reflect DNAemia observed in acute Q fever. [4] In our opinion, this should not be considered definite endocarditis. In addition, 8 of 18 patients (44%) without subsequent endocarditis started prophylactic treatment as late as 2–8 months after the diagnosis of acute Q fever, and, surprisingly, endocarditis developed within 2 months in 11 of 13 patients (85%) without prophylaxis. Moreover, most possible endocarditis cases had rather low immunoglobulin G phase I titers (1.800). Because the diagnosis of endocarditis in most patients with preexisting valvulopathy was based solely on marginally increased serological titers within a short time frame after acute Q fever, we are concerned that these cases may have been misdiagnosed and merely reflected increasing immunoglobulin G phase I titers, which can be observed until 6 months after acute Q fever [5].

Furthermore, potential side effects that accompany a 12-month antibiotic course with doxycycline and hydroxychloroquine were not discussed. In the study of 568 patients with a history of cardiac valve surgery living in an outbreak area in the Netherlands, reported by Kampschreur et al [6], *Coxiella burnetii* antibodies were detected in 20% of patients, of whom 8% had probable or proven Q fever endocarditis. If these patients had been offered prophylaxis, 92% of them would have unnecessarily received toxic antibiotics for 12 months.

Currently, in the Netherlands, routine evaluation for valvulopathies is not advised, based on prospective follow-up findings in a series of patients [7]. In a cohort of 85 patients with acute Q fever and a high prevalence of cardiac valvulopathy (39 of 85; 46%), chronic Q fever had not developed in any of them after 1 year of follow-up. The difficulty in the optimal workup after an episode of acute Q fever is also reflected in the recent formulations of the Centers for Disease Control and Prevention, which advise patients with acute Q fever to undergo careful clinical assessment, including assessment for vascular and heart valve defects, but make no specific recommendations on the most appropriate tools for this assessment [8]. In conclusion, prophylactic treatment for high-risk patients after an episode of acute Q fever can be beneficial, but which patients benefit from such strategy and the optimal duration of prophylaxis still need to be determined.

**Note**

**Potential conflicts of interest.** All authors: No reported conflicts.

All authors have submitted the ICMJE Form for Disclosure of Potential Conflicts of Interest. Conflicts that the editors consider relevant to the content of the manuscript have been disclosed.

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**Reply to Kampschreur et al**

**To the editor**—We thank Kampschreur et al [1] for allowing us to clarify that the strategy applied in the large Dutch Q fever outbreak completely failed to prevent and detect Q fever endocarditis. A key point that has been completely neglected in this context is the preventive treatment of patients with symptomatic primary infection and significant valvulopathy, the efficiency of which was demonstrated in our prospective cohort study [2].

In a recent study performed in the Netherlands [3], 20 of the 32 patients (62%) classified as having “proven chronic” or “probable chronic” Q fever had a previous symptomatic acute primary infection. Furthermore, 9 (28%) had a predisposing valvulopathy, and 20 (62%) had a minor echocardiographic criterion; however, none of them benefited from antibiotic prophylaxis. It is also surprising that only 6 of these 32 patients (19%) had a further diagnosis of possible or definite endocarditis, because the authors recognized “chronic Q fever” in ≥20 patients with significant valvulopathy, without making a diagnosis of endocarditis. Given the rarity of the presence of vegetation or major echocardiographic criteria in Q fever endocarditis, patients with a significant native valvulopathy and elevated Q
fever serologic findings (phase I immunoglobulin G ≥800) should be considered to have Q fever endocarditis [4] and treated accordingly.

Withdrawing the echocardiographic detection of silent valvulopathies during acute Q fever, as recommended in the Netherlands [5], is absurd because it differs from the usual recommendations; this explains the high numbers of unrecognized Q fever endocarditis cases, because no patients were provided antibiotic prophylaxis in the Netherlands.

Notes

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