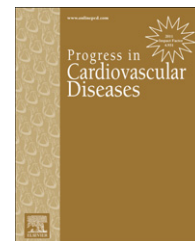


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Syncope Diagnostic Scores

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ABSTRACT

The diagnosis of syncope poses unique challenges. Syncope has multiple etiologies, with most carrying benign prognoses, and a few less common causes carrying a risk of serious morbidity or death. The history at first glance carries few clues. Faced with this many patients are heavily investigated with tests known to be both useless and expensive. For these reasons considerable emphasis has been placed on developing evidence-based and quantitative histories that might distinguish among the main causes of syncope. Quantitative histories were first developed in populations of several hundred patients with definite diagnoses of various losses of consciousness. Their derivation and use mirror those of experienced clinicians. The first score – the Calgary Syncope Seizures Score – discriminates between epileptic convulsions and syncope with a sensitivity and specificity of about 94%. The second score, the Calgary Syncope Score for normal hearts, discriminates between vasovagal syncope and other causes of syncope with a sensitivity and specificity of about 90%. The third score, the Calgary Syncope Score for Structural Heart Disease, diagnoses ventricular tachycardia with 98% sensitivity and 71% specificity. It also accurately predicts serious arrhythmic outcomes and all cause death. Gaps in the accuracy of the second score have been identified and are being addressed. These scores are proving useful in the clinic, and as entry criteria for observation studies, genetic studies, and randomized clinical trials. A very simple score predicts vasovagal syncope recurrences, based on the number of faints in the preceding year. Work from several centres indicates that scores will distinguish among competing causes of syncope in select populations, such as those with bifascicular heart block, Brugada syndrome, and Long QT syndrome.

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The diagnosis of transient loss of consciousness poses practical challenges. The first etiologies to be considered are syncope and epileptic seizures, and the distinction between the two is not always accurately made.¹ Similar diagnostic challenges with significant implications arise within different populations of people with syncope. In the community at large vasovagal syncope is by far the most common diagnosis.² It is generally benign and usually does not require

specific treatment. Conversely, syncope secondary to causes such as cardiac tachyarrhythmias, heart block, or valvular disease may forebode a fatal or non-fatal outcome that might be avoided with appropriate management.^{1,3} The investigation of loss of consciousness can be costly and intrusive and is often inconclusive.^{4–8} Therefore, an early, accurate, efficient, and inexpensive method of diagnosing the etiology of syncope is highly desirable.

Statement of Conflict of Interest: see page 394.

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Abbreviations and Acronyms

EKG = electrocardiogram

POST = Prevention of Syncope Trial

TIMI = Thrombolysis In Myocardial Infarction

UK = United Kingdom

An accurate history is the foundation of an accurate diagnosis.¹ However there is inconsistent agreement among physicians about diagnoses, exacerbated by the variable nature of the presentation of

syndromes. Some diagnostic factors are present more often than others, and patients have variable combinations of them. Indeed van Donselaar et al.⁹ noted that agreement among 3 neurologists about seizure diagnosis could be improved by structured diagnostic criteria. In the Fainting Assessment Study,⁹ whose investigators had deep physiologic and diagnostic experience with syncope, only 24% of patients had a definite diagnosis based on expert bedside assessment. As well, the most commonly used diagnostic tools – tilt testing¹⁰ and implantable loop recorders¹¹ – each has problems. Tilt tests have imperfect sensitivity and specificity, and loop recorders require the patient to faint again before a diagnosis might be made. Can we optimize our use of the history and physical examination?

The effectiveness of the history can be improved

The diagnosis of syndromes of loss of consciousness has been particularly troublesome because the principal symptom is unconsciousness, and bystander histories are often not available. Another difficulty may be the lack of structured, evidence-based histories because there may be specific points in the history that significantly improve the accuracy of the diagnosis. These should be based on quantitative evidence to be maximally credible.

One of the first attempts was by Calkins et al.¹² who studied a mixed population of 80 syncope patients with ventricular tachycardia, complete heart block, or vasovagal syncope, to identify features within the clinical history that predicted the causes of syncope. Four factors (age, sex, duration of the recovery period, and presence of mild or severe fatigue after syncope) identified the cause of syncope with 98% sensitivity and 100% specificity.

Somewhat later Alboni et al.¹³ administered standard historical questionnaires to 341 patients with established causes of syncope. The most specific features of cardiac syncope were loss of consciousness while supine or during effort, blurred vision, and convulsive syncope. The most important historical features of neurally mediated syncope were time between the first and last episode >4 years, abdominal discomfort before loss of consciousness, and nausea and diaphoresis during recovery. In patients without known heart disease, the only significant historical feature to suggest a cardiac etiology was palpitations before syncope.

These earlier studies established the importance of historical features in distinguishing among causes of loss of consciousness. However the results as presented were not easily used, and the populations were not divided into the three common problem areas: syncope versus seizures,

syncope with structurally normal hearts, and syncope with structural heart disease. Accordingly, diagnostic point scores were developed to address these questions.

Point score for distinguishing syncope from epileptic seizures

The major competing diagnoses within transient loss of consciousness are syncope and epileptic seizures, although less common possibilities such as narcolepsy, cataplexy, arrhythmias, and pseudoseizures and pseudosyncope should be remembered. Traditionally, characteristics such as auras, tongue-biting, convulsive activity, and physical trauma are used to diagnose epilepsy; however, this can be misleading in patients with akinetic seizures or convulsive syncope. Part of the problem is statistical: the age-adjusted incidence of epileptic seizures is about 0.5 per 1000 people,¹⁴ while the age-adjusted incidence of first faint is about 7 per 1000 people, and even this may be an underestimate.³ With this large difference in incidence, any diagnostic tool will either over-include syncope patients or under-include epileptic patients. Zaidi et al.¹⁵ emphasized this dilemma, showing that approximately 26% of UK patients originally diagnosed with epilepsy were found to have vasovagal syncope as the cause of their loss of consciousness.

Part of the problem, though, is the frequent association of convulsive activity with syncope,¹⁶ estimated at 12%. The most common is myoclonus, which bystanders frequently confuse with true epileptic seizures. Some patients have generalized but short-lived convulsions, which also result in an assessment for epilepsy. Finally some patients have movements best described as thrashing about. During the 1990s tilt testing was shown to be useful,¹⁷ but could a more structured approach to taking a history provide a more rapid and accurate approach to diagnosis?

Quantitative histories and diagnostic scores are well known in other fields; they are known to improve diagnostic accuracy; and there are known quantitative differences in the histories among patients with different causes of total loss of consciousness. The Calgary Syncope Symptom Study was a multinational, multicentre study that developed evidence-based diagnostic questionnaires.^{18–20} These addressed three specific situations. First, could vasovagal syncope be accurately distinguished from epilepsy? Second, in patients without known structural heart disease, could vasovagal syncope be distinguished from other causes of syncope? Finally, could vasovagal syncope and ventricular tachycardia be accurately differentiated in patients with known structural cardiac disease? A comprehensive questionnaire was administered to 671 patients in three academic centres in Canada and Wales, and point scores developed with logistic regression analysis. The cause of loss of consciousness was known in 539 patients (according to gold-standard criteria), and included various types of epilepsy, vasovagal syncope, and cardiac arrhythmias.

The first point score to be developed distinguished between syncope and seizures¹⁸ having a sensitivity of 94%

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