



Editorial – referring to the article published on pp. 721–731 of this issue

Erectile Dysfunction and Vascular Risk: Let's Get It Right

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The current consensus is that erectile dysfunction (ED) and vascular disease often coexist and that endothelial dysfunction is the common denominator [1]. This is especially true of men aged 40 yr and older. A body of literature has now identified the role of ED as a marker of silent vascular disease and, more significantly, coronary artery disease (CAD) [2]. With a lead time averaging 2–3 yr between ED and CAD presentation, the question is whether an aggressive approach to evaluating vascular risk in men asymptomatic for CAD but with ED is justified. To be justified it would need to translate into an interventional programme to reduce vascular risk with clinical end point data of benefit over time. This supposes, with some justification, that ED falls into the category of secondary prevention of CAD (as if an event had already occurred) rather than primary prevention (before an event). However, these two divisions are artificial (you can be well one day [primary prevention] and suffer a myocardial infarct that next [secondary prevention]) so our focus should be on overall risk and, in turn, reducing the risk.

Montorsi and colleagues [3] along with other authors have set the targets, namely, with the established CAD risk who should be screened and how [4,5]. We do not have 3–5- or even 10-yr follow-up data confirming that intervention will reduce the risk of developing symptomatic CAD, either as an acute or chronic presentation, but we have good data from cardiovascular (non-ED) studies that men of a similar age and vascular risk benefit significantly from risk reduction [6]. Until formal studies are available, these cardiac studies should act as

templates for intervention. It is inconceivable that a significant number of men in the cardiac studies did not have ED—no one asked!

All health care professionals dealing with ED need to be able to take a good cardiac history. Asking about effort-induced chest pain or breathlessness should be complemented with a check of the family history, smoking habits, and any suggestion of hypertension. Is he overweight, what exercise does he take, and is he on any medication? More subtle signs include recent increased fatigue, falling asleep easily when watching television, irritability, and being unusually short-tempered. Clinical examination is usually normal but occasional murmurs are heard, carotid or femoral bruits detected, peripheral oedema noted, and blood pressure found to be raised (>140/90 mm Hg). Routine investigations should include a fasting glucose and lipid profile; any evidence of the metabolic syndrome should be acted on with appropriate referrals [7]. In the era of technology “old-fashioned clinical medicine” remains the basis for directing further tests—in other words as Montorsi et al. [3] suggest—“matching the right target with the right test in the right patient.”

What to do next? The second Princeton Guidelines offer us a good way of stratifying risk and their algorithm is complemented by Montorsi's in this issue [3,8]. Those at low risk need lifestyle advice and regular monitoring by their family doctors. Those at increased risk need their risk factors addressed, which may seem obvious, but it is surprising how often they are not acted on. Specialist referral is recommended where appropriate especially to a diabetologist if an elevated glucose level is detected

because this triggers a more aggressive approach to risk reduction along with the introduction of specific drug therapy. Looking for silent CAD can involve multiple tests of varying degrees of complexity and expense. The resting electrocardiogram (ECG) is usually normal, but if it is abnormal, entry into the high-risk category is automatic. An exercise ECG is the simplest, most readily available, and least expensive evaluation and is recommended for all men at increased risk [9]. If it is abnormal, then in addition to risk reduction therapy, angiography should be considered, but this needs to be set against the age of the patient and his personal wishes. It will be of more value in younger men where prognostic consequences are of greater importance than in older men who may prioritise their quality of life and not wish it disrupted. When an exercise ECG cannot be interpreted (e.g., when there is a left bundle branch block) or when it cannot be undertaken due to mobility problems (e.g., arthritis) or when it is inconclusive, obtaining a perfusion scan or stress echocardiogram is advised. If murmurs are heard, an echocardiogram should be performed before exercise evaluation; the presence of bruits determine the need for vascular ultrasound. If the evaluation is abnormal and silent CAD is suspected, a cardiology referral with a view to angiography should follow.

The current paper by Montorsi et al. [3] moves us from the link between ED and CAD to the next stage of evaluating vascular risk. ED, like diabetes, should be considered a “cardiovascular equivalent.” Our screening weapons primarily focus on detecting obstructive flow limiting CAD, whereas the sub-clinical nonobstructing lipid-rich plaque that ruptures is the more dangerous. Preventing an acute coronary syndrome requires an aggressive risk reduction strategy no matter the result of the tests looking for obstructive lesions. We can match the right test to the right patient but all men with ED

must have their cardiovascular risks evaluated and dealt with. We can and should use ED as a marker of vascular disease and especially CAD but there is no point in doing this unless we act to reduce the risk.

A multidisciplinary approach is needed; whilst cardiologists can educate urologists on how to reduce vascular and especially coronary risk, I am sure urologists can teach cardiologists a thing or two in return.

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