

The trauma of the urban experience

ISIDOR SEGAL, FRCP, *Head of Gastroenterology Unit
Baragwanath Hospital and the University of the Witwatersrand,
Johannesburg, South Africa*

Diseases characteristic of modern western culture are rare or unknown in rural African communities pursuing a traditional life style. When these people migrate to the city, however, environmental factors provoke certain diseases in a manner which is not always predictable. The impact of urbanisation is thus of significance, particularly with reference to such diseases and factors as duodenal ulcer, change of type of alcohol consumed, change to a low-fibre diet, and the exposure to a modified traditional healing influence.

Social factors in the emergence of the 'western type' duodenal ulcer

The association between civilisation and duodenal ulcer is an attractive hypothesis. Susser [1] suggested that duodenal ulcer 'could be a disease of an early phase of urbanisation,' and that its decline in the West may have occurred because 'large sections of the population may by now have learnt to adapt to the demands of industrial society, so that these are felt as less stressful than before.' Johannesburg has a continuing influx of rural black people who merge into and contribute towards an ever-growing, permanently urbanised population. The black immigrant leaves a rural tribal socio-cultural milieu for the multi-ethnic western environment of the city. He moves from a subsistence economy into one requiring new skills, concepts of time, work attitudes and different kinds of work relationships. He is, in addition, removed from the closeness of a rural community to the anonymity of city life where his residential arrangements, family life and physical environment are very different from those which existed in the countryside.

This predicament is probably analogous to that which occurred in England during the industrial revolution. In the early period of industrialisation a wave of peptic ulcers first appeared in young men in the higher social classes [1]. This was confirmed in a study of 105 men with duodenal ulcer carried out at Baragwanath Hospital in 1978 [2]. Men with duodenal ulcers were found to be significantly better educated than their controls, most had been born in the town, and more of them were employed at higher, though not the highest, educational levels. Those who cannot cope with the new stresses, however, are unable to find comfort with the mass of their fellows. This is because they have moved upwards out of their

ranks. This might explain why Dunn and Cobb (1962) [3] found a high rate of peptic ulcer among foremen, and why the blacks in our study with duodenal ulcer were clearly upwardly mobile into the higher ranks of the working class.

Segal *et al.* (1986) [4] in a further study at Baragwanath Hospital confirmed that urbanisation is an important factor in the genesis of duodenal ulcer. Their study of 100 duodenal ulcer patients, and 100 hospital controls, matched according to sex, age, education and occupation, and an additional 50 unmatched endoscopically negative controls, confirmed that duodenal ulcer patients were mainly young men who were more likely to have been born in an urban area than the control subjects. Furthermore, a significant number of duodenal ulcer patients were born and reared in the smaller towns of South Africa where socio-economic mobility and potential is less than that of Soweto and causes frustrated aspirations. It is therefore evident that factors associated with urbanisation, such as education, occupation and the pattern of urbanisation, play key roles in the genesis of duodenal ulcer.

The transition to a western type alcohol consumption

The impact of westernisation on alcohol consumption in an urban African environment is reflected in the change in drinking patterns of the South African black population in general, and Soweto in particular. In traditional African society, drinking a low alcohol ($\pm 3\%$), sorghum based beer was part of the social fabric.

During the past 25 years, however, the consumption of western type spirits and beers in Soweto and other major South African cities has to a large extent replaced traditional home brews [5,6]. This has resulted in the emergence of alcohol-induced pancreatitis and an alteration in the pattern of micronodular cirrhosis [7,8]. Prior to 1962, legislation forbade the sale of western type liquor to the black population. This legislation was subsequently repealed and resulted in a radical change of drinking habits. Evidence for this change is that prior to 1962 iron overload was common in adult black men of Ghana, Malawi and Southern Africa where the majority of adults showed varying degrees of tissue siderosis. In 1950, Walker and Arvidsson [9] demonstrated that the excess iron was due mainly to the uptake of the element from iron utensils used during the preparation of fermented alcoholic beverages.

Correspondence to: Professor I. Segal, Gastroenterology Unit, Baragwanath Hospital, PO Bertsham 2013, South Africa.

