Epidemiology

Eight-year experience with 3392 endoscopically proven duodenal ulcers in Durban, 1972-79

Rise and fall of duodenal ulcers and a theory of changing dietary and social factors

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SUMMARY From 1972 to 1979, a total of 3392 patients with endoscopically proven ulcers were seen, in six ethnic groups. The distribution was as follows: Africans 456 males, 182 females; Muslim Gujerati Indians 206 males, 60 females; Hindu Hindi 433 males, 135 females; total North Indians 639 males, 195 females; Hindu Tamils 593 males, 184 females; Hindu Telegu 179 males, 46 females; total South Indians 872 males, 230 females, and Whites 465 males, 303 females. In the continent of India, it is predominantly the South Indians who suffer from duodenal ulcer. In Durban, the number of North Indians with duodenal ulcers approximates that of those from the South (North: South ratio 0.83). The first question raised by this study is that the protective factors in North Indians in India are not genetic, and are lost when they emigrate to Natal. This may be due to changes in diet. A seasonal analysis indicates that, for females, there is a striking Autumn and Winter predominance in all Indian groups, reaching 80% in Muslims and Telegus but not in African females (52-7%). The second question raised by this study is that protective factors must be sought which operate in Indian females in the Spring and Summer months. The third question emanating from this study is that duodenal ulcers (and ischaemic heart disease) appear to increase in times of dietary and social change. This occurred in the West from 1890 to 1960, and is still occurring in the Third World. The restoration of dietary fibre and unsaturated fat, and the possible adjustment to stress in the West since 1960, has been accompanied by a fall in the incidence of these diseases. A ‘changing factors’ theory of duodenal ulcers and ischaemic heart disease is proposed. These conditions fall when a ‘plateau situation’ is reached.
increase in Indians, from 1950 to 1975. Black patients presented in a ratio of 6:8 men to every woman during 1950–59. Between 1960 and 1962 there appeared to be a fairly rapid drop to a ratio of 2:5 men to every woman, which has remained fairly constant to the present time (1972–75: 2:8:1). No change in interests or diagnostic technique occurred until 1972. The increase in incidence antedated endoscopy, which was not used until the 1972–75 period.

Table 1 depicts the number of endoscopically proven duodenal ulcers seen in Durban in the period 1972–79. The figures were taken from all the major hospitals and gastroenterologists’ practices in Durban. A total of 3392 duodenal ulcers were seen. Patients from North India accounted for 41% and patients from South India accounted for 51% of Indian patients. A comparison between the percentage breakdown of duodenal ulcers in the Indian ethnic groups and the percentage of these ethnic groups in Metropolitan Durban superficially reveals that a greater percentage of Muslim Gujeratis have ulcers than their percentage in the population (13% of ulcer patients compared with 6% in the population). However, the classification of the ethnic groups was inconsistent—that is, in the analysis performed by Mistrya on the population, 18% of Indians were unclassified, whereas, in the classification performed by us, only 8% were unclassified. The male:female ratio altered very little over the eight year period, being 1:6:1 in Whites, 2:5:1 in Africans, 3:3:1 in North Indians, and 3:4:1 in South Indians.

For each ethnic group the incidence (Table 2) appeared to rise progressively in most instances until 1976, and then fell progressively thereafter. This held for both males and females. However, the total number of endoscopies was also rising until 1976, so the apparent rise from 1972–76 can be largely discounted. During the period when the total number of endoscopies were steady—that is, 1976–79—the incidence of duodenal ulcers progressively fell in Africans and both North and South Indians. In Whites the level appeared to be steady during this period. In Durban, Indians from the north of India have a greater incidence of duodenal ulcers than those from South India. This suggests that the protective factor operative upon Indians who live in North Indiaa is environmental, and is lost when they emigrate to Natal, possibly because of urbanisation.

