hepatitis or reactivation of the latent hepatitis B virus infection may have been implicated as the possible cause of the hepatic episode.

As expected, during follow up HBsAg persisted in the serum of many patients without the IgM antibody, whereas among patients with the IgM marker the antigen cleared in almost all. Clinically, therefore, a negative IgM anti-HBc test result predicts the carriage of HBsAg.

Our data show that testing for IgM anti-HBc is important in patients developing an apparent hepatitis B of unrelenting course: a similar clinical entity is mimicked by progressive non-B hepatitis in previously unrecognised carriers of HBsAg, and in this study accounted for most of the cases of HBsAg hepatitis destined to become chronic. A negative IgM anti-HBc test result should prompt search for a cause of the liver damage other than hepatitis B virus.

The magnitude of the problem of false hepatitis B is likely to be proportional to the prevalence of carriage of HBsAg in the population. In our study, however, the relation was not numerical, as in each series examined the proportion of patients negative for IgM anti-HBc was higher than the local rate of carriage.

Though a minority of the IgM anti-HBc negative patients were drug addicts or patients receiving dialysis and therefore heavily exposed to blood borne hepatitis viruses, an epidemiological factor was not obvious in the others. The implication might be that the HBsAg carrier state determines an increased biological susceptibility to hepatitis by multiple viral and possibly non-viral factors. This was shown for delta, a defective virus that is rescued and activated only by HBs antigenaemia.¹⁵

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SHORT REPORTS

Arthropathy induced by beta blockade

Few reports on arthropathy induced by beta blockade have been published. Pain and swelling of both knees developed in a man who took practolol for nearly three years after a myocardial infarction, subsided when he stopped the drug, and recurred when he took it again.¹ Polyarthritides has been described in association with propranolol and oxprenolol²; the same author described a similar case attributable to propranolol.

Paradoxically, propranolol exerts a favourable effect on arthritis.³ The mechanism of action remains to be established. The effect probably occurs as a result of the membrane stabilising or anaesthetic properties of the drug; beta receptor blockade does not seem to be a factor.⁴

In 1980 I began to look for possible cases of arthropathy induced by beta blockade after a patient's shoulder complaint subsided soon after metoprolol was stopped.

Patients, methods, and results

Patients were considered to have suffered from arthropathy induced by beta blockade if their joint symptoms developed during administration of a beta blocker and subsided only after the drug was stopped (or after a change to a different beta blocker). All the patients were seen at this hospital or my private surgery and came from a region of about 25 000 inhabitants.

Eighteen patients (mean age 64 years, range 45-74 years) were seen

with arthropathy induced by beta blockade; six had been taking beta blockers for more than five years and seven for more than two years. The primary indication was hypertension or coronary heart disease or both. In every case an attempt was made to exclude other causes of joint symptoms by radiology and serological tests for rheumatoid disease.

Fifteen patients had symptoms affecting the shoulder joint. These consisted mainly of discomfort on moving the arms and limitation of movement due to pain when the patient tried to raise the arm above the horizontal. Some patients complained of pain and stiffness at rest. None had joint effusion. Radiological examination generally disclosed slight thickening of the soft tissues surrounding the shoulder joint. Nine of the 15 had symptoms in other joints as well. Apart from the shoulder, the knee was most commonly affected (six patients). Five patients had symptoms of polyarthritides. Two patients complained of swelling and stiffness of the small finger joints.

The primary beta blocker used was metoprolol (table). Those patients who developed joint symptoms with propranolol were given metoprolol as the first alternative. All reported exacerbation of the joint symptoms. Associated symptoms such as dry mouth and eye complaints were common as well (table). Despite attempts to substitute other beta blockers for metoprolol and propranolol the same pattern of adverse effects tended to recur, so that beta blockade eventually had to be stopped in 12 of the 18 patients. All the joint symptoms (and the associated complaints) resolved promptly in the patients.

Comment

Joint disturbance must be considered to be a common adverse effect associated with beta blockade. Other adverse effects, notably eye symptoms, dry mouth, and Raynaud's phenomenon, were also common, which points to some common factor, most probably dehydration of mucous membranes and synovia induced by the drug.