The significance of diminished dietary iron consumption

There has been a reduction in both the prevalence and severity of iron overload in urban South African black men over the past 20 years [10]. This can be ascribed to several changes in drinking habits. Commercial breweries are now the major source of fermented alcoholic drinks. In addition, commercially produced beers, which in the past were prepared or transported in iron containers, are now produced in stainless steel equipment. The new legislative directive, however, was the most important reason for the change in drinking habits. This has resulted in an increase in the consumption of spirits, notably brandy and fortified wine and beers, with a corresponding decrease in the consumption of home brewed traditional beverages.

Additional evidence for the change in drinking habits in blacks is the alteration in the manifestation of liver disease. Micronodular cirrhosis associated with iron overload is characterised by the presence of large quantities of haemosiderin and there is no fatty change, alcohol hepatitis, alcohol hyalin or alcoholic cirrhosis. This contrasts with the presentation of micronodular cirrhosis observed in western societies after prolonged consumption of excess alcohol. Micronodular cirrhosis with fatty change, alcoholic hyalin, alcoholic hepatitis and alcoholic cirrhosis have now appeared in the black population [10]. It is significant that 15 years after western type alcohol was made available, alcohol-induced chronic calcific pancreatitis (CCP) began to be diagnosed in Soweto. Observations over a recent three-year period (1981-1983) on 55 patients show that it has devastating effects in terms of morbidity with a high incidence of complications and a mortality of 15 per cent [7]. CCP affects mainly young men (mean age 40 years) and develops approximately 16 years after the commencement of drinking alcohol. Diabetes was the most common complication, occurring in 45 per cent of patients. Obstructive jaundice was observed in 25 per cent. Pulmonary tuberculosis affected 10 patients (18%). Possible reasons for the high morbidity rate are that diagnosis was usually made at an advanced stage of the disease, most patients did not stop drinking and compliance with treatment was poor. With industrialisation and particularly with the introduction of western type alcohol into developing countries of Africa the probable pattern of CCP in these regions will be similar to that observed in the South African black population.

Paradoxically, in areas where traditional brews are still consumed, the replacement of the nutritious sorghum by vitamin deficient maize in the brewing of beer is cited as one of the major reasons for the epidemic of oesophageal cancer in certain regions of Africa [11].

Cook [11] proposed a hypothesis that both the geographical and temporal distribution of oesophageal cancer in Africa could reflect the use of maize as a major ingredient of alcoholic drinks. The traditional beer is made from malted sorghum and a starchy adjunct — sorghum grain or maize (corn) [12]. In fact maize has been an ingredient of beer even before the turn of the

century [12]. It would thus seem that although there is evidence for a geographical association between maize in beer and cancer of the oesophagus, the temporal distribution does not coincide as oesophageal cancer first became a major problem in fairly recent times (1950s) whereas maize has been used for beer making before 1900 [13]. However, the percentage of maize used in beer has increased considerably in recent times. A typical recipe given by Oxford (1926) contains maize meal 27.8 per cent; sorghum meal 37.6 per cent and sorghum malt 34.6 per cent [14]. In 1964, however, 56 per cent of the content was derived from maize [12] and a recent case control study of 201 oesophageal cancer patients showed that 92 per cent of patients used maize as the flour for their beer (I. Segal and S. G. Reinach, unpublished study). The use of maize instead of sorghum grain has resulted in a decrease in the thiamine, niacin and riboflavin content of traditional brews [15]. This would have dramatic effects on the B vitamin status of people who consume large quantities of beer. From his studies in the Transkei, Van Rensburg [16] has concluded that 'the dominant and unifying factor in the aetiology [of oesophageal cancer] seems to be the nutritional status. Most evidence suggests that a chronic low status of zinc, magnesium, riboflavin and nicotinic acid predisposes the oesophageal epithelium to cancer formation' [16,17].

The transition to a low fibre diet

Urbanisation invariably evokes a change in diet and dietary patterns. Evidence indicates a causal relationship between diet and some 'western' chronic digestive diseases [18]. The evidence is particularly impressive in appendicitis, diverticular disease, cancer of the colon and gallstones. The major dietary factors implicated are dietary fibre, animal fat and refined sugar [19-25].

