

Solid science and hard logic — the rock on which good treatment is based

With electron beam computed tomography (EBCT) it is now possible to detect non-invasively and rapidly, and to a degree to quantify, calcium deposition in coronary arteries. This remarkable technique also allows for a detailed evaluation of cardiac function and cardiac chamber volume and mass, both at rest and during exercise. It is likely that myocardial blood flow will soon be similarly quantifiable.¹ We are therefore excited by the existence of this apparatus in the Pretoria Heart Hospital. The ability to detect intracoronary calcium, and implications deriving from the fact that such deposition only occurs in diseased arteries, has sparked major controversy in terms of the application of this technique.

In this issue of the *SAMJ* Dr Nel joins the controversy with an impassioned appeal for widespread utilisation of EBCT for existing or suspected coronary artery disease, either clinically manifest or silent. The sociopolitical parallels he draws, although poignant, are often somewhat obscure and bitter, particularly in relation to issues of ownership and payment. Much of the heat generated in this controversy relates to appeals made directly to the general public, and is often centred around the spectre of sudden cardiac death. Thus the focus is both medical and fiscal.

An excellent, balanced and current overview of the epidemiology, clinical implications, pathophysiology and imaging of coronary artery calcification is offered in a statement from the American Heart Association (AHA), to which the reader is referred.²

EBCT-detected coronary calcification can predict future cardiac events, but not all of them.¹ It is a good predictor of the need for revascularisation in both symptomatic and asymptomatic³ patients, but a poor predictor of coronary death or myocardial infarction.⁴ This regrettably partly closes the 'window of opportunity' optimistically referred to by Dr Nel. The reason for this inability to predict myocardial infarction and infarction-related death is that although EBCT-detected coronary calcium correlates well with the established atherosclerotic plaque that can impair coronary flow when large (the bigger the plaque the higher the calcium score), thrombosis and infarction often occur in unstable plaques lacking or low in calcium. They also occur frequently in the absence of a heavily calcified overall plaque load.⁵⁻⁶ Sudden cardiac death has causes other than acute myocardial infarction. A major cause is predictable and treatable ventricular arrhythmias. Here death is preventable with implantable defibrillators, a fact proven in numerous studies, but payment for the implantation has not been guaranteed by the Representative Association for Medical Schemes (RAMS).

There is an overall correlation between the total area of detected coronary calcification and the total plaque load.^{9,10} Unfortunately there is currently insufficient evidence that EBCT can accurately track the total atherosclerotic load over time. It therefore cannot act as a guide to whether therapy is effective nor not. Although this may improve with new technology, it has not been proved as yet.

In symptomatic patients with angina, EBCT is powerfully predictive. Comparison with stress testing and thallium imaging using coronary angiography as the gold standard, however, shows little difference in predictability of coronary stenosis; in fact the best results in direct comparison have been with the latter. However, the data are inadequate. Four studies referred to above are based on a total of 811 patients, only approximately half of whom had significant angiographic stenosis. EBCT carries a sensitivity of 85 - 100% and a specificity of 41 - 76%.²

For the patient with atypical chest pain, particularly in the younger age group, or patients with some bar to good electrocardiographic assessment (e.g. left bundle-branch block, female gender), EBCT seems to have a definite place at present.

There is a major difference between coronary artery disease with calcification and coronary heart disease with infarction and angina. EBCT as locally practised and promoted efficiently plots the former. The high incidence of coronary calcification in the general population¹¹ and the fact that in any age group the prevalence of coronary calcium is 10 - 100 times the expected incidence of coronary heart disease over a 10-year period, highlight this dilemma.² Over-prediction of coronary heart disease is greater in the younger than the older population, doubtless because of the increasing incidence of coronary heart disease with age.

The 'research project' proposed by Dr Nel for tracking the development of coronary artery and heart disease in the emerging black middle-class population is therefore both fascinating and daunting, financially and in terms of the duration of the study.

The question of payment for services by RAMS must surely be resolved on the basis of a clear role for EBCT above existing and efficient forms of investigation. These do exist (for example atypical chest pain, a bar to the performance of a diagnostic stress test, typical chest pain with a negative stress test, etc.) Undoubtedly the indications will increase with time and further knowledge. Invasive coronary angiography will in the foreseeable future be the only way in which revascularisation can be planned or performed. Stress testing such as EBCT, echocardiography and risk factor detection offer much to the patient if expertly and appropriately applied. Promotion of large-volume EBCT screening by means of broad public appeal has not had endorsement by professional medical bodies. The question of ownership of the facility will undoubtedly resolve in the future, *pari passu* with the interest, commitment and expertise of the practitioner. It is obviously mandatory that referrals for EBCT be via a qualified and informed cardiologist at this stage.

EBCT is clearly an exciting and growing technique. It is certainly not experimental, but its clinical applicability, like all technologically based medicine, needs defining, and will in the future need redefining as evidence accumulates. A set of guidelines is in the process of prepublication review and may shed light on where we are, what we should be doing, and certainly what we should be paid for. It may also help tell us where we are going; an exciting prospect that should not and will not limit itself to the detection of coronary calcification. In the meantime, prophylactic therapy should be based on accepted techniques.

I W P Obel

P O Box 91155
Auckland Park
Johannesburg

1. Brundage BH. Electron beam computed tomography. In: Topol EJ, ed. *Textbook of Cardiovascular Medicine*. Philadelphia: Lippincott-Raven, 1998: 1475.
2. Wexler L, Brundage B, Crouse J, et al. Coronary artery calcification, pathophysiology, epidemiology, imaging methods and clinical implications. A statement for health professionals from the American Heart Association. *Circulation* 1996; **94**: 1175-1192.
3. Arad Y, Spadaro LA, Goodman K, et al. Predicted value of electron beam computerised tomography of the coronary arteries. *Circulation* 1996; **93**: 1951-1953.
4. Secci A, Wong N, Tang W, Wang S, Doherty T, Detrano R. Electron beam tomographic coronary calcium as a predictor of coronary events. *Circulation* 1997; **96**: 1122-1129.
5. Cheng GC, Lorce HM, Camm RD, Fishbein MC, Lee RT. Distribution of circumferential stress in ruptured and stable atherosclerotic lesions, a structural analysis with histopathological correlation. *Circulation* 1993; **87**: 1179-1187.
6. Beadenkopf WG, Daod AS, Love BM. Calcification in the coronary arteries and its relationship to arteriosclerosis and myocardial infarction. *AJR* 1964; **92**: 866-871.
7. Van der Wal AC, Becker AE, Van der Loos CN, Das PK. Site of internal rupture or erosion of thrombus atherosclerotic plaques is characterised by an inflammatory process irrespective of the dominant plaque morphology. *Circulation* 1994; **89**: 36-44.
8. Forrester JS, Shah PK. Lipid lowering versus re-vascularisation, an idea whose time (for testing) has come. *Circulation* 1997; **96**: 1360-1362.
9. Kragel AH, Reddy SG, Wittes JT, Roberts WC. Analysis of composition of atherosclerotic plaques in the four major arteries in acute myocardial infarction and in sudden coronary death. *Circulation* 1989; **80**: 1747-1756.
10. Rumberger JA, Simons DB, Fitzpatrick LA, Sheedy PF, Schwartz RF. Coronary artery calcium areas by electron beam computer tomography and coronary atherosclerotic plaque area. Histopathologic correlative study. *Circulation* 1995; **92**: 2157-2162.
11. Detrano RC, Wang ND, French WJ, et al. Prevalence of pleuroscopic coronary calcium deposits in high risk asymptomatic patients. *Am Heart J* 1994; **127**: 1526-1532.