proteins and human placental lactogen disappear within a few days of delivery. Human chorionic gonadotrophin has a prolonged second half life of nine to 37 hours and is detectable in the blood up to two weeks post partum. Several enzymes are cleared slowly, while pregnancy zone proteins such as α fetoprotein and oxytocinase persist in the plasma for several weeks.

Since, however, postpartum mental disorders affect only some parturient women while all of them experience these profound biochemical changes, it seems more likely that an abnormal reaction to one or other of these changes rather than the changes themselves is responsible for the mental disturbances. No convincing correlation of antepartum hormone concentrations with postpartum mental disturbance has been recognised.[12]

The finding that plasma concentrations of β endorphin are raised during pregnancy and labour and fall rapidly after delivery raised the possibility that withdrawal of these endogenous opioids might be a possible cause of "maternity blues."[13] Newnham and his colleagues have pursued this possibility further and have found a negative correlation between the woman's estimate of her pain in labour and the β endorphin concentrations post partum, suggesting an analgesic role for β endorphins in labour.[14] They also found a positive correlation between β endorphin concentrations at delivery and the woman's attitude to her pregnancy at 36 weeks and a negative correlation between the postpartum "blues score" and the β endorphin concentration at 36 weeks. On the other hand, the blues score did not correlate either with the β endorphin concentration at delivery or 24 hours post partum, nor with its rate of fall in the first 24 hours. These findings do not confirm the withdrawal hypothesis but neither do they refute it, especially as alterations in central opiate activity might well be more relevant than changes in peripheral blood. They found, moreover, no consistent social, psychological, or obstetric correlation with the occurrence of maternity blues, in contrast with what has been claimed by some authors.[15-18]

Another study offered support to altered target organ function rather than to change in humoral factors. Metz *et al* found that platelet α_2 adrenoceptor capacity at seven to 10 days post partum was higher in women with maternity blues than in those without.[19] Platelet α_2 adrenoceptor capacity is affected by circulating concentrations of oestrogen and progesterone and by the falls in these concentrations after childbirth; brain α_2 adrenoceptor capacity is enhanced in depressive illness and probably parallels that of platelets.[20,21] Thus a failure of α_2 adrenoceptor capacity to fall after delivery from the high concentrations of pregnancy to the lower concentrations of the normal non-pregnant state might be a factor in the causation of maternity blues, and its persistence might lead to the more severe later form of postnatal depression.

Lindström *et al* found that the cerebrospinal fluid of psychotic postpartum women had higher concentrations of opioid receptor active components than did normal lactating women; in four of these psychotic patients the "fraction II" activity was very high and on electrophoresis and high performance liquid chromatography the material migrated as bovine β casomorphin, postulated to have been derived from casein formed during lactation.[22] Receptor active material with the same characteristics was also found in the plasma of four psychotic patients but not in a pooled sample from four healthy lactating women. The authors cite other evidence that neuroactive peptides of extraneuronal origin may cross the blood-brain barrier to cause psychiatric disturbance and suggest that their findings may stimulate the search for peptides derived from outside the central nervous system in the plasma of schizophrenic patients.

Despite the attractiveness of the hypotheses linking hormonal changes with puerperal mental disorders, no useful conclusions—whether prognostic, prophylactic, or therapeutic—have emerged from the older studies. The newer developments, such as the studies of opioid peptide and α_2 adrenoceptor briefly discussed above, open up new perspectives for research into these disorders. We can only hope that these may lead to better understanding and so to a rational basis for preventive and curative treatment.

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Time for a new name for "frozen shoulder"

1984 was the 50th anniversary of the introduction by Codman of the term "frozen shoulder."[1] Perhaps, as Roy and colleagues have suggested, "the term frozen shoulder, which has for too long encouraged many doctors to do as little as possible about this common and distressing condition, should be abolished."[2] Their view is shared by Neviaser, who has argued that "the misnomer 'frozen shoulder' should be deleted from the medical literature."[3]

Why is "frozen shoulder" a misnomer? Firstly, "frozen shoulder" is not cold but hot. On histological examination the tissues are infiltrated with inflammatory cells and (as all medical students know) inflammation means calor, as well as dolor and rubor, but never fridgedoor.[4,5] Moreover, radioisotope scans show gleaming hot spots at the shoulder.[6]

Surgically the rotator cuff is far from solid, as if just taken from the freezer, but extremely tender and friable—more, cooked to perfection.[4]

So what should we call this syndrome? "Cooked shoulder?" Certainly not. No, we need a term with a certain panache, a certain style. Historically the first term was "periarthrite scapulohumerale," coined by Duplay in 1896, but, though it seems that the lesion may be peri, it is not an arthritis.[7] Duplay thought the initial lesion was in the subacromial bursa, which is how subacromial bursitis crept in on the act, but now we know this to be secondary and not primary. These concepts should be tossed in the waste bin along with frozen, for pathologically they are misleading and clinically they are unhelpful.

