We read with interest the article by Li and colleagues (Eur Heart J 2002; 517–520) and the accompanying editorial by Professor Muir (Eur Heart J 2002; 517–520). The data and the hypothesis presented by Li and colleagues are both attractive and intriguing, but before this concept can be accepted as contributing to the pathogenesis of rheumatic fever and rheumatic heart disease both the authors of the paper and the editorialist should account for several inconsistencies.

Dr George Burch would be pleased to see the issue of a viral aetiology of rheumatic fever being raised again. As the authors state he published many studies relating to the theory of viral aetiology of rheumatic heart disease. But one of the criticisms levelled at Dr Burch’s work appears to me to still be relative to the current paper and editorial. If viruses, in any way, contribute to the pathogenesis of rheumatic fever, then how do proponents of a viral role explain the incontestable data in numerous careful studies documenting the efficacy of penicillin prophylaxis for prevention of first and recurrent attacks of rheumatic fever? Surely the penicillin has no effect on the enteroviruses?

Until the authors are able to fit this information into their hypothesis, accompanied by supporting data, their hypothesis remains tenuous.

Second, while the authors describe the patients whose valves they studied as having had ‘rheumatic fever’, they present essentially no data about individual individuals patients and about their valves to substantiate this diagnosis. All clinicians are well aware that all valvular disease is not rheumatic in origin. Further, since there is no single laboratory test now (and certainly not in the past) that is diagnostic of rheumatic fever, we are again left with less than adequate supporting data about these valve specimens, or about the patients they came from. In fact, there are some who would take issue with the statement included in the first paragraph of the methods section. Does one really diagnose ‘chronic rheumatic heart disease’ by the Jones criteria? The answer is no! It is rheumatic fever that is diagnosed by the Jones criteria. In fact, if I am correct, the reference cited (reference [26]) makes specific mention that this so-called ‘updated’ version of the Jones criteria is for first attacks of rheumatic fever only. One might be justified in stating that this represents a leap of faith in assuming that these patients were ‘rheumatics’. Can the authors present convincing specific data to substantiate the diagnosis of acute rheumatic fever/rheumatic heart disease in these patients? Would this not be crucial to their hypothesis that the viruses they report caused acute rheumatic fever? To prove this hypothesis would also require documentation that the virus was not present prior to the development of the valvular heart disease they have attempted to describe.

I do not argue that their hypothesis is not a possibility. However, I do firmly believe that they have not provided scientifically and epidemiologically convincing data to substantiate this hypothesis. It would appear to me that these authors have yet to address the very important question of the pathogenesis. Professor Muir admits in his editorial that we do not know the pathogenetic mechanisms responsible for the transition from acute group A streptococcal infection to rheumatic fever. While the authors of the paper can be acknowledged for their attempt, they must be more cautious in their conclusions.

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Combined effects of systolic blood pressure, serum cholesterol and smoking on coronary heart disease and stroke

We read with interest the article by Thomas et al.[1] on ‘Combined effects of systolic blood pressure and serum cholesterol on cardiovascular mortality in young (<55 years) men and women’. The authors have observed a dramatic increase in cardiovascular disease and coronary heart disease risk in young (especially men) French people due to an associated elevation of high systolic blood pressure and high serum cholesterol.

We would like to draw attention to the fact that hypertension and serum cholesterol also enhance the risk of stroke, which is the second most common cause of death worldwide. The authors have recorded a significant proportion of mortality induced by high systolic blood pressure and high serum cholesterol. However, the possibility of a cause and effect relationship between stroke and mortality due to these factors has not been considered in this study. Several studies have shown that treatment of systolic hypertension in the elderly can prevent stroke and cardiovascular events [2]. It appears that few studies have covered the effect of high systolic blood pressure and high serum cholesterol in younger patients. We strongly consider that had the authors taken this point into consideration during their study, it would have benefited the scientific community much more.

Additionally, the information pertaining to the role of certain risk factors, such as smoking, has not been addressed clearly. For example, the earlier study, in which patients with 6.7 mmol·L⁻¹ of plasma cholesterol and a borderline systolic blood pressure of 140 mmHg were prone to coronary heart disease and cardiovascular disease, is almost similar to patients with a high cholesterol (9.1 mmol·L⁻¹) or a systolic blood pressure of 240 mmHg, if the former patient smokes five cigarettes per day[3]. Therefore, the relationship of borderline elevation of two or three risk factors with serious elevation of one factor, as detailed above, deserves more elucidation.

It is documented that isolated systolic hypertension is a good predictor with which to indicate the proneness of cardiovascular disease in those who are above 60 years of age. To the best of our knowledge, information on this is not available in younger patients. In the present study from the provided data, it is difficult to analyse the presence of any case having isolated systolic hypertension. Consideration of this point during analysis also would have strengthened the scientific content.