Benign prostatic obstruction is probably the condition that most urologists want to treat. The term can be taken to mean that cancer of the prostate has been excluded so far as possible by digital rectal examination with the possible addition of an estimation of prostatic specific antigen and transrectal ultrasonography. Benign prostatic obstruction also implies that objective evidence of obstruction exists. In patients with symptoms this evidence would be reduced flow rates or raised voiding pressures in combination with low flow rates.

If we can rid ourselves of imprecise and improperly used terms we will be better able to evaluate our elderly male patients. Any filling or voiding symptoms can be documented for what they are, and, if the symptoms are sufficiently bothersome, further evaluation can be discussed with the patient. The patient can be told that when further evaluation (urine flow studies or pressure flow studies) is carried out and patients are selected for surgery according to their results then the results of surgical procedures are excellent. If the suggestions above are followed patients who will benefit from surgery will be identified more accurately and our limited resources will be used to better the twin goals of improved quality of life and cost effectiveness.

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Milk and bones

You are what you drink

"You are what you eat" has been an popular aphorism in the many branches of medicine in which nutritional aberrations are suspected to have a role in disease. In the pathophysiology of osteoporosis the nutritional questions have always centred on calcium. The importance of osteoporosis is clear, with the current epidemic of hip fractures increasing as populations worldwide gradually age: the global load of hip fracture is expected to treble to over six million cases a year by 2050.[1]

In this week's journal Sean Murphy and colleagues confirm the benefit of a high calcium intake (as milk) on bone mineral density (p 939).[2] In a community based survey of older women (aged 44-74) they found their milk consumption before the age of 25 correlated positively with current bone mineral density. This relation persisted after numerous potential confounding factors were controlled for statistically. A similar, but weaker, association was found between calcium intake during adulthood and bone mass.

Although peak bone mass is primarily under genetic control, it seems logical that to achieve adequate skeletal maturation during growth requires a plentiful supply of the building blocks of the skeleton, of which calcium is one of the most important. During peak growth in early teenage years the calcium requirement for the skeleton can be as high as 400 mg/day. To provide this amount of calcium, even if adolescents absorb calcium more than adults (and this is disputed), would require an intake of at least 1500 mg/day.[3] Dairy products provide the main source of dietary calcium in developed countries; the study's findings are therefore unsurprising and support the importance of calcium nutrition in early life.

Milk is a cheap source of calcium and one of the most bioavailable sources. Past milk consumption is easy to study because people can usually recall the relevant details. But milk is a complex food, providing other nutrients, and the observed effects on bone mineral density may result from an overall better diet and healthier lifestyle. In fact, separating out the skeletal effects of other nutrients found in high amounts in milk, such as protein and phosphorus, from those of calcium is difficult. In addition, milk contains lactose, which increases the absorption of calcium. Of the nutrients supplied by milk, dietary calcium is the single nutrient otherwise limited in a typical diet—bone mineral density associated with greater milk consumption therefore seems most likely to result from its calcium content.

A previous study evaluated a wider range of milk consumption and found that it correlated strongly with skeletal status.[4] The current study evaluated relatively low intakes. The maximal calcium intake of someone drinking a 227 ml glass of milk each day is only 650 mg/day (about 300 mg from the milk and the remainder from non-dairy sources). This may still be below the presumed "threshold" of calcium intake—that level of intake above which most people would be calcium replete and the relation between skeletal status and calcium intake would be lost.[5] This threshold, estimated at 1500 mg for adolescents,[3] may vary at different stages of life and lower at later ages.[6]

For adolescents there seems to be a critical time during which bone mineral density may be increased by optimising the intake of calcium. Only one controlled clinical study of calcium supplementation has been published in growing children.[7] In that study prepubertal children given a 1000 mg calcium supplement increased their bone mass faster than the controls (their monozygotic twin). For those twins who were past puberty supplementation produced no significant effects. In addition, the effects on bone mineral density were lost when supplementation was stopped, which suggests the need to maintain calcium intake throughout adolescence and adulthood.[8] Calcium intake needs to be sufficient to dampen down activity at skeletal remodelling sites—that is, to avoid skeletal calcium being used to keep the serum calcium concentration within its tightly controlled limits.

