*For Debate . . .***Is there an ideal body weight?**

R J JARRETT

The concept of ideal weight standards disregarding age is said to have been developed by the late Dr Louis Dublin, once chief actuary of the Metropolitan Life Insurance Company.¹ This body published tables of "desirable weight" for health education in the early 1940s.^{2,3} Subsequently, the build and blood pressure study, conducted by the Society of Actuaries,⁴ resulted in the creation of revised weight tables, introducing the concept of "frame size," which was not based on measurement of skeletal dimensions but arbitrarily divided the distribution of relative weights on the assumption that skeletal size was associated with a person's position in that distribution. The suggested weights were derived from data on mortality of people insured between 1935 and 1953 and followed to 1954. Measured heights and weights were of people wearing "indoor clothing" and shoes and included 20% of self reported values.

The Fogarty Center conference on obesity, 1973, recommended guidelines for body weight, based on the Metropolitan Life Insurance tables of 1960, but adapted for height without shoes and weights without clothes.⁵ The concept of frame size was abandoned; instead an acceptable weight range was given, which, incidentally, would allow me (height 173 cm, weight 70 kg) to weigh between 60 and 75 kg. The conference also suggested acceptable ranges for body mass index (body mass index = weight (kg)/height (m)²), which were 20.1-25.0 for men and 18.7-23.8 for women. These tables were reproduced and commended in the report on obesity of the Royal College of Physicians.⁶

A second build study was published in 1979 with new tables of desirable weights—higher than in the previous study—based on information on the mortality of insured people from 1950 to 1972.⁷ These would allow me to weigh between 57 and 80 kg.

Possibly and understandably confused by the conflicting advice offered, the National Institutes of Health convened a consensus development conference on the health implications of obesity. A consensus statement was published separately⁸ from the papers commissioned from the participants, which was unfortunate for it is hard to see how much consensus could have been achieved given the diverse views expressed by individual participants. Thus one author, reviewing the studies on obesity, atherosclerosis, and coronary artery disease, concluded that, despite its plausibility, the hypothesis that obesity causes atherosclerosis could not be supported⁹; a group of authors argued for age related norms for weight¹⁰; another author, reviewing epidemiological studies of obesity and commenting on the different results and interpretations stated, rather pessimistically, "We cannot really expect adequate studies or new analytical methods to resolve the discrepancies."

The consensus statement recommended weight reduction for "persons with excess body weight of 20% or more above desirable weights in the Metropolitan Life Insurance Company tables (using

the midpoint of the range for a medium-build person)." This would differ depending on whether the 1959 or 1983 tables were used. Thus the upper body mass index limits would be 26.4 and 25.8 for men and women, respectively, using the 1959 tables, compared with 27.2 and 26.9, respectively, using the 1983 tables. In either case, the values are substantially higher than those recommended by the Fogarty Center and the Royal College of Physicians.

Why is the advice discrepant?

Although there is no dispute that at the upper extremes of body weight distribution longevity is diminished and various morbid events more common, the place in that distribution at which enhanced risk appears does not emerge clearly from the many studies designed to investigate the problem. One can compile a long list of potential reasons why results apparently differ,¹¹ including selected (unrepresentative) populations, inadequate standardisation of weight/height measurement, different end points, failure to consider important covariates—for example, smoking—and inadequate or inappropriate statistical analysis. Furthermore, as always in epidemiology, the possibility that what is true of one population is not true of another must be considered.

Nevertheless, given the association between body weight and several accepted cardiovascular risk factors, plus the observation that weight reduction diminishes the apparent risk, it is difficult for many people to discard the belief that being overweight, even to minor degree, increases the risk of premature death. This opinion was strongly expressed in the Royal College of Physicians' report,⁶ where much emphasis was placed on the build studies of the insurance industry and on the American Cancer Society study.¹² Other studies were criticised as being too short, too small, or both, though many of these criticisms are refuted in Barrett-Connor's review, in which she presents new data from long term follow up for several of the North American studies.⁹ The defects of the build studies have been recently rehearsed: not the least is that the data refer to policies not people; thus one person may have several policies, and policies may terminate for reasons other than death. Furthermore, the statistical procedures used are, to quote Knapp, "almost unfathomable."¹³ I wonder about the "almost."

