To the Editor

in this area we felt unable to draw any limited amount of data that are available. Given these equivocal findings and the /C2 group (0.48 counts tended to be higher in the control controls. In fact, in this study monocyte between patients with periodontitis and 

counts in subjects with periodontitis are However, data on increased monocyte and increased monocyte activity is known the monocyte relationship: Reply disease, periodontitis and Cardiovascular 

increase of cardiovascular disease (CVD). monocytes on the development and pro-
titis,1 given the potential influence of our recently observed association between 

important and interesting issue regarding disease, periodontitis and CVD, and provide a possible explanation for the nature of this association. However, CVD is a complex, multifactorial group of diseases and further studies are clearly warranted. We are currently undertaking a larger intervention study, the aim of which is to assess whether treatment of periodontal disease reduces the levels of serological markers of CVD risk. Should differences/changes in monocyte counts be observed in this study, then we would, in light of Dr. Otis and her colleagues’ suggestion, certainly re-evaluate the relationship between monocyte counts and the risk of CVD, particularly in individuals with periodontitis.

References


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Direct epicardial mapping predicts the recovery of left ventricular dysfunction in chronic ischaemic myocardium

We read with interest the article of Vahlhaus et al.,1 concerning epicardial mapping to predict the recovery of left ventricular dysfunction in chronic ischaemic myocardium. This study has been based on bi-polar voltage mapping criteria during surgery for the diagnosis of myocardial hibernation.

The authors report that there is a great overlap between groups of viable and non-viable myocardial segments found in a grey zone of 5.4–12.3 mV. They also state that the data provided by their study is obviously obtained too late to help in pre-operative decision making, but that bi-polar voltage may be useful to understand the pathophysiology of chronic ischaemic dysfunction. They also admit that it is not the aim of the present study to implement direct electrical mapping for clinical use.

Our criticism of this study is that their method was different from that generally used for electromechanical mapping, namely the combined bi-polar voltage criteria and local linear shortening.2 The recognition of myocardial viability should be based not only on voltage measurement (electrical reserve) but also on local linear shortening (contractile reserve). The method advocated in clinical use is the endocardial electromechanical NOGA mapping (Biosens Webster Cordis, Johnson Johnson, Diamond Bar, CA, USA). The NOGA segmental quantitative analysis is performed after transformation of the three dimensional map into a polar map, with 12 segments from the apical, the mid and basal location. Myocardial viability is present when the voltage is more than 6 mV (Nle value of 15 mV) and local linear shortening is more than 7% (Nle value 11%). The myocardial regions with parallel decreased of voltage and linear shortening identify hibernating myocardium with an improvement in contractility 6 months after coronary revascularisation. The sensitivity and specificity of this test, versus nuclear technique are low and can be improved by further analysis of local voltages.

Even if myocardial viability and its surrogate myocardial ischaemia cannot be related solely to ventricular systolic function, the consideration of bi-polar voltage with local linear shortening, is the recommended method for the diagnosis of myocardial viability. The inclusion of local linear shortening alongside the voltage criteria, decreases the extent of "grey zone and overlap" between groups of viable and non-viable myocardial segments observed in the study of Vahlhaus et al. The authors state that the 18F-FDG-PET represents the gold standard for detection of myocardial viability. However, the "gold standard" test with 18F-FDG-PET, a metabolic marker, is insufficient for viability assessment, and it should be coupled to a flow marker such as N-13-ammonia. Increased, or maintained, FDG uptake on a PET scan in the presence of decreased flow (mis-match) is diagnostic for myocardial hibernation. Conversely a decrease in both the metabolic and flow PET scan (match) is an indication of the absence of viability.

Given the expensive cyclotron generator with high cost and complexity, the use of new tracers detected by planar scintigraphy and SPECT with recently developed 511 keV collimators are more economical and more easily accessible to a wide range of hospitals. The PET with 11-carbon acetate (aerobic or oxidative metabolism) with or without dobutamine infusion is more accurate than conventional FDG PET (anaerobic or glycolytic metabolism). Fatty acid derivatives 123-I-beta-methyl-(anaerobic or glycolytic metabolism). The tissue Doppler measurement of a velocity, at the epicardial and endocardial layers, of less than 5 and 11 cm/s, respectively, allows also the recognition of myocardial hibernation. Concerning the magnetic resonance imaging we have the structural T1 and T2 weighed-ECG Gated technique, functional Gradient-Echo, Harmonic phase, Tagged MRI and Resonance Spectroscopy for "metabolic biopsy".

Nevertheless, the conventional cine wall motion images do not detect early viability episodes in the high-field MRI environment, and lag behind the ECG changes. The recently developed MRI compatible-life support equipment has made this technique easier to use with fringe field situated within the outer Faraday cage, allowing continuous access to the patient throughout the duration of scan.

In conclusion, myocardial hibernation is inherently an unstable state from which patients can progress to more severe cardiac condition and death or return to a better situation with timely coronary revascularisation. In accordance with the concept of unstable angina in coronary insufficiency, we have proposed recently the term of "unstable myocardial" in cardiac hibernation emphasising the life-saving ability of a time-honoured coronary revascularisation.

The downside of the technique proposed by Vahlhaus et al., is that it is hazardous to submit a patient to a major interventional procedure with general anesthesia and cardiopulmonary by-pass without previously assessing the benefits and risk of such a method. The strength of the technique is its safety and ability to identify by a simple predictor of outcome, which is derived from basic information captured during a non-invasive examination. The patients are not subjected to risky and costly procedure before a revascularisation attempt in this era of judicious medical practice and cost-containment. The presence of at least 20% of viable myocardium is mandatory for an increase in left ventricular ejection fraction after coronary revascularisation, in the absence of peri-operative myocardial infarction, with a time window of 3–6 months. Regarding the Dobutamine-Atropine echocardiography, at least 5 g or 6% of total myocardium is necessary for genesis of wall motion abnormality. Moreover, myocardial damage of less than 25% of ventricular wall thickness or less than 3% of left ventricular mass is not going to produce an abnormal segmental motion. However, the dobutamine test has a low sensitivity and may overlook potentially viable myocardium. The recently developed Doppler Strain Imaging can more easily detect the myocardial viability with decreased systolic lengthening and increased post-systolic shortening. The tissue Doppler measurement of a velocity, at the epicardial and endocardial layers, of less than 5 and 11 cm/s, respectively, allows also the recognition of myocardial hibernation. Concerning the magnetic resonance imaging we have the structural T1 and T2 weighed-ECG Gated technique, functional Gradient-Echo, Harmonic phase, Tagged MRI and Resonance Spectroscopy for "metabolic biopsy".

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