I hope Professor Black is genuinely planning to get a grip on this aspect of training. To speak frankly, as it stands the ePortfolio is a crushing piece of bureaucratic nonsense. If you set out to develop an education initiative with the sole aim of making training less enjoyable you would struggle to beat this. There is no doubt in my mind that the ePortfolio and assessments (and by extension JRCPTB) have been a large driver of falling morale among medical trainees. As things stand, there are too many assessments; yes, they are clunky and the IT is abysmal but the real enemy here is the 'linking', so while the focus on outcomes is welcomed, I hope this is also tackled. Currently, a trainee has to link evidence to each part of their ePortfolio, then write about why they are competent in that area and then ask their supervisor to countersign that they are. The idea that the linking process will highlight the failing trainee, or even contribute meaningfully to the training of good trainees, is laughable.

While I congratulate Professor Black on trying to improve things and reduce the tick-box culture (that his organisation was instrumental in creating), it might be prudent to consider why they were unresponsive to criticism for so long. Certainly when I argued for simplification of the system in 2008 I was treated as a wayward schoolboy who, perhaps by virtue of not having paid for a masters in medical education, was unenlightened. When future trainees bring up genuine concerns about the next curriculum iteration these can't be dismissed so easily. ■

Conflicts of interest

The author has no conflicts of interest to declare.

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The latest national clinical guideline for stroke

Editor – There is lack of clarity in the latest *National clinical guideline for stroke* on cardiac monitoring if a cardio-embolic stroke is suspected.[1]

It is estimated that between 25 and 60% of atrial fibrillations are paroxysmal (PAF) and approximately one third of the patients affected are asymptomatic.

Thirty percent of all strokes remain without an identifiable cause even after extensive workup. Occult PAF seems to be one of the culprits of 'cryptogenic' strokes.

All ischaemic stroke patients should have a standard 12-lead electrocardiogram (ECG), which will detect a new PAF in 2–4% of patients.

Cardiac telemetry for 28–72 hours after index hospital admission has been reported to detect new AF in up to 2.4–18.5% of patients with acute ischemic stroke.[2] This facility, however, is often used to monitor patients receiving systemic thrombolysis.

Serial ECG assessments within the first 72 hours of an acute stroke significantly improve detection of AF. This strategy is particularly useful when cardiac telemetry is not readily available.[3]

Given the asymptomatic nature of PAF, patient-activated cardiac monitoring devices are of low clinical value.

Most patients in the UK will receive a single 24/48/72-hour Holter monitor after a cryptogenic stroke as per the current guideline (Fig 1). The diagnosis of PAF is often missed because of low rates of arrhythmia detection by these devices.[4] These patients will thus miss out on anticoagulation, leading to recurrent strokes and a higher mortality.

A 7-day ambulatory ECG monitor detects AF in 5.7% of patients with a normal standard ECG and 24-hour Holter. [5] In our trust, 7-day ambulatory ECG monitors are used instead of 24/48/72-hour Holter to detect occult PAF.

The EMBRACE (Event Monitor Belt for Recording Atrial Fibrillation after a Cerebral Ischemic Event) study demonstrated that the 30-day event-triggered recorder was significantly more effective than 24-hour Holter for identification of AF in patients with recent cryptogenic strokes. My practice is to perform a 32-day ambulatory ECG monitoring if the 7-day ECG does not detect low-burden PAF.

Surface ECG recorders (7- or 32-day ECG monitor) rely on skin contact electrodes. Skin irritation or 'allergic' reactions have been the commonest cause of non-compliance in my practice. This makes it difficult for patients to wear these devices for long durations.

AF detection rate in the CRYSTAL AF (Cryptogenic Stroke and Underlying AF) study were 8.9% at 6 months, 12.4% at 12 months and 30% at 3 years in those with cryptogenic stroke. In view of this evidence, I refer patients in whom prolonged ECG monitoring has failed to detect PAF, have developed allergic reactions to skin contact electrodes or do not wish to wear devices for 32 days to a cardiology colleague for an insertable loop recorder.

Finally, dual-chamber pacemakers and implantable cardioverter defibrillators are cardiac implantable electronic devices. These *in situ* intra-cardiac devices can be programmed for continuous ECG monitoring in patients with suspected cardio-embolic stroke to detect asymptomatic atrial tachyarrhythmia. ■

Conflicts of interest

The author has no conflict of interest to declare.

