Health and poverty—A tale of two cities


**SUMMARY**

This paper examines health inequalities between Scotland’s two largest cities, Glasgow and Edinburgh, by studying the all-cause and cause-specific mortality over a 50-year period (1931–81). The deaths analysed were in 10-year age groups for men and women aged 25–74 years occurring in 5-year periods centred on the census year between 1931 and 1981. The denominator was taken from the censuses for 1931 to 1981 (except 1941 when the mid-point estimate was taken from the 1931 and 1951 census data). The total number of deaths was 106,920 in Glasgow and 46,020 in Edinburgh. The results show that, as expected, age- and sex-specific mortality rates have declined steadily in both Glasgow and Edinburgh from 1931 to 1981, but have always been lower in both men and women in Edinburgh. During the period 1979–83, in all cohorts over 25 years of age, men in Glasgow died 3.9 years earlier than in Edinburgh. The corresponding figure for women was 3.6 years. From 1961 onwards the log death rates have risen linearly with age but are parallel between the cities. These differences have increased and are predicted to increase further in men. The authors conclude that the cross-sectional difference in mortality (40%) seen between the cities is determined by the mortality levels in early adulthood and the difference remains constant within each cohort between 25 and 74. They argue that studies of specific diseases or specific risk factors are inadequate to explain these health inequalities and the differences are likely to be due to socio-economic factors in addition to the prevalence of specific risk factors in both populations. Hence they advocate strategies which combat poverty and poor environment.

**COMMENT**

This is a very interesting analysis of the mortality statistics of two cities which lie in the central (industrial) belt of Scotland—Glasgow and Edinburgh. Historically, Edinburgh has been an important legislative, legal, educational and commercial centre for several centuries and is regarded as more ‘anglicized’ and middle class than Glasgow. Glasgow’s rise to prominence as Scotland’s largest city (currently with a population of 750,000) was due to the tobacco trade with the colonies of the New World in the eighteenth century and thereafter as a result of the rapid industrialization in the nineteenth century. Consequently, Glasgow has been regarded as a more working class city than Edinburgh. With that mantle came more environmental problems for Glasgow; for example, poorer housing than Edinburgh. Hence the differences in mortality between the two cities cited in the paper are not surprising since they reflect long-standing health inequalities between socio-economic groups in Britain. What is of importance is that in every cohort from 25 to 74, Glaswegians at any age have a mortality which is the same as people from Edinburgh who are about three-and-a-half years older. A leading article in the *British Medical Journal* speculated that this was due to the ‘long shadow’ of relative poverty experienced at a young age by Glaswegians being felt at a later age, or to the continual, relatively poorer environment being suffered by all Glaswegians.

The authors of the paper also note that there are larger differences in all-cause mortality in younger age groups in the most recent years analysed, predicting that the differences in mortality between the two cities would increase in the future. Their premise is that if the national health targets in Britain are to be achieved, then in addition to health promotion measures to alter adult behaviour, other strategies are necessary including ‘measures to combat child poverty and housing’.

It may seem ironic that this paper refers to health inequalities in an industrialized country, because health inequalities in Third World countries may be larger, more obvious and more urgently in need of action. However, the
fact is that health inequalities in Britain are longstanding and certainly predate 1921 when the first analysis of mortality by social class was published by the Registrar General.

In recent years these health inequalities have attracted considerable attention following the publication of the Black Report. The report was commissioned by the last Labour Government (1974–79) but was presented on completion to the incoming Conservative Government which found the report ran counter to its own ideology. Successive governments have not taken on the challenge of the Black Report with its explicit and cogent prescription of action against poverty. As a result, in Britain, health inequalities have continued to widen in recent years and this may partly explain the reason for the country slipping down the international health league. There is some evidence that the countries which have the smallest difference in income distribution also have the smallest health inequalities between socioeconomic groups. Internationally if countries are to achieve ‘Health for All’ by the year 2000, the issue of health inequalities needs to be tackled both in the industrialized and Third World countries.

REFERENCES

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COMMENT
External ear canal cholesteatoma is interesting mainly because it is uncertain whether the condition actually exists. Biber from Ohio observed that the so-called primary cholesteatoma of the external canal was actually secondary to middle ear inflammation. Granulations form where two kinds of epithelium meet. The epithelium which has a stronger potency for proliferation buries in its depth the ‘weaker’ one forming islets of epithelium and later a cholesteatoma. The present report describes patients who did not have any disease of the middle ear. It seems therefore that the term ‘ear canal cholesteatoma’ is rarely used, lesions in the external ear canal consisting of epithelial debris are well known and go by various names including molluscum tumours, pearl-tumours, epidermal plugs, myringitis desquamativa, otitis desquamativa externa, cholesteatoma-like accumulations and keratosis obturans.

Are all these identical to the ear canal cholesteatoma? One of the causes of ear canal cholesteatomas suggested by the author is canal wall obstruction or stenosis. This is also the postulated mechanism for the formation of keratosis obturans. However, pain which has been uniformly observed as the main symptom of keratosis obturans was not present in the patients described in this study. In fact, it was named ‘keratosis obturans’ because of the obstructive and painful condition in the external ear canal. The other groups which the author has described as ‘postoperative’ may have had an aetiology similar to implantation cholesteatoma of the middle ear. The cholesteatoma may have resulted from keratinizing epithelium being buried in the canal lining. Whether these two groups can be called ‘primary’ is a matter for debate. However, the spontaneously developing group seems to be a different entity. It has no relation to obstruction in the canal and leads to bone erosion not as a result of

Ear canal cholesteatomas—Do they exist?


SUMMARY
Cholesteatomas, cyst-like lesions lined by keratinizing stratified squamous epithelium, are more commonly found in the middle ear and the mastoid but can also occur in the external ear canal. The author reports his personal experience of ear canal cholesteatoma between October 1984 and June 1992. There were 7 patients, 2 of whom had bilateral disease. The presenting features ranged from no complaints to otorrhea, hearing loss and discomfort. Only 3 patients had conductive hearing loss. Examination of the ears showed an accumulation of exfoliated keratin. Interestingly, the tympanic membrane was intact though in some patients the bone around the annulus had been destroyed. In 1 patient the destruction had extended across the posterior canal wall up to the tip of the mastoid bone. The author divided his cases into five groups depending on the aetiology and divided the disease into three stages depending upon the amount of destruction. He observed certain common factors in these patients. All except one were elderly (median age 69 years), they had hard crusted wax or debris lying over the cholesteatoma and the tympanic membrane was spared. As the patients had hard wax and debris overlying the cholesteatoma the author suggests that the disease may be initiated by the inability of the epithelium to migrate normally. Treatment options include frequent cleaning with debridement of necrotic tissue and wax, topical application of 5-fluorouracil and surgery. Frequent application of oil to the ear canal seems to prevent and cure the disease.

SELECTED SUMMARIES

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