Editorials

So what’s wrong with being fat?

See page 46 for the article to which this Editorial refers

This is a question that my 55-year-old patient asked me in clinic last week. This was not the first time that I had heard this question. Indeed, obesity has become so commonplace in the United States that the asthenic individual has become the exception rather than the rule. With the increasing prevalence (and incidence, too, for that matter) of obesity in our society, patients have come to perceive this state of body habitus as the norm rather than the exception. ‘What’s the matter with being a little overweight?’ my patients frequently ask, ‘Everyone in my family is fat, so why not me? What difference does it make?’

I begin my ‘fat’ talk once again. It seems that I am doing this ever more frequently and probably I am. In this little sermon on obesity, I stress the many health risks inherent in carrying around excess poundage: coronary risk factors, gallstones, diabetes, colonic adenomas, degenerative joint disease, sleep apnoea, and so forth. I emphasize the importance of modest and gradual weight loss as compared with the ‘ping pong’, up and down, nature of so many of my patients’ weight loss programmes. ‘Bouncing your weight up and down may even put you at greater risk for heart disease’ I continue, hoping that my patient will take the message to heart. ‘Regular exercise such as daily walking is a great help in losing weight. Try to cut back on carbohydrates and saturated fat in your diet. Would you like a consultation with one of our dieticians?’

My daily preaching often goes unheeded and leads me and my colleagues to become cynical about the possibility of ever getting our patients to lose weight. This is the price to be paid in a society that gratifies itself with every conceivable type of food taken in excess alongside of widespread physical inactivity.

In this issue, Ashton and colleagues have given me further ammunition to use in the battle against excess poundage. These authors report on the findings of a large, on-going, epidemiological trial involving more than 14 000 employed English women between the ages of 30 and 64 years. Each of these women underwent an examination that included a questionnaire followed by the collection of certain physical exam and laboratory data. From this information, body mass index (a measure of relative obesity) was calculated and this number was, in turn, compared with a variety of coronary risk factors. Statistical correlations were drawn leading to some anticipated and unanticipated results.

In the anticipated category was the finding that increasing obesity correlated nicely with rising blood pressure, lipid levels, and blood glucose, all major coronary risk factors. Thus, the fatter the woman, the worse was her combined burden of coronary heart disease risk factors. However, obesity alone was not a good predictor of coronary heart disease risk. Thus, 84% of women who were not obese (body mass index ≤ 25 kg m⁻²) had a predicted 10 year risk of coronary heart disease of less than 5%. Furthermore, 60% of the most obese women (body mass index ≥ 30 kg m⁻²) were also in this low risk category. This observation has been made before, i.e. obesity per se is not a major risk factor once more prominent risk factors that correlate with obesity have been removed. An obese person without elevated blood pressure or abnormal blood lipids or glucose will not be at major risk for the development of coronary artery disease. As Ashton and colleagues point out, the ‘key issue is not overweight per se, but rather the metabolic consequences of excess adiposity’.

The authors advise physicians not to concentrate on weight alone in advising these patients about health maintenance since this can lead to ‘unrealistic interventions’. I am not in total agreement with this last point since the authors are only looking at the relationship between obesity and coronary heart disease risk factors.

The obese patient is at risk for many other problems as noted earlier in this essay. Thus, an obese patient who is normotensive, normolipaemic, and euglycaemic may still develop severe, crippling degenerative arthritis or sleep apnoea as a result of obesity. Consequently, physicians, including cardiologists, need to look at the entire picture of a particular patient’s health and not just their risk for coronary artery disease. Indeed, the authors recognize this point and make it later in their discussion. I am concerned, however, that a superficial reading of this excellent paper might lead some
physicians to come away with the message that obesity is not a health risk as long as traditional coronary atherosclerotic risk factors are normal.

Two other points concerning obesity deserve mention. First, not all obesity confers the same degree of abnormality in coronary heart disease risk factors. The patient with abdominal obesity (the ‘apple’ form of obesity often associated with males) is linked to insulin resistance and hence significant risk for the development of atherosclerosis. Thigh and buttock obesity (the ‘pear’ form of obesity often associated with females) confers minimal coronary heart disease risk probably because fat in this zone is not as metabolically active as abdominal fat. Another point made by Ashton et al. that deserves repeating is that modest weight loss is capable of remarkable improvement in abnormal coronary heart disease risk factors. It has often been observed that modest (~10% of body weight) loss of weight produces marked amelioration in elevated blood pressure, abnormal serum cholesterol, and hyperglycaemic tendency. I am in complete agreement with Ashton et al. when they advise moderate weight loss for obese patients. Demanding that the patient seek to reach their ideal body weight is often unrealistic and discourages compliance with the prescribed programme of diet and exercise. In the end, ‘the enemy of good is perfect’, that is, we should strive to enlist our patients in a programme that produces moderate, sustained weight loss rather than advising a draconian strategy that eventually fails to induce the patient to lose any weight at all. As recently pointed out by the National Task Force on the Prevention and Treatment of Obesity here in the U.S., the best strategy for weight loss would appear to be one of the moderate calorie restriction, increased activity (that is, regular exercise), and a supportive programme of behavioural modification to assist patients in remodelling their eating habits and style.[2]

J. S. ALPERT
R. S. FLINN
I. P. FLINN
Department of Medicine,
University of Arizona College of Medicine,
Tucson, Arizona, U.S.A.

References

The interrelation between carotid, femoral and coronary artery disease

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Atherosclerosis and thrombosis are the main pathological processes involved in ischaemic stroke, coronary heart disease and peripheral arterial disease. It has long been known from pathological studies that atherosclerosis is a systemic disease[1]. Although certain areas of the vasculature are particularly prone to atherothrombotic disease, it is relatively unusual for individuals to have disease localized solely to one area. Moreover, clinical manifestations of disease in one arterial territory are strongly predictive of clinical events in other territories[2–5]. For example, patients presenting with cerebral ischaemic events have a 5-year risk of myocardial infarction of 10–25% and a 5-year risk of non-stroke vascular death of 10–15%[1–5]. These risks are 5–10 times higher than general population controls. Even the risk of recurrent events in the same vascular territory is increased in those patients with symptomatic disease in other territories[3,5].

Recent developments in non-invasive imaging have allowed the prevalence of disease at multiple sites (usually the carotid, femoral and coronary arteries) to be measured in large numbers of individuals during life. This has led investigators to consider the clinical usefulness of measuring disease in arteries distant from the vascular territory of immediate interest. Some studies have related carotid artery disease and peripheral vascular disease[6,7], but most work has...