Calcium intake may have more impact on cortical rather than cancellous bone: a meta-analysis found little impact of calcium on spine bone mineral density.[9] Murphy and

colleagues also found that bone mineral density at the hip, where more cortical bone is present, seems more dependent on calcium consumption in youth, but this was less the case for the spine, after adjustment for covariates. Our ancestors had calcium intakes in the range of 2000 mg a day or more,[10] whereas our current diets average appreciably less—about 600 mg for adults and not much more for teenage women. With intestinal absorption averaging 30%, the average diet therefore provides only 100-200 mg calcium a day. This is barely enough to maintain calcium balance and clearly insufficient for skeletal accretion during the teenage years.

Doctors should remember that calcium will not prevent the bone loss due to other factors—for example, the loss of oestrogen at the menopause—and should advise their patients accordingly. None the less, milk is a bioavailable, relatively inexpensive source of calcium for those who can ingest it. Fortifying foods such as bread and orange juice may also help to increase the calcium intake, as would using calcium supplements for those who cannot otherwise improve their diet.

Suggestions for an increase in intake of milk or alternative sources of calcium during youth to maximise skeletal potential should not be the subject of controversy. Rather they should be a matter for public health promotion, particularly as calcium intake in the ranges found to be effective bears little or no risk to the consumer and may even afford some protection against several other diseases, such as colonic cancer and hypertension,[11 12] as well as osteoporosis.

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Bone mass measurements: reasons to be cautious

Bone mass and strength not necessarily correlated

Interpretation of a single measurement of bone mass, like evaluation of a child's height, must take age into account. Like cholesterol concentrations, bone mass is associated with risk even when values are in the "normal" range. Considering this will avoid those "below the fracture threshold" being frightened or those who have a "normal" bone mass being falsely reassured. The absolute risk of a fracture doubles with each decade after the age of 50, and, at any age, the relative risk of fracture increases 1·5-fold to twofold for each standard deviation decrease in bone mass (roughly 10%).[1]

Interpreting a change in bone mass requires caution. Problems arise from the imprecision of instruments, the increasing risks with multiple measurements of rejecting the null hypothesis when it is true (type I error), assumptions that bone density is a volumetric density, suggestions that treatment causes linear increases in bone mass, and assumptions that the pattern of bone loss is reversible.

Dual energy *x* ray absorptiometry provides the most precise measurements of bone mass. Even with this new technique, however, the size of clinically important changes in bone mass is less than the measurement error. A walk around the room causes the measurement to change by up to 6% (at the hip), which corresponds to six years of bone lost at the usual rate. Thus, for individual patients, clinically important changes in bone mass may take several years to detect. Only severe loss can be measured with two determinations. Alterations in machine function must be anticipated and careful quality control applied.[2]

In clinical studies the newer technology has improved the power to detect changes in bone mass, and a few subjects are sufficient to show dramatic changes (such as bone loss with gonadotrophin releasing hormone agonists[3]). In normal elderly subjects we estimated that a sample size of 90 over three years would be needed to detect changes in bone mass between placebo and treated groups of 0·8% a year.

Because of the propensity to measure many different anatomical locations there is an unacknowledged risk of type I errors. Commercial densitometers produce a series of related measurements, which are often analysed unquestioningly. For example, a single scan of the proximal femur yields five measurements: femoral neck, trochanteric region, intertrochanteric region, total hip, and "Ward's triangle" (three adjacent regions, their total, and a subset respectively). This process could become worse—the total body bone mineral measurements are divided into 14 areas. Will we be subjected to even larger tables of data every time a study reports bone mass? The sites of physiological interest should be carefully defined when the study is designed.

Another common problem in interpreting changes in bone mass arises when there is also change in skeletal size, which applies to children and teenagers. The only method that measures true bone density is quantitative computed tomography.[4] Dual energy *x* ray absorptiometry measures the amount of bone mineral in a projected area (areal density). Unfortunately, the term "bone density" has been loosely applied: with no change in the true bone density, the measured areal bone density will increase with growth. An independent measurement of the depth of the bone is needed to estimate the true density, and even then there is error in calculating the size of the bone.

Changes in bone mass after pharmacological treatment may not be linear over time, especially with "antiresorbing" drugs such as oestrogen, calcitonin, and the bisphosphonates. With these drugs the recently resorbed bone cavities continue to fill in with new bone, but without further resorption there is eventually no further bone formation. Bone mass increases for