Arterial remodelling and eccentricity of plaque

Sin.—We read with interest the article by von Birgelen and colleagues in which they found both vessel and plaque volume in lesions with inadequate compensatory enlargement to be smaller than in lesions with adequate compensatory enlargement.1 They also found that the eccentricity index (EI)—calculated as minimum wall thickness divided by maximum wall thickness—was higher in lesions with inadequate remodelling than in those with adequate remodelling. Because endothelium, the thicker eccentricity produced by a thick wall with plaque and a thin plaque free wall in lesions with adequate remodelling may support that hypothesis as mentioned by Varnava.2 The EI, however, would decrease in relation to the plaque enlargement with an increase in the maximum thickness even if the amount of plaque free endothelium is unchanged. The assumptions that both arterial contour and luminal contour are true circles lead to the following equations:

1. \[ P + L = \pi \times (D_{\text{max}} + D_{\text{min}} + D_{\text{wall}})/2 \]

where \( P \) is plaque area, \( L \) is luminal area, \( D_{\text{max}} \) is minimum wall thickness, \( D_{\text{min}} \) is maximum wall thickness, and \( D_{\text{wall}} \) is luminal diameter.

2. \[ L = \pi \times D_{\text{max}}^2/2 \]

3. \[ EI = D_{\text{min}}/D_{\text{wall}} \]

Finally, the equation, \[ D_{\text{wall}} = 2 \times EI \times (P/L)^{1/2} - L^{3/2} \text{[in } \pi \text{]} \times (EI + 1) \], can be obtained from equations (1), (2), and (3).

For example, lesion A with \( L = 2.4 \text{ mm} \), \( P = 10.9 \text{ mm} \), and \( EI = 0.28 \) has a \( D_{\text{wall}} \) of 0.518 mm; lesion B with \( L = 2.3 \text{ mm} \), \( P = 15.2 \text{ mm} \), and \( EI = 0.21 \) has a \( D_{\text{wall}} \) of 0.522 mm. Although the EI of lesion B is smaller than that of lesion A, the calculated values of \( D_{\text{wall}} \) are comparable. Therefore, a smaller value of EI does not warrant the larger amount of preserved, normally functioning endothelium. These values used for lesions A and B are the mean values in lesions with adequate and inadequate remodelling, respectively, in the study of von Birgelen et al.

We proposed another hypothesis of mechanical deformation to explain arterial remodelling.3 This suggests that a larger length of plaque along the cross sectional wall may make a larger luminal enlargement. Although precise methods to determine the ratio of plaque free to plaque 当前的电子超声观察。\[ Heart \text{ } 1995;89:37\text{–}42 \]

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the respiratory centres (Cheyne-Stokes episodes). However, the seriousness of this depression requires emphasis.

Nocturnal desaturation in obstructive sleep apnoea has been associated with increased severity of hypertension, congestive heart failure, angina pectoris, atrial fibrillation, complete heart block, and possibly precipitated myocardial infarction. Cerebral blood flow is reduced with the anoxia of obstructive sleep apnoea. Platelet aggregation and activation are increased two- and threefold. These changes are normalised with improved oxygenation. Vgontzas and colleagues and Entzian and colleagues identified sleep apnoea as a risk factor for myocardial infarction, and tumour necrosis factor α and interleukin 6 with anoxia.

Staniforth et al stated that “only 4% of their subjects were obstructive and no subject fulfilled the diagnostic criteria for obstructive sleep apnoea.” However only 41 (of 104) patients had polysomnography. One wonders about the other patients. Mechanic obstruction should be treated mechanically. The authors emphasise beautifully the serious desaturation that occurs in congestive heart failure, but the need for appropriate treatment goes short shrift.

It appears that all congestive heart failure patients who have nocturnal saturation determined and, if low, properly treated (with supplemental oxygen). Polysomnography is warranted in all who are anoxic with nocturnal sleep apnoea. If anoxia is present and oxygen does not correct it, and if an obstructive cause is discovered, continuous positive airway pressure or partial resection of the tongue (where it obstructs the pharynx) may be indicated. The surgery is relatively minor and well tolerated. Weight reduction for the obese is an ideal rarely realised; 20–30% of patients with obstructive sleep apnoea are not obese. Snoring without apnoeaic periods may be the only sign of inadequate ventilation.

Most patients with congestive heart failure are hospitalised at some time. As part of their evaluation, documentation of nocturnal oxygen saturation would be worthwhile. If anoxemia is present, the effect of oxygen supplementation could be recorded and failure to oxygenate properly would suggest a mechanical obstruction. Polysomnography could be done to document the need for mechanical relief (with continuous positive airway pressure, weight reduction, or surgery). If oxygen treatment alone relieves the anoxia, then clearly the patient should be given oxygen at home. The effects of anoxemia are so pernicious that we should not be constrained to withhold oxygen if the levels are borderline.

One wonders if the brain damage manifested by the Cheyne-Stokes breathing pattern is not a result of chronic anoxia. I am not sure we know the lowest safe level of cerebral oxygenation. Hopefully, these techniques will improve the very bleak outlook for the congestive heart failure patient.

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**This letter was shown to the authors, who reply as follows:**

The harmful effects of obstructive sleep apnoea are well documented and are summarised in the letter by Ritter. What is poorly recognised, especially in the UK, is the role of central sleep apnoea (Cheyne-Stokes respiration) as an independent marker of poor prognosis in heart failure. The mechanism behind this is unclear, but our results did not support a causal link between apnoea associated nocturnal desaturation and serious ventricular dysrhythmia. It may be related to activation of the sympathetic nervous system, and we have recently reported the beneficial effects of oxygen treatment on Cheyne-Stokes respiration and sympathetic activation in heart failure.

We accept the criticism that only 41 of our 104 subjects underwent sleep studies. Polysomnography is the gold standard test for diagnosing sleep apnoea, but it is expensive and for that reason we used it only in a representive subgroup of our patients. These studies identified around 3100 apnoeas, 96% of which were central in origin, and no patient met the standard diagnostic criteria for obstructive sleep apnoea. The total number of events we examined was large, and consequently we do not feel that the quality of our data has suffered. Cheyne-Stokes respiration is common in patients with stable controlled heart failure; prevalences of 27–51% are reported in the literature. We documented a prevalence of 22%. Our study shows that obstructive sleep apnoea is an unlikely cause of nocturnal desaturation in heart failure patients who do not have a history of pulmonary disease. In this setting, overnight pulse oximetry is an effective screening tool for Cheyne-Stokes respiration. It is important to screen patients with heart failure for Cheyne-Stokes respiration on prognostic grounds. To avoid overdiagnosis, however, the best time to do this may be following discharge when they are relatively stable, rather than during a hospital admission as Ritter suggests. The major advantages of oximetry over polysomnography as a home screening tool are that it is cheap, easily portable, and widely available.

We did not discuss the possible treatment strategies for Cheyne-Stokes respiration in heart failure as the main thrust of our paper was to establish its prevalence, and to demonstrate the usefulness of oximetry as a screening tool. Oxygen, carbon dioxide, continuous positive airway pressure, aminophylline, and sedatives have all been advocated. The advantage of one treatment modality over another remains to be proved, as indeed does the existence of a survival benefit from treating Cheyne-Stokes respiration in heart failure.