The study conducted by the American Cancer Society had the considerable advantage of large numbers but was not unselected, and heights and weights were self reported.¹² Its analysis was particularly poor because the potentially confounding effects of smoking were either not considered or were crudely analysed. The Royal College of Physicians' report presented recalculated data from the American Cancer Society study showing mortality risk ratio (all ages) against body mass index for non-smokers and those smoking more than 20 cigarettes a day.⁶ For both groups and for both sexes the relation was J shaped—that is, the mortality ratio was higher at low body mass index than intermediate body mass index values, a phenomenon shown in several other prospective studies.¹⁴⁻¹⁶ It has been claimed that this J or U shaped relation is due to the effects of smoking on body weight,^{17,18} but this was not true in the American Cancer Society study or in the Whitehall study¹⁵ (see

figure), where the shape of the relation of body mass index to mortality from all causes was clearly related to age and little affected by adjustment for smoking.

These non-linear associations plus the potential confounding by smoking raise problems in statistical analysis, particularly in performing multivariate or multiple logistic analyses. Garrison *et*

disease, including death.¹⁹ This study did not even mention the fairly high mortality in the lower end of the weight distribution previously reported from the Framingham study^{14, 18} and ignored the competing causes of mortality. It is also worth noting that smoking rates were much higher in the initial Framingham cohort than current rates in the United States and the United Kingdom.

Whitehall study. (a) All cause mortality by fifths of body mass index distribution and five year age groups. Limits are (1) <22.4, (2) 22.4-24.0, (3) 24.1-25.4, (4) 25.5-27.0, (5) >27.0. (b) All cause mortality, standardised for smoking habit, by body mass index quintile, and 5 year age groups.

*al.*¹⁸ using data from the much analysed Framingham study, stated that "this finding (that the U shaped or J shaped univariate relation between mortality and relative weight results from the mortality risks associated with cigarette smoking) implies that standard logistic regression analysis cannot be used for significance testing of the relationship between Metropolitan Relative Weight and mortality . . . adequate statistical modelling of the mortality-relative weight relationship may not be currently possible." Yet in the same year three of the authors published a study using standard logistic regression analysis claiming that metropolitan relative weight was independently predictive of various end points of cardiovascular

Weight and morbidity

Although ideal body weight was originally defined in terms of mortality risks, some authors, perhaps because of the inconsistent results of studies of mortality, have emphasised the association of body weight with blood pressure, serum cholesterol concentrations, and diabetes mellitus.⁸ These associations, as mentioned earlier, lend plausibility to the hypothesis that overweight and obesity are risk factors for cardiovascular disease. At least for the blood pressure association, however, the situation has been complicated by the results of two prospective studies. In California 1727 men aged 50-79 years were followed for nine years. The population was divided into two on the basis of body mass index (<27 and ≥27) and systolic blood pressure (<160 and ≥160).²⁰ Age adjusted death rates from cardiovascular and ischaemic heart disease and all causes were highest in the non-obese men; in multivariate analysis systolic blood pressure was an independent predictor of mortality only in the non-obese. In Paris two populations were studied—7704 male civil servants aged 47-53 years (Paris prospective study) and 19 168 men aged 40-69 years (investigations précliniques study).²¹ There was a mean follow up of 11.2 and 7.6 years, respectively. The report was concerned principally with cardiovascular mortality, and hypertension was defined as a diastolic blood pressure greater than 95 mm Hg. In both populations the ratio of cardiovascular mortality in hypertensive compared with normotensive men declined in successive fifths of body mass index distribution. Thus both the Californian and Parisian studies suggest that a raised blood pressure is less predictive of mortality in the presence of overweight, perhaps because of different mechanisms underlying the hypertension.

With regard to diabetes, as many studies testify, obesity predisposes to (or causes?) non-insulin-dependent diabetes. There are, however, only four prospective studies that have related degree of obesity to subsequent incidence of diabetes.²²⁻²⁵ In none was the relation between body mass index (or relative body weight) and incidence of diabetes linear. An Israeli study found by glucose tolerance testing 132 non-insulin-dependent diabetics among 2140 people (known diabetics excluded) whose height and weight had been measured 10 years earlier.²⁵ Prevalence at the second examination showed little difference across the range of body mass index below a value of 31. Results in the more exotic Pima Indians (who have the highest incidence of diabetes yet recorded) were similar,²⁴ with age-sex adjusted incidence rates being not significantly different in body mass index categories 20-25 and 25-30 and incidence rates rising steeply in the substantial segment of that population with body mass index values exceeding 30.

