

PAPERS

Rising death rate from non-malignant disease of the oesophagus (NMOD) in England and Wales

M Z Panos, R P Walt, C Stevenson, M J S Langman

Abstract

Between 1968 and 1991, the number of deaths from non-malignant oesophageal disease (NMOD) (International Classification of Diseases code 530), recorded by the Office of Population Censuses and Surveys (OPCS) in England and Wales, trebled in women, from 118 to 340 (5 to 13 per million) and doubled in men, from 131 to 251 (5.5 to 10 per million). Calculation of age specific death rates, shows the increase to result from a rise in mortality in those over 75 years and age standardised mortality confirms a rise in overall frequency from 2.9 to 7.0 deaths per million men and 5.2 to 13.1 per million women. Between 1974 and 1988 when specific diagnoses were coded, deaths from oesophageal ulcer rose from 1.5 to 2.5 per million. In men, the death rate from oesophageal stricture increased from 2.5 to 3 per million and in women from 3.5 to 6 per million. Mortality from oesophageal perforation did not change (1 per million). Some of these changes reflect the increasing age of the population in general, but further explanations are required. Review of 84 sets of case notes from a total of 281 inpatients whose coded diagnoses had included NMOD and who had died suggested that in 28 (33%) death was actually due to NMOD, and in seven of these endoscopic intervention was responsible. The certified underlying cause of death was compared with that suggested from case note review in 62 cases; death from NMOD was substantially underestimated. This study concludes that a rising death rate attributed to NMOD is underestimated on death certificates and that endoscopic intervention explains only a few of the cases.

(Gut 1995; 36: 488-491)

Keywords: death rate, oesophageal disease.

Benign oesophageal disease in the form of reflux oesophagitis is a common complaint. The frequency of oesophagitis diagnosed at endoscopy has increased dramatically.¹ The International Classification of Diseases code 530 includes all non-malignant oesophageal disease from achalasia to stricture. Published

data from the Office of Population Censuses and Surveys (OPCS), show that between 1968 and 1988, the number of deaths from non-malignant oesophageal disease (NMOD) (ICD code 530), in England and Wales, doubled in men from 131 to 265 and trebled in women from 118 to 350.^{2,3} This large change requires explanation. The aims of this study were to identify age related changes using age specific rates, to estimate the accuracy of certification in cases of death,⁴ and to identify possible explanations through note review.

Sources and methods

National statistical data

Information published by the OPCS for the years 1968-1991, was used to derive age specific and age standardised death rates for ICD 530 for England and Wales.^{2,3,5,6} Age specific death rates are expressed as the number of deaths per thousand population in each five year age band, as estimated annually at mid-year. Analysis of data for diagnostic subcategories was possible after the introduction of more specific ICD codes in revisions, in 1974 and 1979³ (Table I). Age standardised rates were derived by direct standardisation. Age specific rates were multiplied by the numbers of subjects (of that age band) in the population to obtain the number of cases that would be expected in the standard population if rates in the study population had applied. The total number of expected cases (sum of expected cases of all age bands) in the standard population was then divided by the total number of subjects in the standard population, to obtain a directly standardised rate.

TABLE I International Classification of Diseases codes for NMOD. Revised codes as applied by the Office of Population Censuses and Surveys (England and Wales)

