speed limit on certain roads in built-up areas in cases where surveys show that 85% or more of drivers are exceeding the limit. It is claimed that this results in little change in the distribution of vehicle speeds, and that in some cases accident rates have been reduced.[4] It is further claimed that drivers will not abuse what they regard as a realistic limit, and that, if the frustrations of too low a limit are removed, driver behaviour may change in a manner conducive to accident reduction. These claims do not, of course, take into account the fact that on many urban roads driving speeds are restricted by the sheer volume of traffic as much as by speed limits. Even if these claims are justified, it does not follow, as the Minister has tried to argue[5] in support of his decision to relax the existing limits, that similar considerations will apply on rural roads, where the risk of serious injury is much greater. The Minister has pointed out that compliance with the existing limits fell between 1975 and 1976 and that they are difficult to enforce to the extent needed to maintain respect for road traffic law. What he did not go on to say is that the provisional figures for 1976 show a $4\frac{1}{2}$% increase in casualties over 1975 with a 10% increase in the last quarter.[6]

From the public health point of view it is important to appreciate that the effect of speed restrictions is most definite in those accidents which result in death or serious injury. Experiments on selected stretches of road have shown that repeated stepping up of enforcement rates results in very substantial casualty savings—not only on the selected roads, but on adjacent roads as well.[7] Speed limits will not be effective unless they are properly enforced. A study in Michigan showed that the chance of being detected while exceeding the limit on one road was only once in 7600 violations.[8] The results of such low levels of enforcement are that drivers no longer feel any responsibility for observing the limit and merely consider themselves to be unlucky if they are detected.

Public health authorities, who are concerned about the increasing number of victims permanently incapacitated from road accidents, and the medical profession, which has to deal with the consequences, may not be aware of the arguments put forward in support of relaxing speed limits as they were not included in the list of organisations to which the Minister of Transport sent his consultation letter on this important issue. The leading argument appears to be that changes in driving habits such as the more prudent use of the accelerator and brakes, and increasing resort to smaller cars (which reduce protection against injury) have been more effective than speed restrictions in reducing fuel consumption, which is, of course, the reason why they were introduced. If we did the right thing for the wrong reason in 1973, it is all the more important that we should not now do the wrong thing for the right reason.

[1] Department of the Environment, Scottish Development Department and Welsh Office, *Road Accidents Great Britain 1975*, p xv. London, HMSO, 1977.

[2] Scott, P P, and Barton, A J, *The Effects on Road Accident Rates of the Fuel Shortage of November 1973 and Consequent Legislation*. TRRL Supplementary Report 236. Crowthorne, Transport and Road Research Laboratory, 1976.

[3] *Hansard* (House of Commons), 6 April 1977, cols 1198-1201.

[4] Sabey, B E, in *Epidemiological Effects of Traffic Speed and Speed Limitation*, ed E L Nordentoft, J A Wallin, and H V Nielsen, p 185. Odense, Odense University Press, 1975.

[5] *Hansard* (House of Commons), 6 April 1977, cols 519-520.

[6] Department of Transport, *Road Casualties: Provisional 1976 Figures*. Press Notice No 116, 13 April 1977.

[7] Munden, J M, *An Experiment in Enforcing the 30 mph Limit*, RRL Report LR24. Harmondsworth, Road Research Laboratory, 1966.

[8] Organisation for Economic Co-operation and Development. *Report of Research Group (S 6) on the Effects of the Enforcement of Legislation on Road User Behaviour and Traffic Accidents*. Paris, Organisation for Economic Co-operation and Development, 1974.

Polymyalgia arteritica

Giant-cell (cranial) arteritis was first described by Horton and his colleagues[1] at the Mayo Clinic, Rochester, Minnesota, in 1932; polymyalgia rheumatica was first so named by Hugh Barber[2] of Buxton in 1957, though Kersley[3] and Bagratuni[4] had previously described it as a probable variant of rheumatoid arthritis in the elderly. Barber followed up his patients for as long as 10 years and, finding that they did not develop rheumatoid arthritis, suggested the title polymyalgia rheumatica. Since then, arteritis has been shown to be the underlying pathological process in both disorders, and Hamrin and his co-workers[5 6] suggested it should be termed polymyalgia arteritica. Patients with symptoms of polymyalgia may develop features of giant cell arteritis and vice versa, and Hart[7] among others has emphasised that visual complications occur in both syndromes.

