disease in producing severe disability, and it affects about 10% of the population over the age of 60.[1]

For all these reasons it should have provoked a wealth of research, and at long last it is doing so.[2,3] As a topic it is fraught with difficulties. Even the simple question, "What do we mean by osteoarthritis?" remains unanswered. Recently the American Rheumatism Association published a set of criteria for reporting osteoarthritis of the knee[4] and in a preliminary way of the hip and the hand.[5] Features said to have the most discriminatory power included age over 50, crepitus, bony enlargement, morning stiffness of less than 30 minutes, and osteophytosis—the only radiographic predictor.

The shortcomings of the study have been reviewed by McAlindon and Dieppe.[6] The controls were not matched for age or sex, were younger than the patients with osteoarthritis, and included many patients with rheumatoid arthritis. This may be the reason why age and osteophytosis appeared to be important discriminators and narrowing of the joint space was not.

There are good reasons for believing that osteophytes are an independent, age related variable.[7] In some groups, such as professional footballers, traction osteophytes may occur as a physiological response rather than a degenerative process.[8] The prevalence of features such as crepitus in the population is unknown. Features that seemed to be important in the recognition of osteoarthritis were largely subjective and have not been validated.

In epidemiological studies based on radiology the correlation between radiographic changes and symptoms is poor—apart from in the hip.[9] Moreover, the distinction between primary osteoarthritis and conditions predisposing to secondary osteoarthritis is by no means firm. There are many clinical variants, such as primary generalised osteoarthritis, erosive inflammatory osteoarthritis, diffuse idiopathic skeletal hyperostosis, and chondromalacia patellae.[10,11] Nor is it always clear which changes are primary and which are secondary. The fat lady who implores the orthopaedic surgeon for a hip operation assures him that inactivity due to arthritis explains her obesity. Some evidence suggests that the reverse is true, at least for arthritis in the knee.[12]

One tempting theory is the suggestion that the deposition of calcium pyrophosphate with chondrocalcinosis is a cause of osteoarthritis, but studies of hypermobile patients have suggested that it is secondary—at least in those who developed premature osteoarthritis.[13] These issues are important in considering the biology of osteoarthritis (a subject reviewed in detail recently by Hamerman),[14] and they have obvious therapeutic implications.

The clinician's first concern is to diagnose the condition. When swelling is bony, synovitis is minimal, and acute phase reactants are normal there is little problem. But often life is not so simple. Osteoarthritis with deposition of calcium pyrophosphate may mimic rheumatoid arthritis,[13] and so may acute generalised osteoarthritis in the elderly. Helpful clinical features that support the diagnosis of an inflammatory polyarthritis rather than osteoarthritis are disease affecting the ulnar styloid and synovitis of the radiohumeral, glenohumeral, and second, third, and fourth metatarsophalangeal joints. A raised erythrocyte sedimentation rate or plasma viscosity and raised concentrations of rheumatoid factors are useful pointers to rheumatoid arthritis. Radiological signs of erosion of the metacarpophalangeal and metatarsophalangeal joints and the ulnar styloid would also favour this diagnosis. The synovial fluid of osteoarthritis has characteristics of a transudate rather than an exudate, with a high viscosity and relatively few cells, those present being lymphocytes rather than polymorphs.

The syndrome of osteoarthritis is often the end result of a series of processes, and, as the American experience has shown, definition is not easy. Yet it is vital if appropriate treatment strategies are to be devised.

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Patient power in mental health

Working for users?

The difference between the provider of health services and the patient has been said to "resemble that between the hen and the pig in the preparation of eggs and bacon: the hen is involved but the pig is committed."[1] One way in which the patient's commitment is being acknowledged is "user participation," a jargon phrase that means that patients lead the way in planning new services. Patients want to participate actively in health care rather than be passive recipients,[2] and there is evidence that this will improve outcomes.[3] But "user participation" has also been advocated for ethical or even political reasons.[4] Here confusion begins.

Conflicting views of the patient's role in health care have been apparent for over a decade.[5] Some pressure groups envisage active "consumers" such as those described by Nader.[6] These groups talk about "empowerment," enhanced self respect derived from having a recognised role in services.[7] In contrast the consumers described in the first Griffiths report were patients who exercised their influence obliquely by looking at the services on offer, choosing and trying, and then complaining if necessary.[8] Emphasis on "patient choice" in *Working for Patients* still reflects this view.[9]

The concept of user participation is of particular importance in mental health, where the doctor-patient relationship may be an important part of therapy.[10] A broad range of groups with interests in mental health already exists: charities, support groups for patients and families, and pressure groups. Some accept a biological view of mental disorder, some do not, and some want no contact with formal psychiatry.[7] Yet across this diversity there is increasing emphasis on what the patient wants. For example, over the past year both MIND (the National Association for Mental Health) and the National Schizophrenia Fellowship have launched groups run by users for users.[11]