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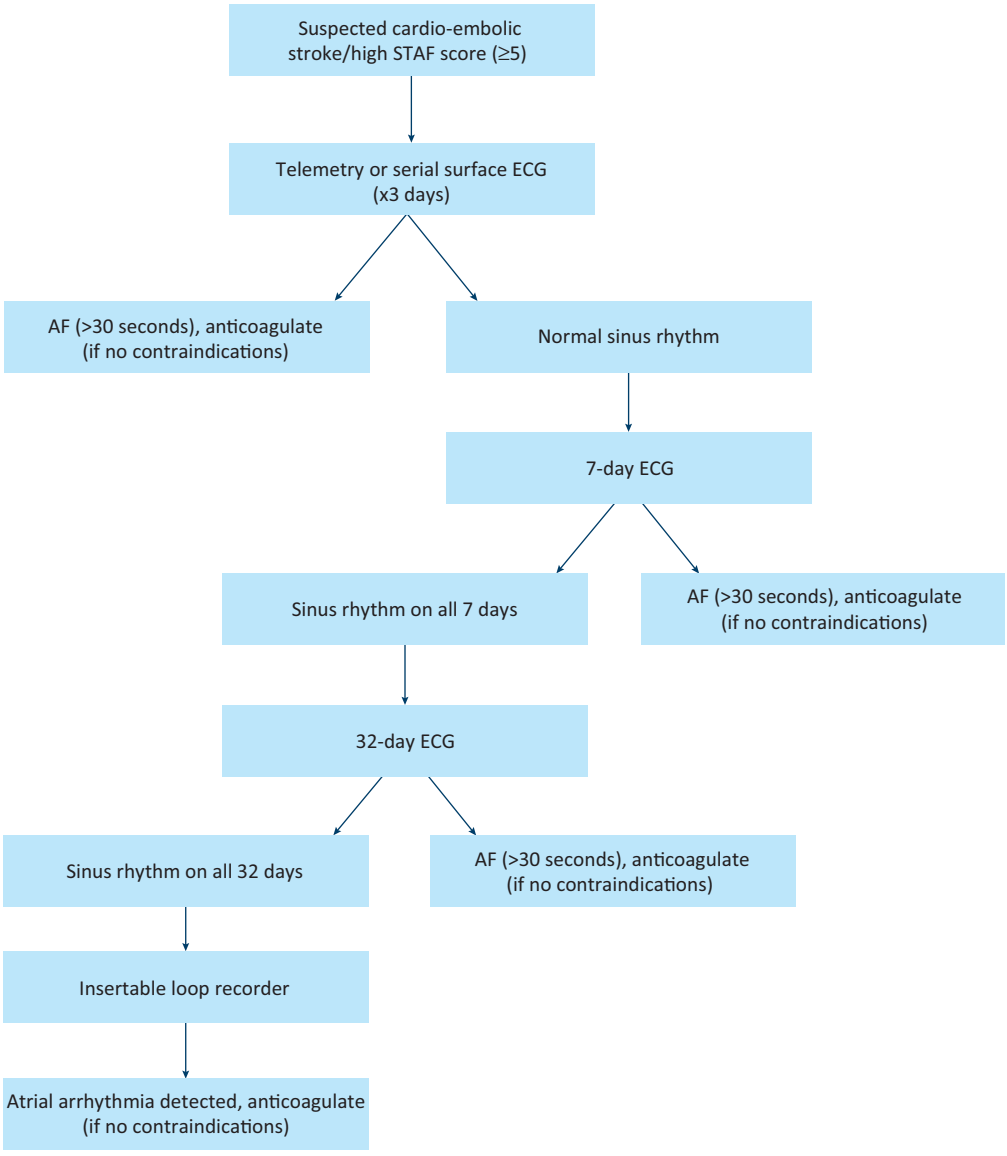

Fig 1. A flowchart of the step-wise detection of PAF in suspected cardio-embolic stroke patients. AF = atrial fibrillation; ECG = electrocardiogram; PAF = paroxysmal atrial fibrillation; STAF = Score for the Targeting of Atrila Fibrillation

5 Jabaudon D, Sztajzel J, Sievert K, Landis T, Sztajzel R. Usefulness of ambulatory 7-day ECG monitoring for the detection of atrial fibrillation and flutter after acute stroke and transient ischemic attack. *Stroke* 2004;35:1647–51.

Getting to the heart of hypopituitarism

Editor – In the reported case of the association of panhypopituitarism, empty sella and pericardial effusion,[1] autoimmune hypophysitis (AH) might have been the underlying basis for all three derangements. Firstly, empty sella may be the eventual outcome of AH.[2,3] Secondly, AH is an example of type 2 autoimmune polyglandular syndrome(APS-2),[4] which is now recognised as sometimes having serositis as a non-endocrine manifestation.[4] In one patient with AH, pericardial effusion was an example of the coexistence of serositis and APS-2.[3] In that 37-year-old woman, serial magnetic resonance imaging studies during 2-year follow-up showed an atrophic pituitary gland with empty sella turcica.[3]

Serositis was exemplified by pericardial effusion (with tamponade) in a 34-year-old woman in whom the endocrine component of APS-2 consisted of the coexistence of primary hypoadrenalism and primary hypothyroidism. Following pericardiocentesis, despite good compliance with hormone replacement therapy, she experienced seven documented recurrences of pericarditis over a period of 28 months,[5] testifying to the observation that serositis may recur after asymptomatic intervals of months or years even in patients treated for endocrine dysfunction.[6] Accordingly, in APS-2 the evolution of serositis does not necessarily parallel the evolution of endocrine dysfunction, and adequacy of replacement therapy does not guarantee freedom from recurrences of serositis. ■

Conflicts of interest

The author has no conflicts of interest to declare.

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