Sympathetic nervous system activity in the obese hypertensive patient: potential role for central alpha-adrenoreceptor agonists

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The relationship between body weight and blood pressure

In most industrialized populations a strong association between blood pressure and body weight has been established. Several large-scale studies in the United States have confirmed the relationship between obesity and hypertension (the Framingham Study (Kannel et al. 1967); the Evans Country, Georgia Study (Tyroler et al. 1975); the Community Hypertension Evaluation Clinic (Stamler et al. 1978); Hypertensive Detection and Follow-up Program Cooperative Group (1979)). In the Evans county, Georgia study it was demonstrated prospectively that weight gain can increase blood pressure. Over the six-year period of the study, weight gain was associated with a two-fold possibility of developing hypertension. The converse was also true: subjects who were hypertensive at outset gained more weight. In those who were both obese and hypertensive at the outset, a weight reduction programme averaging 8 kg per patient produced very pronounced decrements in both systolic and diastolic blood pressure over a year. Comparing black and white subjects, it was concluded that obese white hypertensives benefited more than blacks from weight loss.

Amongst the million people screened in the Community Hypertension Evaluation Clinic Study, hypertension was two to three times more common in overweight subjects, especially between the ages of 20 and 39 years (Stamler et al. 1978). This relationship was further documented by the Hypertensive Detection and Follow-up Program , Cooperative Group (1979), which showed that 60% of participants chosen for having mild hypertension were also 20% above ideal body weight.

The Framingham Study has yielded important information on the relationships between age, the onset of obesity and changes in blood pressure (Kannel et al. 1979; Gordon and Kannel 1976). The results show that weight is related to blood pressure most strongly in persons under the age of 20 years; as age increases the relationship weakens, especially for diastolic blood pressure. The offspring of the original cohort in the Framingham Study also demonstrated a stronger relation between weight and blood pressure in early adulthood, especially in men (Kannel and Gordon 1968). Other smaller studies have shown that overweight children and adolescents already show higher blood pressure levels than their non-obese counterparts (McCue et al. 1979; Lynds et al. 1980), implying that weight-related blood pressure elevation can begin early in life. In fact, Schacter and co-workers (1982) have noted a significant correlation between weight and blood pressure during the first two years of life. The importance of minimizing weight gain at certain critical periods of life, such as young adulthood, have been emphasized in the prevention of subsequent hypertension (Havlik et al. 1983).
Longitudinal studies have provided further evidence that weight changes can alter blood pressure. The Framingham Study, for example, followed its initial cohorts prospectively and observed that a 10% increase in weight between examinations was accompanied by a 7 mm Hg increase in systolic blood pressure (Ashley and Kannel 1974). In a 30-year follow-up of initially thin, normotensive aviators, those within the upper limits of systolic blood pressure at the end of the study were also the ones who gained the most weight (Harlan et al. 1973). An almost linear relationship between weight and blood pressure could be implied from these studies. But several additional variables such as age, race, sex, types of obesity, methods of measuring obesity and blood pressure, together with family and environmental factors all enter into a seemingly simple relationship.

Bergland et al. (1982) have compared the type of obesity and the incidence of associated hypertension. Body cell mass and fat cell number were unrelated to blood pressure but fat cell size (hypertrophic obesity) was positively correlated. In a careful measurement of body composition and fat cell properties, including underwater weighing and fat cell biopsy, blood pressure correlated best with total body fat mass and fat cell number but not lean body mass or fat cell size (Siervogel et al. 1982). It is also not clear whether skin-fold thickness or body weight is the better predictor of blood pressure. Simple measurements of waist girth may be as good a predictor of blood pressure as more complicated parameters (Berglund et al. 1982). It is also important to bear in mind cuff size. A recent study has re-emphasized the need to measure arm circumference in obese subjects and to use an appropriate cuff size (Maxwell et al. 1982). The use of the regular cuff, it was suggested, may have led to an overestimation of the prevalence of hypertension in obese subjects. There may also be differential effects of obesity on blood pressure. A 32-year follow-up of young men indicated that increased body weight led to higher blood pressure, but it affected systolic pressures more than diastolic (Gillum et al. 1982).

**Cardiovascular risk of obesity and hypertension**

It is clear that obesity, either directly or indirectly, is a major risk factor for cardiovascular disease. The Framingham Study showed that, in comparison with subjects of normal weight, overweight subjects are four times more likely to have coronary artery disease, as manifested by angina pectoris and sudden death, and seven times more likely to have a stroke (Kannel et al. 1967). Hypertension in obese subjects appeared to be the most likely factor in these cardiovascular conditions. The Framingham Study has also suggested, however, that obesity itself may be an independent risk factor for cardiovascular disease, acting through yet unidentified mechanisms (Hubert et al. 1982). Recent evidence indicates that obesity increases cardiac work and is associated with a higher incidence of congestive heart failure (Messerli 1982).

The compounded effect of other risk factors – such as hypertriglyceridaemia, hyperuricaemia, hypercholesterolaemia, hyperinsulinaemia and low serum HDL-cholesterol – although less important, must also be considered in obesity-related cardiovascular risk. Indeed, less than 10% of obese subjects are entirely free of one or more risk factors, and these are mainly younger, less obese subjects (Berchtold 1981, Patel et al. 1980). The Ad Hoc Committee of the Build and Blood Pressure Study (1980) has reconfirmed