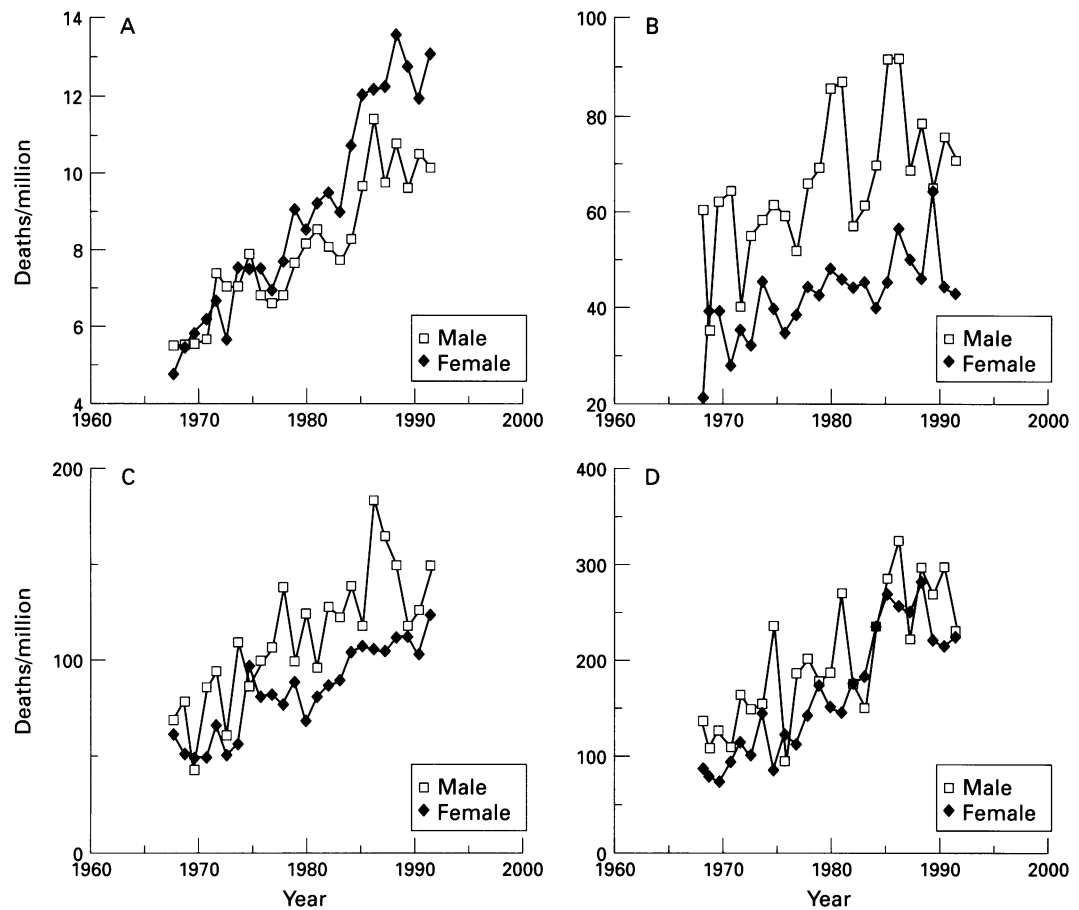
	ICD code	Diagnosis
1968-1973	530	Diseases of oesophagus (all)
1974-1978	530.0	Functional diseases of oesophagus
	.9	Other diseases of oesophagus
1979-1988	530.0	Achalasia/cardiospasm
	.1	Oesophagitis
	.2	Ulcer of oesophagus
	.3	Stricture/stenosis
	.4	Perforation of oesophagus

Department of
Medicine, Queen
Elizabeth Hospital,
University of
Birmingham,
Birmingham
M Z Panos
R P Walt
M J S Langman

West Midlands
Regional Health
Authority,
Birmingham
C Stevenson

Correspondence to:
Dr R P Walt, Department of
Medicine, Queen Elizabeth
Hospital, University of
Birmingham B15 2TH.

Accepted for publication
15 July 1994



(A) Death rate (all ages) from NMOD for men and women in England and Wales, between 1968–1991. Calculated from OPCS data. (B) 75–79 years, (C) 80–84 years, (D) 85 years and above: age specific death rate, from NMOD for men and women in England and Wales, between 1968–1991, calculated from OPCS data. Note different scales.

Review of case notes and death certificates

To obtain further details of deceased patients with a discharge diagnosis of NMOD and to assess the accuracy of death certificates for this diagnosis we obtained discharge/death statistics (based on form KMR1), from the West Midlands Regional Health Authority. In the three years 1987–1989, there were 281 such deaths. As it is not clear from KMR1 records whether NMOD was the cause of death or an incidental diagnosis, we requested the case notes of these patients from their individual hospitals. Written approval was sought from consultants who had attended the patients during their final illness. Notes obtained were examined by two reviewers (MZP and RW) independently. All relevant clinical, radiological, histological, and postmortem details were recorded. The cause of death was established using an agreed proforma based on these details. In cases of uncertainty or disagreement, a final decision was taken after discussion between the two reviewers. To assess the accuracy of death certification for NMOD we compared diagnoses on death certificates with the results of case note review in cases from four West Midlands hospitals (selected for ease of access and large catchment population).