Fat distribution

Several investigators have reported that fat cells from different sites also differ in their metabolism.²⁶ Fat distribution has been estimated simply in several population studies by measuring the circumference at waist and hips and expressing the waist:hip ratio. Correlations have been observed between the waist:hip ratio and body mass index, blood pressure, and serum cholesterol, triglycerides, insulin, glucose, and urate concentrations, though not with absolute consistency between populations. In Gothenburg two prospective studies have examined the waist:hip ratio as a putative risk factor. One study followed 1462 women aged 38-60 years for up to 12 years.²⁷ At baseline the waist:hip ratio was positively and significantly related to age, systolic blood pressure, serum cholesterol and triglyceride concentrations, blood glucose values, proportion of cigarette smokers, body mass index, and sum of skinfold thicknesses. Prospectively, age adjusted values of body mass index

and skinfold thicknesses were significantly related only to the incidence of myocardial infarction, whereas the waist:hip ratio was significantly related to myocardial infarction, angina pectoris, stroke, and all cause mortality. For stroke and mortality from all causes, however, and, to a less extent, for myocardial infarction, an increased age standardised incidence was obvious in only the top 5% of the waist:hip ratio distribution.

The 792 men were from the 1913 birth cohort, aged 54 years at inception into the study, and were followed for 13 years.²⁸ At baseline there were positive and significant correlations between the waist:hip ratio and blood pressure (systolic and diastolic), body mass index, and sum of skinfold thicknesses, but not with serum cholesterol and proportion of cigarette smokers (unlike their female counterparts). Prospectively, the waist:hip ratio was significantly related to the incidence of stroke and ischaemic heart disease, but not to mortality from all causes. Body mass index and skinfold thicknesses were not significantly related to any of these end points.

Thus, although the waist:hip ratio seemed to be more predictive of cardiovascular morbidity than body mass index or skinfold thickness in both sexes, the relation with mortality was significant only in women, and associations of waist:hip ratio with other variables also differed between the sexes. Furthermore, in the men from Gothenburg a curious phenomenon was reported: the lowest risk of ischaemic heart disease occurred in men with high body mass index and low waist:hip ratio, with the highest risk in men with low body mass index and high waist:hip ratio. A similar trend occurred for mortality from all causes. We are familiar with obesity clinics: can we now expect the advent of fat redistribution clinics?

As an inveterate empiricist, I find myself unable to answer the question posed in my title. Indeed, the concept of ideal body weight might be better left to aesthetics than statistics.^{29 30} In either event it appears to be in the eye of the beholder.

I thank Martin Shipley for the analyses depicted in the figure.

References

- 1 Keys A. *Seven countries: a multivariate analysis of death and coronary heart disease*. Cambridge, London: Harvard University Press, 1980.
- 2 Ideal weight for women. *Statistical bulletin of the Metropolitan Life Insurance Company* 1943.
- 3 Ideal weight for men. *Statistical bulletin of the Metropolitan Life Insurance Company* 1943.
- 4 *Build and blood pressure study*. 1959. Vol 1. Chicago: Society of Actuaries, 1959.
- 5 Bray G, ed. *Obesity in perspective*. Vol 2, part 1. Washington, DC: National Institutes of Health, 1975. (DHEW publication No (NIH) 75-708.) (Fogarty International Center. Series on Preventive Medicine.)
- 6 Obesity: a report of the Royal College of Physicians. *J R Coll Physicians Lond* 1983;17:3-58.
- 7 *Build study* 1979. Chicago: Society of Actuaries and Associations of Life Insurance Medical Directors, 1980.
- 8 Health implications of obesity: National Institute of Health consensus development statement. *Ann Intern Med* 1985;103:147-51.
- 9 Barrett-Connor E. Obesity, atherosclerosis, and coronary artery disease. *Ann Intern Med* 1985;106:1010-9.
- 10 Andres R, Elahi D, Tobin JD, Muller DC, Brant L. Impact of age on weight goals. *Ann Intern Med* 1985;103:1030-3.
- 11 Stallones RA. Epidemiologic studies of obesity. *Ann Intern Med* 1985;106:1003-5.
- 12 Lew EA, Garfinkel L. Variations in mortality by weight among 750,000 men and women. *J Chronic Dis* 1979;32:563-76.
- 13 Knapp TR. A methodological critique of the 'ideal weight' concept. *JAMA* 1983;250:506-10.
- 14 Sorlie P, Gordon T, Kannel WB. Body build and mortality: the Framingham study. *JAMA* 1980;243:1828-31.
- 15 Jarrett RJ, Shipley MJ, Rose G. Weight and mortality in the Whitehall study. *Br Med J* 1982;285:535-7.
- 16 Rhoads GG, Kagan A. The relation of coronary disease, stroke, and mortality to weight in youth and in middle age. *Lancet* 1983;i:492-5.
- 17 Björntorp P. Obesity and risk of cardiovascular disease. *Acta Med Scand* 1985;218:145-7.
- 18 Garrison RJ, Feinlieb M, Castelli WP, McNamara PM. Cigarette smoking as a confounder of the relationship between relative weight and long-term mortality: the Framingham study. *JAMA* 1983;249:2199-203.
- 19 Hubert HB, Feinlieb M, McNamara PM, Castelli WP. Obesity as an independent risk factor for cardiovascular disease: a 26-year follow-up of participants in the Framingham heart study. *Circulation* 1983;67:968-77.
- 20 Barrett-Connor E, Khaw K-T. Is hypertension more benign when associated with obesity? *Circulation* 1985;72:53-60.
- 21 Cambien F, Chretien JM, Ducimetiere P, Guize L, Richard JL. Is the relationship between blood pressure and cardiovascular risk dependent on body mass index? *Am J Epidemiol* 1985;122:434-42.
- 22 Westlund K, Nicolaysen R. Ten-year mortality and morbidity related to serum cholesterol. *Scand J Clin Lab Invest* 1972;30 (suppl 127):3-24.
- 23 Modan M, Karasik A, Halkin H, et al. Effect of past and concurrent body mass index on prevalence of glucose intolerance and type 2 (non-insulin-dependent) diabetes and on insulin response. *Diabetologia* 1986;29:82-9.
- 24 Knowler WC, Pettitt DJ, Savage P, Bennett PH. Diabetes incidence in Pima Indians: contributions of obesity and parental diabetes. *Am J Epidemiol* 1981;113:144-56.
- 25 Ohlson L-O, Larsson B, Svardsudd K, et al. The influence of body fat distribution on the incidence of diabetes mellitus: 13.5 years of follow-up of the participants in the study of men born in 1913. *Diabetes* 1985;34:1055-8.
- 26 Kissebah AH, Vydelingum N, Murray R, et al. Relation of body fat distribution to metabolic complications of obesity. *Journal of Clinical Endocrinology* 1982;54:254-60.
- 27 Lapidus L, Bengtsson C, Larsson B, et al. Distribution of adipose tissue and risk of cardiovascular disease and death: a 12 year follow up of the participants in the population study of women in Gothenburg, Sweden. *Br Med J* 1984;289:1257-61.
- 28 Larsson B, Svardsudd K, Welin L, et al. Abdominal adipose tissue distribution, obesity and risk of cardiovascular disease and death: a 13 year follow up of participants in the study of men born in 1913. *Br Med J* 1984;288:1401-4.
- 29 Clark K. *The nude*. Harmondsworth: Penguin Books, 1960.
- 30 West KM. Culture, history and adiposity, or should Santa Claus reduce? *Obesity and Bariatric medicine* 1974;3:48-52.

