EDITORIALS

CONTRACTING IN RHEUMATOLOGY

The most fundamental change in the 'reformed' Health Service is the purchaser-provider split. Contracts are its written embodiment. They are untried, untested craft launched on a stormy sea, but despite the uncertainties produced by this, they are almost certainly here to stay irrespective of electoral change. It is essential that everyone realizes that contracts are locally negotiated. They are not and cannot be national agreements. They rely on a clinician in every locality being involved in their formulation. This is complex, time consuming and threatening, but it cannot be delegated.

Contracts attempt to match the desires and needs of the customers—a term used deliberately to encompass patients, patient organizations and general practitioners—with the services provided. One major benefit of this matching has been a greater need for us to find out what our customers want, and to discover from them and from our own audit process how well we meet their needs. The most common needs are for short waiting times for appointments, timely, relevant communication, and prompt, courteous consultations which impart appropriate information. They also want open access to physiotherapy services. In short, they want a quality service, and I have been heartened by the accent on pushing up quality rather than dragging down price which has typified our own local negotiations. They have, however, provided some rude shocks to those whose service provision fails to meet their customers’ needs, especially where this has been allied to reluctance either to accept criticism or introduce change.

The negative side of the contracting process is the enormous bureaucracy it is spawning. Regional and District authorities, shorn of much of their previous responsibility, are ideally placed to substitute new functions—and to skim off the cash to sustain them before it gets near service providers. This in turn generates matching bureaucracies in provider units and adds to the massive intrusion into professional time which the new structure has produced. In addition, a new Quality Industry has grown up, an unholy alliance of the great and the good in the health-care professions with bureaucrats, academics and external consultants, all thrusting their own view of quality down our throats while leaving us with less and less time to do more and more work.

The other danger of contracting, and one which is likely to become more important with time, is its potential to be used in an indiscriminate manner to drive down costs. The spread of rheumatology service costs being quoted at present suggests that widely different methods are being used in their calculation; certainly it would be impossible to produce any service let alone a quality one at some of the prices quoted. The risk is that price-obsessed managers with limited experience of the needs of our patients will attempt to use such figures to drive down costs irrespective of their derivation or the quality of the service they represent. Rheumatologists who lack experience in the contracting process are likely to be poorly placed to resist such pressures, and run the risk of being forced into the provision of a service which is unsatisfactory both to them and their patients. We must never allow price to replace quality as the contractual issue of major importance.

What can the BSR do to help with the contracting process? As there is no national contract it certainly cannot negotiate nationally on behalf of its members. What it can do is act, through its Clinical Affairs Committee, as a conduit for information. But this produces a dilemma. Information must come from individual members, but there will be pressure from their managers not to divulge information because of 'commercial confidentiality'. This phrase is being widely used at present to try to constrain medical staff. In the long term it is vital that the interests of the doctor–patient relationship take precedence over the pseudo-commercial needs of management. We must share information if 'divide and rule' is not to dominate our lives. Our Society can also give guidance about standards which we can use in our local contractual negotiations. Examples of this are the papers on audit, produced jointly with the Royal College of Physicians, and on outpatient services which have already been distributed to members. It can also help by advising purchasing authorities regarding the evaluation of rheumatological health needs in the population, so that purchasing decisions are informed by population need rather than drama and shroud-shaking. The Clinical Affairs Committee is already discussing health needs assessment with purchasing colleagues, and will produce a paper on this subject shortly.

However, the essence of the contracting process is that it is local and local consultants must be involved in all stages of it.* Involvement in some aspects, such as delineation of the service and the clinical details of its provision, appears obvious. There must be equal involvement, however, in more 'managerial' aspects of the contract especially coding, acquisition of accurate data and statistics and, above all, the way in which the contract is priced. There must also be clinical involvement in setting quality and audit standards, and those rheumatologists who do get involved may well find that the explicit quality targets of new contracts will provide a useful lever to institute service improvements which they have long desired.

