pedal cyclists, 98 (19%) had mild concussion, 13 (3%) had severe head injuries, and 58 (11%) had cuts and grazes.

The Oxford ring road encloses 227 km of road and only 23 km of cycle lane, of which 8 km are part of the road and 15 km are separate. The number of injuries per kilometre was 0·9 on the road, 0·4 on the road cycle lane, and 0·3 on the separate cycle track. Another vehicle caused or was involved in 1012 (55%) of the accidents.

Comment

In accidents motorcyclists have been shown to sustain more severe injuries to the body and pedal cyclists more severe head injuries,[2] a finding confirmed by our study. Although the two groups are not strictly comparable, we suggest that if cyclists wore helmets the number of head injuries in this group would be reduced. The incidence of head injuries sustained by pedal cyclists is similar whatever their age.[2] Though children form the largest group of cyclists with head injuries, it would be desirable for all cyclists to wear helmets.

Our study suggests that cycle lanes are safer than ordinary roads for cyclists. After the introduction of cycle lanes on certain routes in Oxford the number of accidents remained the same despite double the number of road users (unpublished report of city engineer). It has been suggested that cycle lanes relocate bicycle accidents to the place where the lanes end[3]; thus special cycle routes using quieter parts of the existing road system should perhaps be developed. All the deaths in our study and 87% of those in the study by Nixon *et al*[1] occurred after an accident with a motor vehicle and could have been averted by complete segregation of cyclists. Although this aim is currently impracticable because of the dominance of cars, cycle lanes and especially separate cycle tracks are a worthwhile addition to city centres.

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(Accepted 12 February 1987)

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Increased prevalence of non-secretors in patients with Graves' disease: evidence for an infective aetiology?

Graves' disease is associated with HLA-B8 and HLA-DR3 on chromosome 6. The abnormalities of humoral immunity causing the production of antibodies that stimulate thyroid stimulating hormone receptors, and the role of cell mediated immunity, are well known, but the trigger that allows predisposed people to develop the disease is not. Environmental influences such as stress and infection have been proposed.[1] An inability to secrete the water soluble glycoprotein form of the ABO blood group antigens is associated with increased susceptibility to several infections, particularly in children in the time between their losing maternal antibodies and developing their own active immunity. This characteristic does not alter with age or environment. The secretor gene is on chromosome 19 and is not linked to sex or the HLA markers. There is a significantly higher proportion of non-secretors in patients with insulin dependent diabetes mellitus[2] and ankylosing spondylitis[3] than in the general population. These conditions are closely associated with HLA markers, and an infective aetiology has been suggested for each. We examined the secretor state of patients with organ specific autoimmune thyroid disease. This group of diseases is not uncommon in patients with insulin dependent diabetes mellitus and their first degree relatives.

Patients, methods, and results

We studied 77 patients with Graves' disease, 33 with spontaneous primary atrophic hypothyroidism, and 26 with Hashimoto's thyroiditis. ABO and Lewis blood groups were determined by agglutination and the secretor state from saliva by the haemagglutination inhibition method.[4] Secretor state was confirmed by the Lewis blood group. The results were compared with those from local blood donors with the χ^2 test. The table shows that the proportion of non-secretors with Graves' disease was significantly increased compared with that in the control population ($p<0·005$). This was not true for patients with primary atrophic hypothyroidism or Hashimoto's thyroiditis. There was no significant difference in the distribution of ABO blood groups between any of the categories of thyroid disease and the control population.

Blood group and secretor state of patients with autoimmune thyroid disease and controls. Values are numbers (percentages) of subjects

	ABO blood group				Secretor state	
	A	B	O	AB	Secretor	Non-secretor
Graves' disease (n=77)	28 (36)	6 (8)	40 (52)	3 (4)	43 (56)	34 (44)
Spontaneous primary atrophic hypothyroidism (n=33)	11 (33)	3 (9)	18 (55)	1 (3)	23 (70)	10 (30)
Hashimoto's thyroiditis (n=26)	5 (19)	1 (4)	19 (73)	1 (4)	19 (73)	7 (27)
Controls (n=334)	104 (31)	42 (13)	173 (52)	15 (4)	245 (73)	89 (27)

Comment

Our results show that the inherited susceptibility to Graves' disease is associated with genetic markers on chromosome 19 as well as on chromosome 6. As some patients with Graves' disease eventually develop hypothyroidism it might have been expected that patients with primary atrophic hypothyroidism would show the same increased proportion of non-secretors. This was not so, but the number studied was small.

The increased susceptibility of non-secretors to infection raises the possibility that an infective agent may play a part in the development of Graves' disease. A striking association has been shown between the prevalence of antibodies against the enteric pathogen *Yersinia enterocolitica* and autoimmune thyroid disease, particularly in Scandinavia.[5] This organism, in addition to *Escherichia coli* and other Gram negative bacteria, has been shown to contain a binding site for thyroid stimulating hormone, and this is recognised by immunoglobulins from patients with Graves' disease.[1] Possibly the increased susceptibility of non-secretors to bacterial infection might result in the production of antibodies to microbial antigens that then cross react with the receptor for thyroid stimulating hormone and trigger the onset of hyperthyroidism.

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(Accepted 18 January 1988)

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