(Accepted 26 June 1986)

MEDICINE AND THE MEDIA

FOR CLIVE, waking each morning was like throwing back the curtains on a coma he couldn't explain. He had been dead, he puzzled. He had been deaf, dumb, and blind. Now he could see, and in colour. The next day, though, brought another baffling rebirth: yesterday's had gone, sucked into an impenetrable amnesia. It was his wife who struck the right metaphor: "A great deal of his time is spent in agony," she said, "trapped in the present."

Fortunately, herpes encephalitis is rare. Surviving it is rarer. Clive Wearing, the subject of *Prisoner of Consciousness* (Channel 4, 14 August), did. But its aftermath was a brutalised memory, a devastating inability to remember new events or to recall all but a smattering of the past.

The illness documentary is of course a well established piece of television theatre. We gasp. We pity. We don't know we're born. Then we unplug the set and pour out a nightcap. Sympathy served neat soon runs out. It's a genre that craves wary walking if it is to stand out above the routine. The excellence of *Prisoner of Consciousness* lay not only in its sensitive touch but in its attempts to use Clive's disability—and in particular the retention of his consider-

able musical skill—to understand the nature of memory; director John Dollar neatly switched technique from fly on the wall to Jonathan Miller on the sofa.

Clive had forgotten his wedding but knew he was married. He couldn't believe that he had ever conducted a choir but, brought to church, his arms flashed faultlessly once the music started (and he sang the words too). Watching himself on screen later, he grew angry. I know nothing about that, he huffed. Later came hesitation before sitting at the church organ, dubious of his wife's encouragement. Within seconds his fingers were stroking the layered keys and dexterously pulling out rows of organ stops. The emotional stops were out too. Meeting his wife after a ten minute absence that he believed had been weeks, perhaps years, he hugged her, sobbing, like a soldier in a quayside reunion.

Jonathan Miller did his best to explain, hanging his observation on currently fashionable models of registration, retrieval, and the rest. The task, needless to say, was hopeless. Though his shrug was always eloquent, he was the gloss on our fundamental ignorance. Eventually the coding system began to sound like an officious usherette—"Music? You want the right hemisphere. Language on the left please." Despite our black boxes and our careful flow charts, we're still shaking the wireless to see if there's a man inside. —LOUIS APPELEY, registrar in psychiatry, London.