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New words for old: lower urinary tract symptoms for "prostatism"

Avoids spurious suggestion of diagnostic accuracy

Extraordinary interest currently exists in the treatment of men over 45 who are referred with the label "prostatism." This has been generated partly by commercial interest and patients' increased awareness and expectations and partly by the advent of new treatments. Several new drugs to relieve bladder outflow obstruction, including α adrenergic blockers and 5-α reductase inhibitors, have been or are about to be licensed. Drug companies have expended huge efforts in increasing patients' and doctors' awareness of prostatic disease.

New techniques to destroy part or all of the prostate have been developed, including thermotherapy, thermal ablation, high intensity focused ultrasonography, transurethral needle ablation, and laser prostatectomy. Added to this are the techniques of intraurethral prostatic stenting and balloon dilatation of the prostate.[1]

Surveys have shown that urinary symptoms are very common in older men, with prevalences varying from 11% for straining up to 78% for nocturia.[2] Interestingly, British and American research has suggested that symptoms are also very common in elderly women.[3,4] Historically, we have used the terms "prostatism" and "symptoms of benign prostatic hyperplasia" to describe lower urinary tract symptoms in men. Yet because these symptoms are also common in women of similar age these terms become less sensible.

Other arguments exist against their use. Although the term prostatism implies a prostatic cause for symptoms, almost no evidence exists for such a cause. Most attempts to correlate either individual symptoms or groups of symptoms with objective measurements have failed to show any significant associations. (The exception has been the association between the symptoms of daytime frequency, urgency of micturition, and urge incontinence with the urodynamic finding of detrusor instability.[5]) The conclusion follows that no symptoms are specific to either benign prostatic hyperplasia or one of its complications—bladder outflow obstruction.

Benign prostatic hyperplasia is a precise histological term, yet many older men with lower urinary tract symptoms are described as suffering from the symptoms of benign prostatic hyperplasia or from clinical benign prostatic hyperplasia. The use of the specific histological term is confusing in everyday clinical practice.

Why is this seemingly pedantic discussion important? There are several reasons. Firstly, terms such as prostatism and clinical benign prostatic hyperplasia carry a spurious diagnostic authority, which may be translated into treatment without a proper diagnosis. Secondly, about one third of men with prostatism do not have bladder outflow obstruction secondary to prostatic enlargement.[6] Some 30 000 prostatectomies are performed each year in Britain,[7] and although increasingly urologists are defining bladder outflow obstruction objectively (usually by urine flow studies), many men with prostatism without bladder outflow obstruction are still being subjected to prostatectomy. The outcome of operations on such men is unsatisfactory.[8] Additionally, transurethral resection of the prostate is associated with low but important morbidity and mortality: some men may die unnecessarily.

If we reject the term prostatism and restrict the use of the term bladder outflow obstruction, is there an alternative? I believe that we should use the term "lower urinary tract symptoms." This describes patients' complaints without implying their cause. This is important as the symptoms are neither sex, age, nor disease specific. Hence, lower urinary tract symptoms could be used as a collective noun for any constellation of symptoms at any age, in either sex.

"Filling symptoms" would be a better term than "irritative symptoms" as irritative implies a pathological finding such as infection, stone, or tumour. The symptoms of frequency, urgency, and urge incontinence (traditionally grouped together as irritative symptoms) almost always indicate a functional abnormality rather than a structural fault or inflammatory process.

"Voiding symptoms" could replace the term "obstructive symptoms" (which include hesitancy, poor stream, straining, a feeling of incomplete emptying, and intermittency). We know that up to one third of men with low flow rates do not have bladder outflow obstruction but have detrusor underactivity as a cause of their reduced stream.[9] This seems part of aging and may be common to men and women.[10] Furthermore, two of the alleged obstructive symptoms—straining to micturate and intermittency of urinary stream—probably have no association with bladder outflow obstruction (J Reynard, personal communication).

Benign prostatic enlargement is a preferable term to benign prostatic hyperplasia as enlargement can be assessed, to some degree, by digital rectal examination and precisely defined by transrectal ultrasonography. Benign prostatic enlargement does not, however, imply the presence of bladder outflow obstruction, and many patients with enlargement do not have obstruction.

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