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EDITORIAL COMMENTARY

Homeostatic effects of carotid stenosis

Signalling of blood pressure changes is partly the result of carotid sinus function and activity within the afferent glossopharyngeal and vagus nerves. Carotid stenosis is common and often affects the carotid bulb where the arterial baroreceptors are situated. However, little attention has been paid to the homeostatic effects of stenosis at this point. Akinola *et al* in this issue (pp 428–32)¹ compare autonomic reflexes in a group of hypertensive patients with transient ischaemic attacks (TIAs) and unilateral or bilateral carotid stenosis to matched groups of hypertensive and normotensive controls. Their findings indicate that baroreflex sensitivity is blunted, the effects being equal in patients with either unilateral or bilateral carotid stenosis compared with both control groups. In other respects, autonomic reflexes are blunted to the same extent as seen in hypertensive controls, indicating no specific involvement of cardiovascular efferents in the stenosis group. There are two main caveats. The first concerns the use of antihypertensive medication. Such treatment, especially with angiotensin converting enzyme activators (which may act on AT receptors in the area postrema) or β -blockers can affect baroreflex sensitivity. Although these medications were stopped within 1 day of the study, their central effects may be longer lasting. Secondly, other mechanoreceptors in the left ventricle and aortic arch may be intact in these patients. However, the shrewd use of a hypertensive control group

on similar medication to some extent allays some of the interpretative complications. Why the effects are similar in patients with severe unilateral and bilateral carotid stenosis is a matter of conjecture. This could indicate a complex non-linear compensatory effect within the brain, using baroreceptor input from the aortic arch and other regions. The importance of this study is threefold: firstly, such patients may be at risk of presyncopal symptoms which may be erroneously diagnosed as focal brainstem transient ischaemic attacks; secondly, impaired peripheral blood pressure regulation could affect cerebral blood flow in the post-stroke state as such patients lose cerebral autoregulation. The effects could also be accentuated in patients with tandem intracranial stenosis. Finally, it should not be forgotten that control of blood pressure may be impaired after carotid endarterectomy and the effects of antihypertensive medication in these patients should be monitored most carefully.

S M OPPENHEIMER

Correspondence to: Dr S M Oppenheimer, Cerebrovascular Program, Department of Neurology, Meyer 5–185, Johns Hopkins Hospital, 600 North Wolfe Street, Baltimore, MD 21205, USA. Telephone 00 1 410 502 5356; fax 00 1 410 614 9807.

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EDITORIAL COMMENTARY

Resurgence of diphtheria in the newly independent states of the former Soviet Union: a reminder of risk

The introduction of universal childhood immunisation programmes with diphtheria toxoid in the 1950s has led to virtual elimination of diphtheria from most developed countries. In the past, the highly contagious acute infectious disease caused by toxigenic strains of *Corynebacterium diphtheriae* had endangered in particular children of preschool age. Given a case fatality rate of 5% to 10%, diphtheria had been considered one of the most serious communicable diseases of childhood. The practice of routine mass immunisation of children resulted in a dramatic decline of incidence and mortality from diphtheria in many European countries, including the former Soviet Union where, by 1976, the incidence rate had fallen to 0.08 per 100 000 population.¹ In 1989, a World Health Organisation (WHO) meeting endorsed a recommendation that envisaged full control of diphtheria throughout Europe by 1995.² Contrary to this optimistic prediction by international experts, 1990 saw the beginning of a diphtheria outbreak in Russia that rapidly spread throughout the newly independent states of the former Soviet Union and by 1995 a total of 47 808 cases had been reported.¹

The Baltic countries were not spared. The paper by Logina and Donaghy (this issue, pp 433–8) reports on the diphtheria epidemic that swept through Latvia in 1994 to 1996, affecting a total of 731 people, mostly adults aged 30 to 50 years. Neurological complications, especially diphtheric polyneuropathy, were found in about 15% of patients admitted to hospital. This coincidence provided the opportunity for a detailed study of 50 patients with diphtheric neuropathy. The authors remind the readership of the typical biphasic disease course of diphtheric polyneuritis, with the characteristic early bulbar dysfunction due to toxic paralysis of the soft palate and pharyngeal muscles, followed by the delayed onset of other cranial nerve palsies and often a generalised peripheral neuropathy. Neurological and cardiac complications were particularly common in patients with severe faucial diphtheria. The paper further emphasises the importance of administering antitoxin as soon as the diagnosis of diphtheria is suspected, without waiting for culture confirmation. Effectiveness of the anti-toxin can only be expected as long as the toxin has not yet bound to tissues— that is, within the first 2 days from onset of symptoms. As diphtheria antitoxin is derived from horses, appropriate precautions and sensitivity skin testing should be performed before its administration.

The reasons for this resurgent diphtheria epidemic are not fully understood. Major contributing factors were economic hardship, crowding due to large urban migration, and failing health systems as a result of the dissolution of the Soviet Union. Consequently, low vaccination coverage and inappropriate primary vaccination practices led to the presence of many susceptible children. Moreover, as vaccine induced immunity wanes over time unless periodic boosters are administered every 10 years, many adults had again become susceptible to the disease. Before the vaccine era, most persons had acquired natural lifelong immunity in childhood through exposure to *C. diphtheriae*. Thus the susceptibility of adults to diphtheria is a new phenomenon of the vaccine era. Serological studies in the United Kingdom, Germany, newly independent states of the former Soviet Union, and in the United States indicate that 20% to 50% of adults aged >20 years are susceptible to diphtheria.^{2–5} These studies showed a significant trend of decreasing immunity with increasing age, resulting in lack of protection, particularly for adults aged 30 to 50 years. This potential risk assumes a particular significance in today's international travel. The WHO Advisory Committee on Immunisation Practices recommends that all children receive routine vaccination with a series of five doses of DTP at ages 2, 4, 6, and 12–15 months and 4 to 6 years; booster diphtheria-tetanus immunisations should then be administered every 10 years. Persons travelling to areas with diphtheria activity should have completed the primary series and should have received a dose of vaccine within the previous 10 years.

A F HANN

Department of Clinical Neurological Sciences, London Health Sciences Centre, University Campus, 339 Windermere Road, London, Ontario N6A 5A5, Canada

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