A dietary survey of urbanised blacks in Soweto (1986) [26] showed that the diet was adequate in energy intake (1840 kcal) and gross protein, and high in carbohydrate (266.3g). Sugar intake was modest (52.2g), and animal fat intake was low (17.8g). A dramatic change is the reduced intake of dietary fibre (13.7g). Reasons for the low fibre intake are that the staple maize meal which forms a large portion of the everyday diet of urban and rural dwellers, is refined; the consumption of legumes has fallen and vegetables and fruit are expensive. Thus the dietary fibre intake of present day urban blacks is explainably low and likely to remain so. It is puzzling that the dietary changes have not yet evoked significant increases in diet-related diseases. World War II caused the national diets of some western populations to be altered in pattern: the fat intake fell slightly, yet the fibre intake rose considerably. These changes were propitious for a decrease to occur in frequencies of non-infective bowel diseases. In the countries most affected by the dietary changes, the frequency of appendicitis and of diverticular disease fell. Moreover, there is some evidence that the mortality rate from colon cancer was also affected [27-29].

These decreases were associated with changes in diet that lasted only a few years. It is of considerable import-

ance that the frequencies of the diseases mentioned rose when war-time diets reverted to their former higher-fat, lower-fibre intake pattern. Turning to the local situation, our initial view was that the duration of reduced fibre intake among urban blacks had not been long enough to evoke significant rises in the frequency of most bowel diseases. Although the period of low fibre intake is unknown it is believed to be less than a decade. In 1974, a dietary survey of urban blacks in Cape Town showed a fibre intake of 25 g daily [30]. Another possibility is that a significant increase in the occurrence of bowel diseases requires not only a low or decreased fibre intake but also an increased fat intake.

Urbanisation and the changing relationship between the patient and the traditional healer

Urbanisation may have bizarre implications. This is reflected in the attempt of the urban black to retain vestiges of traditional healing; most of the population of Soweto consult initially with a traditional healer [13].

For effecting a cure, the traditional healer uses enemas and emetics more commonly than other forms of treatment. They are used for various reasons including for ritual purposes; as an aperient, aphrodisiac or emetic; and for the treatment of impotence, gastrointestinal disturbances and dysmenorrhoea. In the city, use and abuse of emetics and enemas can result in damage to almost the entire gastrointestinal tract [31,32].

In particular, the use of enemas containing substances such as vinegar, soap, caustics, chloroxylenol, potassium dichromate, copper sulphate, potassium permanganate and brown sugar can result in colitis, peritonitis and rectal bleeding. It may be thought paradoxical that urban people should accord such respect to what in western terms may seem an anachronism. The belief in mysticism is, however, an integral element of African culture and is fostered in the urban situation by the traditional healer.

Conclusion

Urbanisation is proceeding at an alarmingly high rate in developing countries. It is predicted that by the year 2025 more than half of the people of Asia and Africa will be living in cities [33]. This is particularly significant considering how overwhelmingly rural these continents have been up to now.

Undoubtedly diseases associated with urbanisation will become more prevalent in developing countries. Unfortunately, because of the impoverished state which most of these countries experience today it is probable that it will take place in a chaotic manner and the urban areas are likely to remain poor. The key question will be how to provide food and essential services on the scale required in these areas. Based on our limited experience, and concerning mainly gastroenterology, it is predicted that socio-economic factors will determine the trends of disease patterns. Consequently, there will be an increase in diseases associated with stress, adaptation to urban life, and alcoholism (duodenal ulcer, alcohol-induced pancreatitis, alcoholic liver disease). Diseases associated with

westernisation, such as appendicitis, diverticular disease, inflammatory bowel disease and colo-rectal cancer will increase, although at a slower rate. A formidable challenge will be to prevent the catastrophic effects of infections such as gastroenteritis, and deficiency diseases, principally kwashiorkor, which already haunt the developing countries.

Acknowledgements

I wish to acknowledge the support of the Chairman's Fund, Anglo-American Corporation.

