come are obtained). This is shown by two controlled studies, which found that self reported diagnoses of connective tissue disease and symptoms were more common among women with breast implants but that medical evaluation failed to confirm the diagnosis of connective tissue disease or any difference in objective findings between women with and without breast implants.[6 10]

What should doctors advise women who have silicone breast implants? If they are well and have not had local problems such as hardening or rupture of the implant we recommend that they do nothing. They should be reassured by the epidemiological studies, all of which show no association.[5-9] Patients with connective tissue diseases or rheumatic complaints and silicone breast implants need to be treated on a case by case basis.

Whether removing the silicone breast implants alters the course of a connective tissue disease is unknown. Among 12 reported cases, some improvement was described in seven.[1] Four of nine patients with scleroderma had cutaneous improvement (one of them also had visceral improvement). In two cases of systemic lupus erythematosus both clinical and serological manifestations improved. In one case of "human adjuvant disease" some improvement was noted. No firm conclusion can be drawn from the reports.

Whether silicone breast implants are associated with connective tissue diseases remains controversial. Despite the increased number of cases reported in the literature no association has been convincingly established.

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Purchasing clinically effective care

National directives cannot be fulfilled without local collaboration

Research findings are often poorly translated into clinical practice. One example is the management of acute myocardial infarction, where the evidence of the effectiveness of aspirin and early thrombolysis is overwhelming.[1 2] Despite this the proportion of patients receiving the treatment may be low.[3 4] Ensuring that patients receive the best possible care should be important for all doctors.

Should purchasers care as well? The NHS Executive thinks so and believes that the issue should be addressed through contracting. Last December all fundholding general practitioners, trusts, and health authorities received a letter from the executive urging them to take clinical effectiveness and clinical guidelines into account in contracting.[5] Seven guidelines were attached for consideration, with the hope that purchasers would include at least one of them in their contracts.

The NHS Executive clearly believes that clinical effectiveness should form part of the NHS's medium term objectives. Planning guidance already issued for 1995-6 has included the objective that the NHS should "invest an increasing proportion of resources in interventions known to be effective and where outcomes can be systematically monitored, and [that it should] reduce investment in interventions shown to be less effective."[6] Purchasing authorities will be expected to increase investment in at least two interventions known to be effective, to reduce investment in at least two interventions that evidence has identified as likely to be ineffective, and to increase the use of clinical outcomes and audits in contracts.

Now a further letter from the executive, issued last week, shows some softening of approach.[7] The complexity of the task is acknowledged, as is the length of time needed to adapt suitable evidence based clinical guidelines for local use. This shift of emphasis is welcome because evidence of the effectiveness of clinical guidelines themselves shows that a top down approach is less likely to change behaviour than the development of guidelines by those who are to use them.[8] Another new approach is the suggested involvement of primary care; family health services authorities are asked to work with medical audit advisory groups, general practice postgraduate tutors, and local practitioners in the development of local documents. Great benefits could accrue from doctors in primary and secondary care working together on clinical policy; it would be wrong to restrict all initiatives regarding clinical effectiveness to hospital providers. Lastly, the letter suggests that patients should be involved in developing guidelines.

Whether any of these initiatives will change doctors' practice—for example, increasing the chances of patients with an acute myocardial infarction receiving aspirin and thrombolysis—is unknown. Haines and Jones have advocated an approach to implementing research findings in clinical practice that incorporates work with opinion leaders, purchasers, and professional organisations; programmes of education and clinical audit; and the use of "patient specific reminders" to support clinical decision making.[9] Most of these approaches have been shown to affect clinical practice, although mostly outside Britain. As systematic reviews of research evidence begin to emerge from the Cochrane Collaboration[10] and the NHS Centre for Reviews and Dissemination we need to establish which methods of implementation work best in the NHS and to

create an infrastructure which could enable the new material to be put to best use.