In 1945 Neviaser adopted the term "adhesive capsulitis."[8] He operated on 10 patients with stiff, painful shoulders, but he found no adhesions. He meant adhesive as in Elastoplast, for he found that the synovium peeled off the head "like adhesive plaster from skin." Ever since, surgeons have been looking for the elusive adhesions and not finding them, even at arthroscopy.[9] Some surgeons are so confused that they even state that they tear adhesions during closed manipulations. What they actually tear, as was elegantly shown at operation by De Palma[10] and has been confirmed by Macnab,[11] is the rotator cuff and capsule itself. Adhesive is so confusing it ought to be relegated to the history books. Should we drop the adhesive and just say "capsulitis?" Certainly the capsule is inflamed, but the adhesive has stuck like glue. To some they are inseparable, and what is left?

Why not start afresh? What do we know of this clinical syndrome? It is a common condition accounting for 1% of all orthopaedic referrals, and that means 11 000 new cases a year in England and Wales.[12] It may be up to four times as common in general practice.[13] Most frequent in the age group 50 to 70, it is more common in women. The clinical pattern goes through three phases—pain, stiffness, and recovery—and, contrary to popular belief, the recovery may not be complete: 42% of Reeves's patients had a deficit in their range of motion and 6% a functional deficit at 10 years.[14]

Treatment of "frozen shoulder" can be only symptomatic until its cause is unravelled, and Lee *et al* have listed 14 different forms of treatment which have been used—from massage to radiotherapy.[15] The problems in sifting through these treatments are, firstly, confusion in terminology[16] and, secondly, that very few studies have used controls—which in a naturally resolving condition diminishes the worth of their authors' conclusions. Perhaps the top two contenders for effective treatment are local injection of steroids and manipulation under anaesthesia. Of the controlled studies Lee *et al* found no advantage in steroid injections plus physiotherapy against heat and physiotherapy[15]; Berry *et al* showed no difference between steroid injection, acupuncture, and placebo[17]; Bulgen *et al* showed there was no long term advantage of steroid injection over mobilisation, ice, or no treatment, but that steroid injection might reduce pain and increase movement in the early stages[18]; and, finally, Richardson showed no difference in relief of pain or pain on resisted movement between injection of steroids and placebo, but the range of movement was increased in the steroid group.[19] A recent editorial in the *Lancet* concluded: "The message is not that local steroid injections have no part to play—in some patients the effects may be startling—but that unprecise diagnostic labels hamper attempts at prognosis."[16]

Charnley showed how manipulation under anaesthetic could be used safely,[20] suggesting that surgeons' reservations against early manipulation were "deeply coloured by the classical management of bacterial inflammation as taught by Hilton in the famous monograph *Rest and Pain* published in 1863."[20] Kessel, Bayley, and Young did a carefully controlled trial which showed the worth of manipulation plus systemic steroids and stated "the catchy term 'frozen shoulder' trips readily off the tongue and this may be one of the reasons why this condition is considerably overdiagnosed."[21]

The painful shoulder remains an enigma, since we are still unsure of its cause and hampered by confusing terminology. It remains as mysterious (or more so) than the painful knee before the advent of the arthroscope, when doctors, if they were honest, put down a diagnosis of "IDK." To the patient this was translated as "internal derangement of the knee" but to the doctor, even after a thorough history and examination, it meant "I don't know." This humble statement implied a lack of knowledge that needed to be overcome before rational treatment could be applied. Arthrography and arthroscopy were used to investigate "IDK," and now surgeons can tell precisely what is going on in the knee and plan surgery accordingly.

Although arthrography shows a small joint volume[22] and arthroscopy has shed little further light[9] on the painful shoulder, perhaps a similar humble term should be applied to replace frozen shoulder. Until the cause or causes of the stiff, painful shoulder are unravelled I would like to propose an interim term "HGAC." This has the benefit of brevity (indeed it is a four letter word) and rhythm, for gee rhymes with cee, and it implies no false knowledge of aetiology or histology. To the patient it can mean "humeroglenoid acromioclavicular syndrome," and to the doctor "Haven't Got A Clue"—which should stimulate us to overcome our lack of knowledge and seek the cause or causes of this common, disabling, protracted, and painful condition so that we may one day offer our patients an effective method of treatment.

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