Adverse effects caused by beta blockers, and number of patients per beta blocker and per adverse effect

Beta blocker	Average daily dose (mg)	No of patients with:							No of patients per beta blocker
		Shoulder joint affected	Polyarthritic symptoms	Blocked nose	Impaired peripheral circulation	Eye symptoms	Bronchial obstruction	Dry mouth	
Metoprolol	100	13	4	1	7	2	1	10	15
Propranolol	75	3	1		2	1			4
Pindolol	5	5	3	2	2	1		1	10
Acebutolol	240	3	2	2	2	1		1	6
Atenolol	50	2	1	2	1	1		1	2
Alprenolol	600	1	1	1				1	1
Timolol	30	1							1
No of patients per adverse effect		15	5	2	7	3	1	10	

The rapid resolution of the symptoms when the drug was stopped supports this hypothesis.

Why so few cases of arthropathy related to beta blockade have been reported over the years is difficult to explain. No similar cases have been reported to the National Centre for Monitoring Adverse Drug Reactions of the Medical Board of Finland. The main manufacturers of metoprolol have received several reports of joint complaints occurring in patients taking the drug, but in none was a clear cause and effect relation established. It may be that joint complaints are so common in the age group commonly treated with the drugs that the part these drugs play in causing joint problems escapes attention. It may also be that the phenomenon occurs more often with certain beta blockers; in our experience metoprolol seemed most likely to induce joint complaints.

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Seasonal trends in childhood asthma in south east England

Studies in other countries¹⁻³ have shown pronounced seasonal variation in admissions for acute asthma, but there are few published data from Britain. Examination of the asthma admission data for our hospital (Royal Alexandra Hospital for Sick Children, Brighton) and for the South East Thames region showed clear seasonal variation with a notable increase in admissions in the autumn.

Present study and results

Hospital admission data for asthma (code number ICD 493) were obtained through Hospital Activity Analysis for this hospital and the South East Thames region. Information was requested by age group (0-4, 5-14 years) and month and year of admission. Data for the Royal Alexandra Hospital were cross checked against the ward admission book. Full information was obtained for the 10 years 1971-80 for our hospital and the five years 1975-9 for the South East Thames region.

The figure shows the cumulated monthly admission data for the Royal Alexandra Hospital for all ages over the 10 years and the cumulated monthly admission data for the South East Thames region for the age groups 0-4 and 5-14 years. In all three groups the admission rate was low during January to April and rose in early summer. Admissions consistently fell in August and were followed by a peak in September and October. Subdividing the data for our hospital into 0-4 and 5-14 year age groups disclosed an identical pattern.

Cumulated monthly hospital admissions* of children with asthma. ●—● Royal Alexandra Hospital for Sick Children, Brighton (0-14 years), 1971-80. □—□ South East Thames region (0-4 years), 1975-9. ○—○ South East Thames region (5-14 years), 1975-9.

*In fact, Hospital Activity Analysis records asthma admissions as asthma discharges.

Comment

Inaccuracies in Hospital Activity Analysis data are most likely to arise due to diagnostic transfer. There is reluctance to use the word asthma, especially in younger children, in whom the terms wheezy bronchitis or asthmatic bronchitis have been used. These and other synonyms are usually coded as bronchitis unqualified (ICD 490). Acute bronchiolitis and acute bronchitis are coded together under ICD 466. Criteria for the diagnosis of asthma have changed little since 1970 at the Royal Alexandra Hospital and the number of admissions diagnosed as bronchitis unqualified is extremely small. Similarly the admission rate for acute bronchiolitis has not altered over the years and is mainly restricted to the months December to April.

As well as pronounced seasonal variation, examination of data for our hospital and the South East Thames region showed a steadily increasing yearly admission rate. Total admissions for asthma at the Royal Alexandra Hospital in 1971 were 75, increasing to 284 in 1980. The reasons for this are complex and the phenomenon has been noted before.⁴

The early summer peak was expected and may be related to inhaled pollens. We were surprised to find a consistent fall in admissions in August both locally and regionally. Environmental or social factors may be responsible and these need further investigation. The peak in admissions in September and October concurs with parents' views that their asthmatic children are more wheezy in the autumn and agrees with studies from other countries. Environmental trigger factors such as climatic changes, fungal and pollen spores, air pollution, viruses, and house dust mite may be important aetiologically.

It seems unlikely that childhood asthma in south east England