The treatment of both polymyalgia rheumatica and giant cell arteritis is the same—prednisolone or a similar corticosteroid. A low dosage of 6-10 mg prednisolone daily will control morning stiffness and girdle pains in the shoulders and hips very adequately, but it is not enough to prevent visual complications, for which 40-50 mg daily is required. In 1974 Anderson and Bayles[8] claimed that no patient on this full dosage and with a sedimentation rate restored to normal had been reported as losing her vision, but this remains the crucial hazard—thrombotic obliteration of the central artery of the retina can occur in a few hours at lower dosages. Any patient with the polymyalgia syndrome should therefore be warned to report immediately to her general practitioner any sudden exaggeration or spread of symptoms to affect temples, scalp, or occiput, and particularly any changes in vision, so that corticosteroid dosage can be instantly increased to prevent the blindness which may otherwise occur. A 12-hour delay, or even less, may prove disastrous.

Polymyalgia arteritica will disappear naturally, even if untreated, within four to eight years, and its control is usually assessed by relief of symptoms and the fall in the sedimentation rate. If the patient is symptom free, has no morning stiffness in the girdle joints, feels well, and has a sedimentation rate under 20 mm in one hour all should be satisfactory, and the daily prednisolone dosage may be reduced very gently—by as little as 0·5 to 1 mg every few weeks or months. A recent case report,[9] however, has shown that inflammatory arterial disease may persist despite normal sedimentation rates and complete absence of symptoms. Rynes and his colleagues in New York described a 70-year-old woman with typical polymyalgia rheumatica and normal findings on biopsy of the temporal artery who, despite a normal sedimentation rate and complete absence of symptoms, had a return of symptoms on reducing the dosage of prednisone from 5 mg to 4 mg daily. At this time, though the sedimentation rate remained normal (20 mm in one hour), she experienced firstly angina of effort and then pains in her temples; her right temporal artery became clinically inflamed; and her temporal artery biopsy showed giant cell arteritis. The symptoms rapidly disappeared on increasing the dose of prednisone to 20 mg daily.

Clearly, inflammatory arterial disease can escape from control without the sedimentation rate becoming truly abnormal; in this particular case the symptoms were more helpful than the results of the laboratory tests. Clinicians should depend more on symptoms and signs than on investigations and be very suspicious of cases, such as this one, where symptoms abate early under treatment. Though some cases of poly-

myalgia arteritica do abate spontaneously and completely within two years of onset, most do not: the disorder tends to return if the dosage is dropped too soon below the critical level. All too often treatment is stopped prematurely before the disease has burnt out; not only may relapse occur on reductions as small as 0·5 mg of prednisone daily, but after steroid treatment has been stopped the disease may apparently light up again some weeks or even months later. Nevertheless, given the right drug in the correct dosage for the right period of time, few conditions in medicine are more amenable to treatment, and few patients are more grateful to their doctor for the rapid and lasting relief than those with this strange disease.

[1] Horton, B T, Magath, T B, and Brown, G E, *Proceedings of the Staff Meetings of the Mayo Clinic*, 1932, **7**, 700.
[2] Barber, H S, *Annals of the Rheumatic Diseases*, 1957, **16**, 230.
[3] Kersley, G D, II Congreso Europeo de Rheumatologia. Barcelona, Editorial Scientia, 1951.
[4] Bagratuni, L, *Annals of the Rheumatic Diseases*, 1953, **12**, 98.
[5] Hamrin, B, Jonsson, N, and Landberg, T, *Lancet*, 1964, **1**, 397.
[6] Hamrin, B, *Acta Medica Scandinavica*, 1972, Suppl 533.
[7] Hart, G I *Practitioner*, 1975, **215**, 763.
[8] Anderson, L G, and Bayles, T B, *Disease a Month*, January 1974, 1.
[9] Rynes, R I, Mika, P, and Bartholomew, L E, *Annals of the Rheumatic Diseases*, 1977, **36**, 88.