References

1. Susser, M. and Stein, Z. (1962) *Lancet*, **i**, 1115.
2. Segal, I., Dubb, A. A., Ou Tim, L., et al (1978) *British Medical Journal*, **1**, 469.
3. Dunn, J. P. and Cobb, S. (1962) *Journal of Occupational Medicine*, **4**, 343.
4. Segal, I., Unterhalter, B. and Rosenbusch H. (1986) *Social Science and Medicine*, **23**, 417.
5. Moshal, M. G. (1973) *Digestion*, **9**, 438.
6. Van Niekerk, M. C., Schmid, E. U. and Mieny, C. J. (1986) *South African Medicine*, **70**, 58.
7. Segal, I., Leros, M. and Grieve, T. (1984) In *Pancreatitis, concepts and classification* (eds K. Gyre et al.) p417. Amsterdam: Excerpta Medica.
8. Isaacson, C. (1982) In *Pathology of a black African population*, p37. Berlin: Springer Verlag.
9. Walker, A. R. P. and Arvidsson, U. B. (1953) *Transactions of the Royal Society of Tropical Medicine and Hygiene*, **47**, 536.
10. Isaacson, C. (1982) In *Pathology of a black African population*. Berlin: Springer Verlag.
11. Cook, P. (1975) *British Journal of Cancer*, **25**, 853.
12. Novellie, L. (1968) *Wallerstein Laboratory Communications*, **31**, 17.
13. Novellie, L. (1986) In *Indigenous fermented foods of non-western origin*. (eds C. W. Hesselbline and H. L. Wong) Mycologia Memor No. 11, p219. Berlin: J. Cramer.
14. Oxford, T. (1926) *Journal of the Institute of Brewing* **32**, 314.
15. Zamit, I. V. (1980) *South African Food Review* (Suppl), **7**, 73.
16. Van Rensburg, J. J. (1981) *Journal of the National Cancer Institute*, **67**, 243.
17. Craddock, V. M. (1987) *Lancet*, **i**, 217.
18. Trowell, H., Burkitt, D. and Heaton, K. (eds) (1985) *Dietary fibre, fibre-depleted foods and disease*. London: Academic Press.
19. Ashley, D. J. B. (1967) *Gut*, **8**, 533.
20. Burkitt, D. P. (1971) *British Journal of Surgery*, **58**, 695.
21. Painter, N. (1985) In *Dietary fibre, fibre-depleted foods and disease*. (eds H. Trowell et al.) p289. London: Academic Press.
22. Nelson, R. L. (1983) *Current Surgery*, **40**, 419.
23. Nigro, N. D. and Bhadrachari, C. C. (1973) *Diseases of the colon and rectum*, **16**, 438.
24. Hill, M. J. (1986) In *Dietary fiber: basic and clinical aspects*. (eds B. V. Vahouny and D. Kritchevsky). New York: Plenum Press.
25. Heaton, K. (1985) In *Dietary fibre, fibre-depleted foods and disease*. (eds H. Trowell et al.) p 289. London: Academic Press.
26. Segal, I. and Walker, A. R. P. (1986). *Nutrition and Cancer*, **8**, 185.
27. Fleisch, A. (1946) *Schweizerische Medizinische Wochenschrift*, **16**, 889.
28. Chi, G., Minowa, K. and Oyama, T. (1983) *American Journal of Clinical Nutrition*, **38**, 115.
29. Magee, H. E. (1946) *British Medical Journal*, **1**, 475.
30. Manning, E. B., Manning, J. I., Sophangisa, E. and Truswell, A. S. (1974) *South African Medical Journal*, **48**, 485.
31. Segal, I., Ou Tim, L., Hamilton, D. G. et al. (1979). *Diseases of the Colon and Rectum*, **22**, 195.
32. Segal, I. and Ou Tim, L. (1979) *South African Medical Journal*, **56**, 308.
33. United Nations (1985) *World population trends, population and development interrelations and population policies*. 1983 Monitoring Report. Vol 1. Population trends. Department of International Economics and Social Affairs. Population Studies No. 93, p181. New York: United Nations.