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Comparison of the natural history of untreated acute gouty arthritis *vs* acute gouty arthritis treated with non-steroidal-anti-inflammatory drugs

Bellamy *et al.*'s (1987) recent paper, concerning the natural history of untreated gouty arthritis confirmed the clinical impression that the pattern of resolution of the acute attack is markedly different from the pattern of treated gouty arthritis. Their conclusion, that controlled studies may not be necessary to evaluate the efficacy of newer anti-inflammatory agents stimulated us to review briefly the literature regarding the resolution of pain in treated acute gouty arthritis to test this proposition. We considered trials where the available data was suitable for re-analysis, to determine the general trend of response.

To achieve some degree of uniformity between the different trials which used diverse methods for the quantification of pain in small numbers of patients, under variable study conditions, we re-analysed the available data, expressing pain relief as a percentage change from the baseline measurement (Figure 1). As a result, all the patient groups had a pain score of 100% at the time of entry to the trial. As the author's original raw data were not available to us, we have not performed formal statistical analysis on the data presented in the following papers (Ahern *et al.*, 1987; Bellamy *et al.*, 1987; Bluestone, 1982; Marcolongo *et al.*, 1980; Murphy, 1979; Ruotsi & Vainio, 1978; Widmark, 1982), as this would have overinterpreted the available data.

From the data presented, it appears that the rate of resolution of pain in acute gout is strikingly modified by therapy. Ahern *et al.* (1987) were the first to report a controlled clinical study of colchicine in acute gouty arthritis. It can be seen that the pain relief experienced by their placebo group was similar to the untreated group of Bellamy *et al.* (1987), where the improvement in pain was slow and incomplete, even after 5 days of non-intervention. It should be noted that

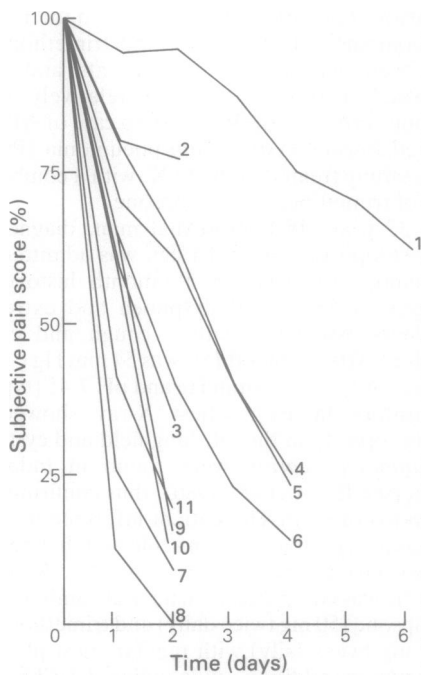

Figure 1 Graph of change in pain (%) *vs* time (days) (pain at entry to study was by definition, 100%)

Key to figure

1. Bellamy *et al.* (1987); untreated
2. Ahern *et al.* (1987); placebo group
3. Ahern *et al.* (1987); colchicine
4. Bluestone (1982); low dose piroxicam
5. Bluestone (1982); high dose piroxicam
6. Murphy (1979); piroxicam
7. Marcolongo *et al.* (1980); indoprofen infusion
8. Marcolongo *et al.* (1980); indoprofen i.v. bolus
9. Ruotsi & Vainio (1978); proquazone
10. Ruotsi & Vainio (1978); indomethacin
11. Widmark (1982); piroxicam

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Ahern *et al.*'s (1987) placebo group showed the expected response to placebo, with some degree of improvement. Despite this small improvement the response of the palcebo group is none-theless of considerably smaller magnitude than the response of those patients treated with active drugs after 24 h of treatment.

It can also be seen that the response in Bluestone's (1982) low (20 mg day^{-1}) and high dose (40 mg day^{-1}) groups were little different from the response seen in Ahern *et al.*'s (1987) placebo group. This may reflect the prolonged plasma $t_{½}$ life of this drug, since effective plasma concentrations may not have been achieved at this early stage in the low dose group. Also of interest is the variability of relief of pain in the trials of active drugs in the initial treatment period.

Also of interest is the variability of relief of pain in the trials of active drugs in the first 24 h of therapy. In particular, the results of Bluestone (1982), Murphy (1979), Ahern *et al.* (1987) and Ruotsi & Vainio (1978) show at best only a 12% improvement over placebo, and a 27% improvement over the untreated group of Bellamy *et al.* (1987). This may reflect factors of patient selection, but may also poorly reflect on the capacity of treatment (in some instances) to achieve the desired result; namely the swift relief of pain.

The other studies with active drugs (Marcolongo *et al.* (1980), and Widmark (1982)) show pronounced differences when compared with Bellamy *et al.*'s (1987) untreated group which are greatest after 24 h of therapy. This confirms the latter authors' conclusions regarding the potential value of using their observations on untreated gouty arthritis patients as a control group for anti-inflammatory drug trials in this disease. Bellamy *et al.*'s (1987) study may be useful as an external control group against which the rapidity of pain relief and time to resolution of the acute attack in further trials of non-steroidal anti-inflammatory drugs in this disease may be assessed.

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Propafenone in the treatment of chronic ventricular arrhythmias in a pregnant patient

Propafenone is widely used in the treatment of chronic ventricular arrhythmias (Harron & Brodgen, 1987) but, so far, no data are available on the use of this drug during pregnancy (Mitani *et al.*, 1987). We assessed the maternal-foetal transfer of propafenone in a pregnant patient

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