Results

Death

Between 1969 and 1991 in men, the NMOD

death rate rose from 5.5 to 10 per million and in women it nearly trebled from 5 to 13 per million (Figure (A)). The increase was due to a rise in deaths in those over 75 years (Figure (B) to (D)). Between 1974 and 1988 mortality (all ages) from functional disorders (ICD 530.0) remained unchanged at 1 per million for both sexes, while for all other causes (ICD 530.9, 530.1 to 530.4) there was a rise from 6 to 12 per million. In both sexes, between 1979 and 1988 there was a rise in deaths from oesophageal ulcer (ICD 530.2) from 1.5 to 2.5 per million, and deaths due to oesophagitis (ICD 530.1) rose from 0.75 to 1.5 per million. Death rates from oesophageal stricture (ICD 530.3) almost doubled in women from 3.5 to 6 per million and rose only slightly in men from 2.5 to 3 per million. Mortality from perforation of the oesophagus (ICD 530.4) did not change in men or women. Death rates in persons aged 35–65 years remained unchanged (between 1–6 per million, rising with age) between 1968–91. Below age 35 years, deaths due to NMOD remained negligible. Age standardised mortality from NMOD rose from 2.9 to 7 deaths per million men and from 5.2 to 13.1 deaths per million women (Table II). The elderly male population (>75 years) increased from 706 900 in 1968 to 1 249 100 in 1991 (3.0 to 5% of total male population). Over the same period the number of women over 75 years increased from 1 493 000 to 2 356 500 (6 to 9% of total female population).^{5 6}

TABLE II Total population, age specific death rate, and age standardised rate for deaths from NMOD

Year	Total population (million)	Age specific death rate (deaths/million)					Age standardised rate (deaths/million)
		<45	45-64	65-74	75-84	85 plus	
<i>Male</i>							
1968	23-6299	0.4	2	2	63	138	2.9
1969	23-7520	1.2	2	2	49	108	3.1
1970	23-8309	1.3	3	3	56	127	3.7
1971	23-7196	0.1	5	5	71	110	4.5
1972	23-8379	1.5	3	3	59	167	4.2
1973	23-9158	1.5	3	3	55	151	4.1
1974	23-9410	1.3	4	4	76	158	4.9
1975	23-9679	1.3	4	4	69	235	5.2
1976	23-9533	0.6	3	3	72	195	3.8
1977	23-9226	0.5	3	3	69	188	4.2
1978	23-9136	0.5	2	2	88	203	4.6
1979	23-9501	0.5	4	4	78	176	4.1
1980	23-9848	0.9	4	4	97	188	6.0
1981	24-1207	0.6	4	4	89	271	6.1
1982	24-1454	0.4	3	3	80	177	4.9
1983	24-1759	0.8	2	2	80	151	4.8
1984	24-2442	0.6	1	1	92	233	5.4
1985	24-3300	0.3	3	3	99	287	6.6
1986	24-4035	0.4	3	3	123	327	8.0
1987	24-4928	0.6	3	3	103	222	6.9
1988	24-5760	0.3	3	3	103	298	7.4
1989	24-6691	0.8	3	3	84	269	6.9
1990	24-7656	0.6	3	3	94	296	7.6
1991	24-8915	0.95	1	1	98	233	7.0
<i>Female</i>							
1968	24-9631	0.4	2	19	36	87	5.2
1969	24-0748	0.9	2	17	44	81	5.7
1970	25-1568	1.3	3	13	42	72	5.7
1971	25-0954	0.9	5	15	36	96	6.1
1972	25-1910	0.9	3	19	47	116	6.9
1973	25-2588	0.3	3	16	39	101	5.8
1974	25-2541	0.7	4	18	48	143	7.7
1975	25-2508	0.3	4	18	61	86	7.4
1976	25-2311	0.4	2	20	52	120	7.3
1977	25-1969	0.2	3	18	55	113	7.4
1978	25-2037	0.4	2	16	56	144	7.7
1979	25-2207	0.2	4	25	60	182	9.8
1980	25-2595	0.5	4	22	55	151	9.1
1981	25-1472	0.5	4	20	59	146	9.4
1982	25-4614	0.1	3	18	61	178	9.4
1983	25-4778	0.3	2	19	62	184	9.6
1984	25-5194	0.5	1	20	65	233	11.0
1985	25-5935	0.3	3	23	71	271	12.6
1986	25-6719	0.1	3	28	76	258	13.4
1987	25-7501	0.1	3	24	72	254	13.0
1988	25-8173	0.3	3	28	73	296	14.6
1989	25-8934	0.3	3	22	85	219	13.4
1990	25-9532	0.2	3	20	69	214	12.2
1991	26-0633	0.5	1	23	77	225	13.1

