Patients with ischemic stroke and atrial fibrillation (AF) are treated with long-term oral anticoagulation (OAC) because it is considerably more effective than antiplatelet (AP) therapy for the prevention of subsequent strokes and systemic emboli. Therefore, detecting AF changes therapy and makes secondary prevention more effective. Despite extensive inpatient workup, including telemetry monitoring, one-quarter of stroke patients are classified as cryptogenic. However, in up to 30% a paroxysmal AF (PAF) may be detectable by long-term outpatient cardiac monitoring1 Often at hospital discharge, stroke neurologists nicely rebuild the crime scene, including pictures of a multi-shot death body (embolic looking infarct), many tracks and solid evidences (left atrial dilatation, premature ventricular beats, normal MRA), even a motive and witnesses (history of palpitations), but we still need to see the killer gripping the smoking gun to make sure it was not a suicide.

The decision to start OAC depends on the balance between early recurrent embolism and bleeding risk on anticoagulation in patients with cryptogenic stroke while the results of long-term cardiac monitoring are pending. Drs Sacco and Katsnelson need to see the smoking gun and defend a conservative evidence- and guidelines-based position. They consider that given the relatively high patient risk of systemic and intracranial hemorrhage, OAC therapy should not be started until PAF detection is confirmed on long-term cardiac monitoring. Conversely, Dr Diener trusts his intuition and experience and expresses a more straightforward position. He considers that PAF is the likely cause of the embolic looking occipital infarct, “if it looks, walks and quacks like a duck... it must be a duck,” and recommends initiation of OAC while awaiting the results of long-term cardiac monitoring, because it is superior to aspirin in the secondary prevention with a similar bleeding risk.

Although the American Heart Association guidelines recommend starting AP therapy in patients with cryptogenic stroke, treatment decision should be driven by the combination of level of clinical suspicion of cardioembolism, balance between recurrent stroke and bleeding risk, timing of diagnostic work-up, and broadness of the definition of cryptogenic stroke. Moreover, although identification of other potential causes of stroke is needed, coexistence of multiple causes does not rule out a causal role of an occult PAF on the index stroke. In fact, the presence of potential multiple causes probably confers higher risk of stroke recurrence and therefore warrants more effective treatment. Therefore, because cardioembolism is the more likely suspected stroke cause and the patient has a moderate risk of stroke recurrence in case of an eventual diagnosis of PAF (CHADS2 score 5), it is not unreasonable to start OAC while the results of long-term cardiac monitoring are pending. However, we advocate thoroughly discussing the rationale for this decision-making and the benefits versus risks of preemptive use of OAC as opposed to AP in such cases with the patients and their healthcare providers.

Our opponents also disagree about the anticoagulant to be used in our patients with CrCl of 30 ml/min. Dr Diener recommends warfarin, because recent clinical trials of new anticoagulants, dabigatran (150-mg dose) and rivaroxaban, have excluded patients with CrCl <30ml/min, and both drugs had higher rates of gastrointestinal bleeding compared with warfarin.2 Drs Sacco and Katsnelson consider that low-dose dabigatran (75-mg dose) or rivaroxaban are better choices. However, as Dr Diener points out, the low dose (75-mg dose) of dabigatran was approved based on pharmacological calculations and not on data from treated patients. In our patient with borderline CrCl of 30 ml/min and peptic ulcer disease, we would favor starting warfarin, because the patient’s renal function is likely to decline below this level over time. Warfarin is less dependent on kidney function and can be monitored by international normalized ratio measurements.

The optimum monitoring duration and method of PAF detection after stroke are unknown. Furthermore, the minimum threshold of PAF burden or duration that mandates OAC instead of AP therapy is also unknown. Brief AF episodes (<30 seconds) may be biomarkers of more prolonged and clinically significant AF. Prolonged Holter monitor up to 7 days represents one of the most frequently suggested methods to detect AF after stroke. Detection rates by Holter monitoring are higher for 7 days (12.5%) as compared with 48 h (6.4%) or 24 h (4.8%). Although 21-day ECG monitoring with mobile...
cardiac outpatient telemetry systems provides detection rates of PAF = 20%, any long-term cardiac monitoring may result in compliance problems.\(^3\) Implantable ECG devices, currently under evaluation in Cryptogenic Stroke and Underlying Atrial Fibrillation (CRYSTAL-AF) study, may provide higher detection rates. However, little information is available on the cost-effectiveness of different cardiac monitoring methods to detect PAF and subsequent treatment with anticoagulants. In our case, if 30-day monitoring is still unrevealing, the patient should be switched to aspirin. Predictors of PAF, including dilated left atrial on echocardiography, premature atrial and ventricular beats of ECG, serum biomarkers, such as brain natriuretic peptid, and the stroke pattern on MRI may be helpful to identify a stroke population in which extended cardiac monitoring is highly likely to unmask the occult diagnosis: atrial fibrillation...what else!

**Disclosures**

None.

**References**

"If It Looks Like a Duck, Walks Like a Duck, and Quacks Like a Duck… It Must Be a Duck": Anticoagulation in Stroke Patients With Suspected Atrial Fibrillation
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The online version of this article, along with updated information and services, is located on the
World Wide Web at:
http://stroke.ahajournals.org/content/44/1/302