duration of each procedure was similar: Bier's block 21·1 (7·9) minutes; local infiltration 20·3 (12·5) minutes, but after Bier's block patients required radiography before the tourniquet was released (20·0 (9·2) minutes) and were observed for a further 20 minutes. For Bier's block staff were committed for twice as long (41 (11) minutes), and patients waited longer before the procedure began (89 (52) minutes) than did those receiving local infiltration (67 (37) minutes).

No patient suffered significant complications or side effects. Inadequacy of reduction of the fracture was rare and not related to the technique.

Relative merits of Bier's block and local infiltration. (Values are scores)

	Bier's block	Local infiltration
Complications	4	1
Effectiveness	3	2
Simplicity	2	3
Speed	1	4

Comment

For patients with fresh Colles' fracture local anaesthetic infiltration was more popular among accident service staff (table), giving satisfactory anaesthesia, being simpler and quicker to perform, and avoiding risks of a large intravenous dose of local anaesthetic agent reaching the general circulation. No documented reports exist of infection after its use. Unfortunately, objective comparison of the efficacy of the technique with the widely used and tested Bier's block has shown it to be inferior.

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Do β blockers cause arthropathy? A case control study

Savola described 18 patients with arthritis that he attributed to treatment with β blocking drugs, particularly metoprolol, and he suggested that arthritis is a common adverse reaction to treatment with β blockers.[1] We investigated this hypothesis in a case-control study.

Patients, methods, and results

All patients with both hypertension and arthropathy affecting peripheral joints who had attended the Sheffield Hypertension Clinic during the three years 1980-2 were identified by a search of the formal problem lists used routinely in the clinic. When a specific diagnosis (for example, osteoarthritis) was stated on the problem list this was accepted to avoid importing bias into the study. When no specific diagnosis was stated the clinical and laboratory findings were reviewed. Patients with acute gouty arthritis and lupus induced by hydralazine were excluded because these conditions might have biased the choice of antihypertensive drugs. Each patient in the study was matched for age (same decade) and sex with two control patients who had hypertension but no arthritis and had attended the clinic during the same year. Patients aged over 80 could not be matched and were excluded. The use of β blockers by patients with arthritis and controls was compared. The approximate relative risk and 95% confidence limits were calculated as described by Armitage.[2]

We identified 127 patients with arthritis (80 women, 47 men; mean age 61·4 years). The 254 controls were well matched for age and sex. There was no association between arthritis of all types and the use of β blockers (approximate relative risk 1·14, 95% confidence limit 0·74-1·77) (table). For atenolol, the β blocker used most commonly, the approximate relative risk was 1·00 (95% confidence limit 0·65-1·54). Osteoarthritis, rheumatoid arthritis, and other specific arthropathies were not associated with use of a β blocker (table).

In 42 patients the cause of the arthritis had not been determined. These patients were no more likely than their controls to be taking a β blocker (69% *v* 64%). Seventeen patients had been investigated in depth in the clinic, but arthritis had remained unclassified despite this. These patients were no more likely than their controls to be taking a β blocker (approximate relative risk 1·13, 95% confidence limit 0·33-3·91). Arthritis had developed during treatment with a β blocker in only five of this group of patients.

Comment

In this study the use of β blockers was not associated with arthropathies in general or with unclassified arthritis. The 17 patients with arthritis that had remained unclassified despite investigation corresponded most closely to the patients described by Savola,[1] but they were no more likely than control patients to be taking a β blocker. Our study had insufficient power to exclude completely an association between use of β blockers and arthropathy (as shown by the upper 95% confidence limits in the table), but it does not lend support to the suggestion that arthritis is a common adverse reaction to β blockers. The observations differ in two important respects from those of Savola.[1] In his series of cases metoprolol was the drug implicated most often. Few patients in our study population were taking metoprolol, and it remains possible that arthritis is related specifically to this β blocker. It is, however, most unlikely that there is an association between atenolol, or β blockers in general, and arthropathy. The second important difference between the two sets of observations is that Savola's series was uncontrolled. It is often difficult to be certain of cause and effect when assessing possible adverse reactions, and the data reported by Savola did not remove all doubt, as others have pointed out.[3]

We conclude that arthritis that cannot be classified is not uncommon in hypertensive patients, but there is no evidence that β blockers are responsible for it.

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Use of β blockers by patients with arthritis and controls

	No of patients		No (%) taking a β blocker*		Approximate relative risk	95% confidence limits
	Cases	Controls	Cases	Controls		
All arthritis	127	254	80 (63)	152 (60)	1·14	0·74-1·77
Osteoarthritis	58	116	32 (55)	69 (59)	0·84	0·44-1·59
Rheumatoid arthritis	19	38	13 (68)	21 (55)	1·75	0·55-5·58
Other arthropathies†	8	16	6 (75)	8 (50)		
Unclassified arthritis	42	84	29 (69)	54 (64)	1·24	0·56-2·74

*β Blockers taken were (cases/controls): atenolol 55/110, oxprenolol 11/16, propranolol 5/14, metoprolol 2/6, acebutolol 2/3, sotolol 2/0, timolol 1/1, pindolol 1/1, labetalol 1/1.
†Ankylosing spondylitis (five patients), Still's disease (one), polymyositis with arthralgia (one), and polymyalgia rheumatica (one).