### Table 1 Duodenal ulcers, during 1972–79

<table>
<thead>
<tr>
<th>Ethnic Group</th>
<th>Male (%)</th>
<th>Female (%)</th>
<th>Total (%)</th>
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</thead>
<tbody>
<tr>
<td>North Indians</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Muslim Gujerati</td>
<td>206 13</td>
<td>60 13</td>
<td>6</td>
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<tr>
<td>Hindu Hindi</td>
<td>433 28</td>
<td>135 30</td>
<td>26</td>
</tr>
<tr>
<td>South Indians</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Hindu Tamils</td>
<td>593 39</td>
<td>184 41</td>
<td>38</td>
</tr>
<tr>
<td>Hindu Telugu</td>
<td>179 12</td>
<td>46 10</td>
<td>12</td>
</tr>
<tr>
<td>Unclassifiable</td>
<td>122 8</td>
<td>28 6</td>
<td>18</td>
</tr>
<tr>
<td>Total Indians</td>
<td>1533</td>
<td>453</td>
<td></td>
</tr>
<tr>
<td>Africans</td>
<td>456</td>
<td>182</td>
<td></td>
</tr>
<tr>
<td>Whites</td>
<td>465</td>
<td>303</td>
<td></td>
</tr>
<tr>
<td>Total ulcers</td>
<td>2454</td>
<td>938</td>
<td></td>
</tr>
<tr>
<td>Total</td>
<td>3392</td>
<td></td>
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</table>
Experience with 3392 endoscopically proven duodenal ulcers

HYPOTHESIS

"CHANGING FACTORS" THEORY OF DUODENAL ULCERATION

It is postulated that duodenal ulcers occur in groups subjected to change. The change may be due to stress, diet, genetics, or some other as yet undefined factor. In the case of stress or diet, such changes may be sudden or gradual. Genetic changes may occur over a generation, because of migration or assimilation. Duodenal ulcers have been reported where each of these changes has occurred (Figure), and decline where these changes cease.

West and South Africa, 1900–39

In the West and South Africa during the period 1900–39 (Table 3), duodenal ulcers increased in White executives during a period of social change with little economic security. In the workers, the lower incidence of ulcers cannot be explained. In the West, the incidence of duodenal ulceration in executives rose from being a rare disease in the 19th century to a peak in the early 1960s, a rise which was accompanied by very specific social change. The occurrence of duodenal ulcer in populations who had recently left rural areas led Susset to suggest that 'early urbanisation' played a prominent part in its pathogenesis. Stress may have occurred because of upward social mobility, and loss of social support systems. The London blitz was associated with an increase in perforated duodenal ulcers and army training resulted in an increase in duodenal ulceration and acid output. The prospect of instant nuclear annihilation may have created a very specific stress because of the feeling of 'helplessness' induced in the population. Profound dietary changes occurred during this period. Fibre was being rapidly removed from the diet. Cleave has suggested that the introduction of refined carbohydrate foods such as white flour, polished rice, and refined sugar, results in a loss of protein buffer and predisposes to duodenal ulceration. This concept fits with the 'theory of changing factors'. As the diet became more refined, duodenal ulceration increased in the West.

In areas of Africa and India, duodenal ulcers occur in districts where polished rice or starch foods are consumed. These include South India, the coastal areas of West Africa, and the cities of both continents. In the low incidence areas of North India and rural Africa unrefined wheat or maize is consumed. This may require more mastication, and the saliva thus produced may protect against duodenal ulceration. It was shown in a controlled study that ulcer patients from southern India were protected from ulcer recurrence when they were placed on the unrefined northern diets. Tovey has experimental evidence to show that there are protective dietary factors in unrefined wheat or rice, horse gram, millets, some fresh vegetables, eggs, butter, and full-fat milk. Some changes were occurring because of urbanisation. Diet and habits were changing (removal of fibre, increased saturated fat and refined carbohydrates), and there was little economic security.