The consumer network of MIND lists over 70 "user groups."[12] In Chesterfield user groups are self run with financial sponsorship from social services.[13] In Nottingham groups participate in planning mental health services. They

also support patients' councils in psychiatric hospitals and social and recreational activities in day units.[14] In London a consortium of patients, mental health workers, and carers has influenced statutory planning[15] and hospital practice.[16]

Health professionals can learn from feedback, but patients' expressed satisfaction with services varies according to who asks the questions. Studies initiated by providers of services yield high ratings for satisfaction. Quantitative assessment of satisfaction with treatment is readily attainable,[17] even from severely disabled patients.[18] In the United States community mental health centres have been required by law since 1975 to evaluate patients' satisfaction.[19] Results, however, may be artificially improved by low response rates and biased assessment techniques.[20,21] Qualitative surveys conducted by patients' groups are more critical[16] but may also be biased: such groups set out to find and solve problems and they may overgeneralise without being representative of all patients.[22] Methodological factors also exaggerate this contrast between quantitative and qualitative research.[23]

How can psychiatry respond to the growth in these groups? The second Griffiths report does not mention user participation directly.[24] Neither does the Royal College of Psychiatry's paper on community care.[25] But the psychiatric profession must learn more about the scope and utility of user participation and encourage it: the persistence of patients' groups seems to underlie many of the most innovative programmes in mental health care. The King's Fund has called for increased collaboration between patients, planners, and managers in mental health care.[26] This could improve clinical outcome, encourage agreement about priorities in treatment, and resolve an embarrassing divergence between providers and users of services.

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The senses of the newborn

Tests for hearing and vision have improved

Shakespeare's description of the infant, "Mewling and puking in the nurse's arms,"[1] was echoed in the attitudes of doctors earlier this century. The newborn baby was thought to be either drowsy, asleep, or crying, and to experience the world as a "great, blooming, buzzing confusion."[2] But we have learnt over the past 30 years that the healthy newborn baby can discriminate between different sensations from the environment and respond selectively.[3] Within hours of birth the baby will look at the mother's face, and given the choice newborn babies prefer to look at a card showing the features of another human being rather than the same features jumbled up or the features condensed into a large black patch.[4]

The newborn baby spends only 11% of the time awake and alert in the first week of life, a proportion that rises to 21% in the fourth week.[5] This small fraction of wakefulness hindered the early development of methods of testing senses. When eliciting responses it is important to record the baby's state of arousal—between deep sleep at one extreme and crying at the other—and Prechtl's group first described five possible behavioural states.[6] Brazelton extended this work to include items of higher neurological function, including visual and auditory responses to a ball and rattle, and his neonatal behavioural assessment scale is a means of scoring interactive behaviour.[7]

Why do we need to test the senses of the newborn? We want to ensure that the baby is able to interact with the parents and with the environment and that there is no impairment to social, emotional, cognitive, and linguistic development. It is often difficult to prove that early intervention is effective in minimising handicap, but there is evidence—for instance, that deaf children fitted with hearing aids in the first six months of life have better speech than those fitted later.[8] And all parents and most therapists agree that they would like to know of any handicap as early as possible.

Finding reliable and practicable methods of testing hearing and vision in newborn babies has proved difficult. They show behavioural responses to sound, blinking and startling to a sudden clap and "stilling" to interesting noises, with alteration in their breathing pattern. Every mother recognises these responses, but they cannot be used to detect deaf babies reliably because of the spontaneous random movements babies make and possible bias on the part of the observers. The use of a simple rattle to produce head and eye turning has been described,[9] but the method has not found widespread acceptance. Behavioural responses may be recorded by devices incorporating microprocessors such as the auditory response cradle (which should eliminate observer bias). The sensitivity and specificity of this cradle have varied among trials,[10,11] and the sound stimulus has to be very loud (80-85 dB) to result in a behavioural response by the baby, so that moderate hearing losses are missed.

The electrophysiological response to sound may be detected by audiometry based on evoked responses in the brain stem, and this is considerably more sensitive. Simpler and more portable brain stem screeners have now been developed.[12] Most recently newborn babies' hearing has been tested by using otoacoustic emissions, a phenomenon first reported by Kemp in 1978.[13] A click stimulus delivered to a normal ear results in an "echo" sound generated by the cochlea, which can be detected by a miniature microphone. The method is quicker and less invasive than brain stem audiometry and can detect even mild hearing losses. Stevens and his colleagues