran 10-20 miles per week and participated in marathons. Earlier that day after his 3 mile run, he was at a family barbecue with his family and had alcoholic beverages instead of other hydration solutions.

Examination was unremarkable. Pertinent negatives included focal neurological signs and carotid bruit.

Clinical Question
In patients with syncope and no neurological signs or symptoms, does carotid ultrasound help to determine the underlying etiology?

Research Article

Literature Review
We started our literature search in PubMed and Google Scholar using "syncope", "carotid ultrasonography" and "diagnosis." We also reviewed references in review articles about evaluation of syncope that were contained in American Family Physician, UpToDate and DynaMed. For each article that was found, we reviewed the references looking for more primary research studies that addressed

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our clinical question. In an iterative search using key words “carotid artery”, “outcome and process assessment”, “syncope”, “stenosis” and “fainting”, no additional studies were found. We also reviewed recent syncope evaluation guidelines (from the American Heart Association/American College of Cardiology Foundation), National Institute for Health and Clinical Excellence, European Society of Cardiology and Canadian Cardiovascular Society) to see if they recommended carotid ultrasound.

We found seven papers describing original research which evaluates the use of carotid ultrasound in patients with syncope. All papers were retrospective chart reviews. Beyond the retrospective nature of the studies, many of them had other flaws or study characteristics which made the result less applicable to our patient. Many of the studies did not exclude patients with high-risk history or patients with focal neurological findings. The Vanbrabant study evaluated many different diagnostic tests, and only 23 of their 117 study patients received carotid ultrasound. Some studies evaluated only for diagnostic yield and not for changes in therapy, which was felt to be less patient-oriented. Only the Schnipper study investigated the risk of subsequent syncope or cerebrovascular accident, but only did so in patients who had significant findings on ultrasound. Only 18 patients were evaluated in this manner and there was no control group.

In our review of the most current guidelines described above, none recommended ultrasound in patients without focal neurological deficit or carotid artery bruit.

All of the primary research in this literature base share an important logical flaw. While studying the diagnostic value of carotid ultrasound, they assume that isolated carotid artery stenosis does not cause syncope unless severe. Furthermore, they assume that carotid artery stenosis severe enough to cause syncope would also cause focal neurological deficits. If these things are assumed to be true, then there can be no diagnostic utility to performing an ultrasound in patients without focal deficit. By definition, any abnormality in these patients would be an incidental finding. It would be impossible to construct a 2 × 2 table or calculate a sensitivity or specificity.

These assumptions appear to be well accepted, though not rooted in empiric evidence. A brief literature review did not reveal any primary research investigating a connection between carotid artery stenosis and syncope. Carotid artery stenosis as a potential etiology for syncope is not mentioned in several prominent medical texts or review articles. It is mentioned as a possibility in the case of compression of a carotid artery contralateral to a completely obstructed carotid, but there is no reference given. We found no instances of a medical text or reference resource that refuted these assumptions.

Ultimately, we chose the Scott study because it studied therapeutic outcomes and looked specifically at low-risk patients without focal neurological findings or carotid bruits, which was most specific for our patient.

**Critical Appraisal**

This is a two-part study. The first part aims to investigate the prevalence and cost-effectiveness of this diagnostic examination in a population based analysis. We have focused more on the second part of the study, a retrospective case review in which investigators examined individual medical records. As a retrospective case review study, this is a SORT level 3 study.

A sample of charts were taken from a large academic medical center. A total of 313 charts were gathered and analyzed. Inclusion criteria were cases in which ultrasounds were ordered for a provider-specified indication of syncope. Excluded were cases in which there was a documented focal neurological finding or carotid bruit. These are appropriate exclusion criteria, since these findings more strongly indicate a need for evaluation of the carotid arteries. The last exclusion criteria was ultrasound studies ordered in patients who did not have documented syncope upon chart review, or had other indications for the evaluation of the carotid arteries (e.g. preoperative evaluation for cardiac surgery).

The investigators conducted a detailed review of the chart that included outpatient clinic notes, outpatient consultant notes, hospital admission and consultation notes and hospital discharge summaries. For each case, the two notes immediately prior to the study were reviewed, in addition to the two notes immediately following the study or after discharge. A description of desired outcomes and how the reviewers defined how they decided on each outcome was adequate. However, details about other parts of the chart review process were not described completely. Specifically, there is no discussion about the number of reviewers who analyzed each case and how disputes were solved.
Outcomes of interest included the prevalence and severity of carotid artery stenosis, changes in medical or surgical management for stroke risk reduction, referrals to vascular surgery and attribution of syncopal events to the ultrasound examination results. In addition, information on age, sex and other risk factors for carotid artery stenosis were collected. The Wilcoxon rank-sum test was used for statistical analysis of outcomes.

After analysis, 48 (15.3%) out of 313 patients had stenosis ≥ 50% on ultrasound. In only 7 (2.2%) of these patients did the provider change medical management after the ultrasound. Only 1 (0.3%) patient ended up receiving a carotid artery stent, and there were no cases in which the patient received endarterectomy. Interestingly, the consultation note noted "asymptomatic carotid disease" as the indication for stenting. In no cases did the provider attribute the syncopal event to carotid artery disease or the results of ultrasound examination.

As a retrospective case review investigating only 313 patients, this is not a particularly strong study. The investigators only reviewed cases in which ultrasound was performed; there was no control group with which to compare. Investigating which ultrasounds resulted in changes in management was a more patient-oriented outcome than found in other studies, but the risk of recurrent cerebrovascular accidents would have been a more clinically relevant outcome. As in the rest of the literature, it is assumed that in the absence of focal neurological signs or carotid bruit, carotid artery stenosis is not a cause of syncope. To some degree, the authors have begged the question, but investigating changes in therapy and attribution help them to mitigate this logical flaw. It allows them to suggest that even if simple syncope is caused by carotid artery stenosis, performing carotid ultrasound very rarely causes significant changes in therapy and therefore is a low-yield diagnostic test.

Despite these flaws, it is the most appropriate study to answer this questions. It is the largest study of its kind. In addition, it is the only study which excludes patients who had focal neurological signs or carotid artery bruit, allowing it to focus on a specific clinical scenario described in the Clinical Context.

### Clinical Application

Because this patient was an otherwise healthy gentleman without any indication of severe carotid stenosis (focal neurological signs or symptoms) and without carotid bruit, we felt it was unlikely that carotid artery atherosclerotic disease would have caused his syncope.

All of the evidence available at this time is retrospective in nature. Every study we found has noted that carotid ultrasound was unlikely to help clinicians arrive at a diagnosis or assist them with management decisions. This was the case even in studies which investigated patients who are at higher risk of atherosclerotic disease than ours. While the evidence discouraging the use of carotid ultrasound is not of the highest quality, we could find no evidence of any quality that would suggest that carotid ultrasound is an effective diagnostic tool for patients presenting similarly to ours. We cannot find a compelling reason to order ultrasound and question why it is still done so frequently in low-risk patients from our clinical practice.

Three Learning Lessons:
1.) We felt it was a waste of resources to pursue carotid ultrasonography in our patient because the diagnosis was vasovagal syncope.
2.) We were able to successfully cancel an ultrasound that had been ordered in a similar patient.
3.) We want clinicians to become more aware of unnecessary testing and care. Any lesions found in this patient would expose the patient to potential harm from overtreatment.

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