ACTH-secreting lung tumours

Inappropriate hormone secretion occurs in many malignant tumours, and ectopic corticotrophin (ACTH) production is one of its more common varieties. Most ACTH-secreting tumours arise from the lung, almost exclusively oat-cell carcinomas and bronchial carcinoids. Some patients develop the florid clinical manifestations of Cushing's syndrome with hypokalaemic alkalosis, but usually there are no obvious signs and the condition is recognised by biochemical assays of plasma concentrations of cortisol and ACTH.

Bloomfield and his colleagues[1] have recently measured the concentrations of ACTH in 14 lung tumours selected at random but not associated with the ectopic ACTH syndrome. The tumours were removed by pneumonectomy or lobectomy or by local excision in the case of carcinoid tumours. At the same time lung tissue at a distance from the tumour and macroscopically free from it was used as a control. All the examples of oat-cell carcinoma and carcinoid tumour contained substantial amounts of ACTH, as did also an adenocarcinoma with large-cell carcinoid elements. By contrast, the squamous-cell, anaplastic, and glandular tumours contained insignificant amounts of ACTH, the only exception being a poorly differentiated squamous-cell carcinoma which may possibly also have contained carcinoid elements.

Of even greater interest was the finding of ACTH in the non-tumorous lung tissue, correlating well with the tumour concentrations. This could not be attributed to contamination by sequestered blood, since there was no significant correlation between ACTH concentrations in the plasma and the lung tissue, and the latter was taken at a site as remote from the tumour as possible. Bloomfield *et al* suggested that ACTH was being produced in widely dispersed cells in the lung, either as a premalignant change or secondary to tumour formation in a field of growth that included the whole lung. Alternatively, the hormone-secreting granules might themselves have metastasised and become incorporated into other parts of the lung tissue.

Just how frequent ectopic hormone secretion is in malignant tumours is only now becoming clear. Probably all oat-cell carcinomas and carcinoid tumours of the lung synthesise ACTH-like materials, though clinical evidence of the ectopic ACTH syndrome is usually absent. Presumably it is the level of secretion that determines whether clinical effects occur or not. If there were a greater correlation between tumour and plasma concentrations of ACTH it might be possible to diagnose these tumours biochemically or to detect an early recurrence after removal. But at present the observation is more of pathological interest than clinical importance.

[1] Bloomfield, G A, *et al*, *Clinical Endocrinology*, 1977, **6**, 95.

Anticoagulants and heart valve replacement in pregnancy

More and more women of childbearing years have undergone heart valve replacement. The indications have been various: congenital, postinfective, or rheumatic heart disease (which is still as common as ever in many parts of the world). Patients given a prosthetic valve are generally believed to require life-long anticoagulants[1] to reduce the incidence of thrombo-embolic complications.[2] Though the risks of thrombo-embolism were appreciable with the older types of heart valve prostheses, experience with the Starr-Edwards and homograft valves have shown that these reduce the incidence of such complications.[3] Furthermore, the risks associated with replacing the aortic valve alone are much less than with mitral valve prostheses. From a recent questionnaire survey of current practice among cardiologists in the United Kingdom Oakley and Doherty[4] concluded that anticoagulants are mandatory in patients with mitral valve prostheses, but that the indications appear less strong after tissue valve replacement, particularly of the aortic valve.

Problems inevitably arise in continuing anticoagulant prophylaxis during pregnancy, for, while the treatment may be safe and effective for the mother, there may be hazards to the fetus. During pregnancy the concentrations of some blood clotting factors are increased while the protective influence of the blood fibrinolytic mechanism is reduced. These changes promote good haemostasis at delivery, but they may produce an increased tendency to thromboembolism. Any decision to withdraw long-term anticoagulant prophylaxis is complicated by clinical evidence of an increased risk of embolism when anticoagulants are stopped in pregnancy,[2] particularly during the withdrawal period, presumably owing to rebound hyper-coagulability.[5]

Hazards to the fetus result from the passage of coumarin-type drugs through the placental barrier; fetal haemorrhage may occur, partly due to immaturity of the fetal liver. Coumarins also have teratogenic effects. Abortion, stillbirth, congenital abnormality, and perinatal morbidity are all thought to be increased by use of oral anticoagulants, and the fetal mortality from anticoagulants during pregnancy has been put as high as 15%.[6]

Subcutaneous heparin appears to be a much safer way of anticoagulation during pregnancy, though the need for twice-daily injections throughout is somewhat daunting. But, though inconvenient to administer, heparin has the great