No one doubts the critical importance of clinical effectiveness, and the NHS Executive is right to make it the concern of both purchasers and providers, but the use of contracting to change clinical practice will need evaluation (just as any other intervention requires evaluation). Stipulating that purchasing authorities should divert investment towards effective interventions and away from ineffective ones has a mechanistic feel to it. Purchasers need to have a more interactive role than this: they need to establish dialogue with local hospital doctors, general practitioners, and patients. In addition, hospital doctors need to talk to each other about policy and practice, and purchasers should insist that they do so.

At this stage the role of the contracting mechanism should perhaps be to tie providers to this dialogue; to ensure that clinicians address issues of clinical policy and practice with their colleagues (including the local adaptation of evidence based clinical guidelines); and to enable local users of health services to have an informed voice. That would give all parties sufficient freedom for local collaboration, while ensuring the commitment of providers.

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Evening primrose oil

Currently used in many conditions with little justification

Oil extracted from seeds of the evening primrose (*Oenothera biennis*) contains linoleic acid, γ linolenic acid, and vitamin E. γ Linolenic acid is a precursor of prostaglandin E and several other active substances and is said to be the constituent of the oil responsible for its therapeutic effects. Disorders for which evening primrose oil has been tested in controlled clinical trials include atopic dermatitis, rheumatoid arthritis, diabetic neuropathy, multiple sclerosis, various cancers, Raynaud's phenomenon, ulcerative colitis, pre-eclampsia, the premenstrual syndrome, menopausal flushing, breast cysts, mastalgia, Sjögren's syndrome, schizophrenia, and hyperactivity.[1] What are the results of these clinical trials?

Many of the studies have been crossover trials, which is a pity for two reasons. Firstly, crossover trials are really suitable only for assessing drugs whose effects fade rapidly after treatment has been stopped. Any persistent effects will disappear provided there is a "washout" period before the crossover. Secondly, if the explanation given to patients before their informed consent is obtained includes the timing of the crossover their expectations may become a major source of bias. The treatment, the natural course of the disease, and placebo effects will induce changes that the patient may or may not have expected. For example, patients who receive active treatment in the first period and who notice improvements will have low expectations for the next period; and patients who notice no improvement in the first period will assume that they were taking placebo. These expectations would result in a bias, increasing the measured difference. Evening primrose oil is claimed to have effects that are both sustained and subjective, and so parallel trials should be used to assess its effects.

Turning to the published work, for atopic dermatitis Wright and Burton reported positive effects from a double blind crossover trial of evening primrose oil in 99 patients (60 adults and 39 children).[2] Bamford *et al* found negative effects in a double blind crossover trial in 123 patients.[3] A meta-analysis of nine trials (five crossover trials) including the trial of Wright and Burton but excluding that of Bamford *et al* reported positive results, and, more recently, negative results were reported from a parallel trial in 123 patients.[4,5]

For rheumatoid arthritis Joe and Hart discussed three randomised trials with parallel groups and Leventhal *et al* reported another one.[6,7] These trials were small—the largest group was of 19 patients, and the results were mixed. The investigators concluded that further trials were warranted.

For the premenstrual syndrome four trials (three crossover trials) have reported positive results,[8] but more recently Khoo *et al* found no differences between evening primrose oil and placebo in a crossover trial in 38 women.[9] The effects of evening primrose oil on mastalgia, one of the symptoms of the premenstrual syndrome, have been investigated in three small randomised trials, with favourable results.[10]

After a preliminary trial in 22 diabetic patients reported favourable results the Gamma Linolenic Acid Multicenter Trial Group reported positive effects on many neurological and neurophysiological end points in a well performed parallel double blind trial in 111 patients with mild diabetic neuropathy.[11] These encouraging findings indicate that further investigations of evening primrose oil in diabetic neuropathy should be given priority.

Evening primrose oil seems to be safe. Its reported side effects include nausea, softening of the stools, and headache. One recent comment warned of a potential risk of inflammation, thrombosis, and immunosuppression due to slow accumulation of tissue arachidonate after prolonged use of γ linolenic acid for more than one year.[12]

The optimal dose and duration of treatment with evening primrose oil seem not to be known. Trials that