*Guidance on involvement in contracting is found in the NHS Management Executive document EL(91)84.
The contracting process is one of the many innovations in the new Health Service environment which are causing rheumatologists disquiet and discomfort at present. It does contain risks to our professional services, but I believe that enthusiastic participation in it does have the potential to improve the care of patients with rheumatic diseases.

**DO ‘PAIN CENTRES’ EXIST?**

Pain is one of the most important symptoms in rheumatology and yet little is known about the central mechanisms involved in the human pain experience. Pain is a strictly subjective experience and can only be quantitated as such. However, the nervous system is well equipped to respond to pain in a very precise manner as shown by the consistent results obtained using both simple and complex psychometric measures of pain in different pain syndromes [1, 2]. For example, visual analogue scores of pain correlate well with objective measures of inflammation in patients with rheumatoid arthritis under carefully controlled conditions [3]. Pain report also correlates well with intensity of C-fibre stimulation in a population of normal volunteers [4]. However, many different central phenomena such as mood change or placebo response can interfere with such simple relationships. Experiments in animals have provided some insight into how some of the mechanisms of change in central processing might operate, but it is difficult to extrapolate from animal studies to the human experience.

Pain is an experience that comprises sensory-discriminative and motivational-affective components [5]. The term discriminative refers to the ability to identify the type of stimulus as well as its temporal and spatial components. The motivational-affective components relate to behavioural consequences of such stimuli that may vary considerably in their expression.

The ‘classical’ pain theory that has been established over the last 20 years is that there are two parallel pain processing systems operating at a cortical and subcortical level [6]; the so-called ‘lateral’ and ‘medial’ pain systems. The ‘lateral pain system’ comprises the lateral projections of the spinothalamic tract. This has a crossed monosynaptic projection to the lateral thalamic nuclei and subsequently to the primary somatosensory cortex. It has been identified as the main system projecting via the brain-stem reticular formation to the anterior cin-
gulate cortex and the prefrontal cortex [7]. The ‘medial pain system’ has been implicated as mainly involved in response to chronic pain and in particular the emo-
tional components [6].

One of the main objections to this neat compartmentalization is that it is difficult to elicit pain by stimul-
ating anywhere in the primary somatosensory cortex in awake patients undergoing craniotomy [8]. Indeed, the difficulty in eliciting pain from stimulation anywhere on the superficial cortex makes it difficult to accept the idea of cortical pain centres and has led some to question whether the cortex is involved in the experience of pain at all.

Positron emission tomography (PET) studies have been used to identify the main areas in the normal human brain that respond to the suffering components of pain. Using cerebral blood flow (CBF) as a measure of neuronal activity, the contralateral thalamus, lentiform nuclei and anterior cingulate cortex contralateral to the side of stimulation were found to respond specifically to thermal pain [9, 10]. The anterior cingulate cortex is part of the paralimbic cortex, lying on the medial surface of the cerebral hemispheres just above the corpus callosum. It has been implicated in the attribution of emotional significance to sensory stimulation in monkeys [11]. In this context it is interesting that deafferentation of this area of cortex in patients with intractable pain produces not an ablation of the chronic pain, but an indifference to the pain [12]. The anterior cingulate cortex also has important connections with the limbic system and the periaqueductal grey in the brain-stem. The latter is one of the areas mediating opiate and non-opiate descending inhibition of pain [13]. The cortical projections of the medial pain system are therefore likely to be more important to acute pain processing than had previously been realized. The lack of response in the primary somatosensory cortex suggests that the importance of the lateral pain system has been overemphasized.

Primates post-mortem autoradiography has demonstrated high concentrations of opioid receptors in the medial pain system and some of its connections, whereas at least at a subcortical level lower concentrations are found in the constituents of the lateral pain system. More recently a focal deficit of in vivo opioid receptor binding has been demonstrated in the primary motor-sensory cortex of normal human volunteers using [11C] diprenorphine and PET [14]. This would suggest that if the lateral pain system is involved in pain response, it is poorly equipped to participate in opiate induced analgesia. Acute pain has been shown to be both decreased by morphine and increased by the opiate antagonist naloxone [15]; this is more compatible with an effect via the medial pain system. Preliminary PET studies have demonstrated that the main acute