Clinical guidelines have become a core element in optimising care for patients with cardiovascular disease. However, the quality of guidelines depends on a rigorous unbiased process that integrates the clinical evidence with input from a range of stakeholders. In this issue of Heart, Garbi summarises the National Institute for Health and Care Excellence (NICE) principles and processes for development of clinical guidelines in England. The discussion is divided into four key areas: (1) Guideline development by an independent advisory committee includes aligning recommendations with national health policies, and involvement of patients, patient-advocates, and the public as well as healthcare professionals. (2) Recommendations should be based on relevant, reliable and robust evidence and should include consideration of cost-effectiveness and population benefit. (3) Guidelines should support innovation and reduce healthcare inequalities. (4) Finally, ensuring guideline implementation and providing regular updates are essential.

In the accompanying editorial, Otto, Kudenchuk and Newby compare the NICE methodology with the current approach of our cardiovascular professional societies, as well as to established reporting criteria for clinical practice guidelines (figure 1). They propose several areas for improvement including cooperative development of a common evidence database; a rigorous transparent process based on established standards; a more diverse group of stakeholders; minimising conflicts of interest; support by information specialists, medical writers and other relevant experts; regular updates; adaptation for regional considerations; and improved methods for dissemination and access. As they conclude: ‘Current cardiovascular society guidelines fall short of best practice. We can and must do better.’

In patients with atrial fibrillation (AF) at moderate or high risk of stroke, randomised controlled trials (RCTs) have shown superiority or non-inferiority of non-vitamin K oral anticoagulants (NOACs) over vitamin K anticoagulants (VKA) for prevention of stroke or systemic embolism along with reduced rates of intracranial haemorrhage. However, patients in RCTs may not be representative of the full range of patients seen in clinical practice. In order to address this issue, Camm and colleagues used a method called overlap propensity matching to compare the effectiveness of VKA and different NOACs for mortality, stroke/systemic embolism and major bleeding in patients with newly diagnosed AF and an indication for oral anticoagulation. Based on 25 551 patients in the Global Anticoagulant Registry in the FIELD-Atrial Fibrillation (GARFIELD-AF) study, they confirmed that ‘Important benefits in terms of mortality and major bleeding were observed with NOAC versus VKA with no difference among NOAC subtypes’ (figure 2).

In the accompanying editorial, Choi and Lee point out the strengths of this study including a clinically diverse international patient cohort with regular audits and a low rate of loss to follow-up, a sophisticated matching method, and results consistent with previous RCTs. However, limitations include the possibility of residual confounders; possible discontinuation or switching of medications during
Heartbeat

Figure 2 Adjusted* HRs and corresponding 95% CIs for selected outcomes at 2 years of follow-up by OAC treatment at baseline. The reference considered is the treatment reported as second. *Obtained using an overlap-weighted Cox model. Variables included in the weighting scheme are: country and cohort enrolment, sex, age, ethnicity, type of AF, care setting specialty and location, congestive heart failure, acute coronary syndromes, vascular disease, carotid occlusive disease, prior stroke/TIA/SE, prior bleeding, venous thromboembolism, hypertension, hypercholesterolaemia, diabetes, cirrhosis, moderate to severe chronic kidney disease, dementia, hyperthyroidism, hypothyroidism, current smoking, heavy alcohol consumption, body mass index (BMI) heart rate, systolic and diastolic blood pressure at diagnosis and baseline antiplatelet use. DTI, direct thrombin inhibitor; FXa, factor Xa inhibitors; NOAC, non-vitamin K oral anticoagulants; OAC, oral anticoagulants; SE, systemic embolism; TIA, transient ischaemic attack; VKA, vitamin K antagonists.

Figure 3 Mechanism of mitral regurgitation (MR). The mechanisms of valve dysfunction in patients with moderate or greater MR are shown, according to Carpentier classification. Type 1, normal leaflet motion and position; type 2, excess leaflet motion; type 3a, restricted leaflet motion in systole and diastole; type 3b, restricted leaflet motion in systole.

Figure 4 Concerns of the pregnant cardiologist.
for women considering pregnancy during cardiology training (or as a consultant cardiologist) for those providing training and support to those women (figure 4).

**Funding** The authors have not declared a specific grant for this research from any funding agency in the public, commercial or not-for-profit sectors.

**Competing interests** None declared.

**Patient and public involvement** Patients and/or the public were not involved in the design, or conduct, or reporting, or dissemination plans of this research.

**Patient consent for publication** Not required.

**Provenance and peer review** Commissioned; internally peer reviewed.

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**To cite** Otto CM. Heart 2021;107:937–939.

doi:10.1136/heartjnl-2021-319647

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