West, 1960–80

In the West from 1960–80, fewer executives developed duodenal ulcers; this was a period of little dietary change, and there was a possible adjustment to stress and other adverse social factors. In the 1960s stress may have increased in the West to the point where the population ceased to react to it. During this period fibre, unsaturated fat, and unrefined sugar were reintroduced into the diet. It is suggested that, because of these conditions, duodenal ulceration began to decrease, and will, it is predicted, continue to diminish unless relevant social or other changes intervene once again. An inconsistency remains in the situation of the workers.

Table 3 Social and dietary changes and duodenal ulcers

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<td>Executives:</td>
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<td>Changes in society</td>
<td>Yes</td>
<td>No</td>
<td>No</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Changes in diet</td>
<td>Yes</td>
<td>No</td>
<td>No</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Workers:</td>
<td>Changes in society</td>
<td>Yes*</td>
<td>No*</td>
<td>Yes</td>
<td></td>
</tr>
<tr>
<td>Changes in diet</td>
<td>Yes*</td>
<td>Yes</td>
<td>Yes</td>
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*Inconsistencies.
who developed duodenal ulcers in a pattern of unchanging social security. Other factors, as yet undefined, may play a role in the occurrence of duodenal ulcers in the working class during this period. A form of continuing change in the West is provided by migration, especially of working classes. A greater incidence of duodenal ulceration in foreign workers was noted in Germany both for men and women, aged between 20 and 30 years in one report, and for men only, in unskilled labourers.

South Africa, 1960–80
In South Africa from 1960–80, the incidence of duodenal ulcers fell in executives, in a period of unchanging and stable social security and dietary factors. African and Indian workers did develop duodenal ulcers under the conditions of increasing social stress and dietary changes (removal of fibre, increased refined sugar, and increasing saturated fat). Most reports from areas of high duodenal ulcers in Africa report a concomitant high level of stress. This is especially so in the situation of rapid westernisation in Nigeria, the upheavals in Burundi, Ethiopia, Uganda, and Zimbabwe, and the rapid social changes in South Africa. In all of these situations, there is a continuing system of change (both stress and dietary) and an increase in duodenal ulceration.

Other reports from Africa have claimed that patients with duodenal ulceration are in lower social classes, are not different from control subjects, and are not in any way changed from their centuries-old behaviour by the modern world. It must be difficult to substantiate the latter claim. It has been suggested that imposition of the Western way of life has introduced many peculiar types of stress which are difficult to define, and may have been overlooked. The distribution of duodenal ulcer in rural India is not related to stress, although stress may be a factor in urban areas.

Conclusion
Duodenal ulceration appears to occur in areas subjected to an interplay between adverse social and dietary change. These factors have occurred in South India (increase in refined diet), the coastal areas of West Africa (increase in refined diet and social change), and the industrialised countries from 1890 to 1960 (both dietary and social change). It is postulated that it is the constant adverse changing of these factors which predisposes to duodenal ulceration. When these adverse dietary changes cease, or are reversed, the incidence of duodenal ulcers falls.

It is the reaction to stress, rather than the level of stress itself, which may be important in the pathogenesis of duodenal ulceration. When the level of stress reaches a critical level, the reaction to it diminishes.

The rise and fall of duodenal ulcers appears to parallel the rise and fall of ischaemic heart disease. Similar changing factors may be required for the pathogenesis of both these conditions.

It is suggested that increased diagnostic technology and access to it by the population, both in industrialised and developing countries, cannot explain the rise and fall of these conditions.

There are, of course, problems with the 'theory of changing factors', even apart from the obvious problem of translating these changes into physiological terms (aggressive vs protective factors). First, sociologists may well tell us that earlier eras were subjected to stress similar to that of modern Western man without the development of duodenal ulceration. Perhaps dietary habits protected them (fibre, unrefined sugar, little fat). Second, the shift of duodenal ulceration to the lower social classes in the West needs to be critically examined.

I wish to express my grateful thanks to Dr F I Tovey for reviewing and criticising the manuscript.

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