Review of case notes and death certificates

Records of the West Midlands Regional Health Authority showed that a diagnosis of NMOD (ICD 530) was included on the KMR1 of 281 West Midland residents who had died in hospital between 1987-1989. Permission to review the case notes was withheld, or notes were unavailable in a large proportion. Nevertheless the notes of 84 of these deaths were located and reviewed. On review, NMOD was believed to have been the underlying cause of death in 28 of 84 deaths (33%). In three of 28 cases the reviewers could not be confident that NMOD was the underlying cause of death, and these cases were therefore labelled as 'possible'. The most common conditions that led directly to death in the 28 cases were pneumonia (due to aspiration, 15 cases) and sepsis secondary to mediastinitis from oesophageal perforation (nine cases), cardiac arrest (two cases), and bleeding (two cases). Perforation of the oesophagus was spontaneous in three cases, due to rigid oesophagoscopy with dilatation in three, and secondary to diagnostic fiberoptic upper gastrointestinal endoscopy in three. A further endoscopic complication arose from misplacement of an oesophageal prosthesis at rigid oesophagoscopy, which necessitated laparotomy and was followed by cardiac arrest.

Therefore, complications attributable to endoscopic procedures totalled seven of 28 (25%). These cases include the three 'possible' deaths due to NMOD that were attributable to an endoscopic procedure.

Accuracy of death certificates

Sixty four of 84 deaths where case notes were available had been treated in four hospitals. Death certificates were traced in 62 cases. Our assessment of the validity of death certification relates to the 62 cases for whom both the case notes and the death certificates were available. On case notes review, it was possible to decide confidently whether the cause of death was NMOD in 60 of 62 cases. In two cases NMOD was considered to be only a possible cause of death. Table III shows results assuming the cause of death in these two 'possible' cases was NMOD. Death certificates underestimated NMOD as the cause of death by about 50%. There were no instances of malignant (oesophageal or other) disease having been wrongly identified on death certificates as NMOD.

Discussion

This analysis shows that the recent rise in deaths from benign oesophageal disease seen in England and Wales is due to an increased death rate in very elderly men and women. The numbers of deaths remain small in comparison with other causes of death, but the time trends are large and deserve explanation. We were initially surprised that so many patients die from NMOD. For the purposes of death certification, however, the underlying cause of death is defined as the condition that initiated the train of events leading to death.⁴ Thus, in most of the patients whose notes were reviewed, where the underlying cause of death was NMOD, the direct cause of death had been sepsis related to either aspiration or intubation.

We considered the possibility that the recorded deaths were misclassified and hence compared case notes with death certificates. Our findings show that death certificates underestimate the frequency of NMOD as the underlying cause of death by as much as 50%. We cannot comment on the accuracy of certification in patients dying in the community.

It is tempting to ascribe the time trends to an ageing population. It seems unlikely that an increase in the average age within five year age bands up to the age of 85 is responsible for the changes seen, because the increase in average

TABLE III Comparison of case note review and death certification as record of NMOD as underlying cause of death

	Notes review		Total
	Caused by NMOD	Not caused by NMOD	
Death caused by NMOD	12	0	12
Certification not caused by NMOD	9	41	50
Total	21	41	62

age between 1971 and 1991 has been minimal while death rates at least doubled. The numbers of men over the age of 75 increased by two thirds (from 3 to 5% of the total male population) and of women by half (from 6 to 9% of the total female population) during the 23 years of study. It is possible to estimate the effect of the ageing population by extrapolating the numbers and proportions of people over the age of 80 using data from the 1961 and 1991 censuses. Such calculations suggest an expected increase in death, attributable simply to an increase in the numbers of people over 85 of about 11% in men and 80% in women. The average age of this group has also changed, which would lead to a further contribution to the increase. These demographic changes have contributed to, but have not completely explained death rate increases, which doubled in men and trebled in women.

There has been no concomitant rise in deaths from NMOD in younger people, so any explanations for these trends must consider why risk is increasing in the oldest group of people. A possibility is that better diagnostic techniques have allowed more accurate diagnosis and thus increased the recording of the underlying cause. It is impossible to exclude this possibility and indeed it is possible that the use of endoscopy in old people has been increasing faster than that in younger patients. Perhaps old patients with oesophageal disorders were labelled with a malignant diagnosis before the endoscopic era, which has permitted more accurate diagnosis. Thus diagnostic transfer may explain some of the rise in deaths. Another possibility is that the disease processes themselves may be becoming more common. Gear and Wilkinson reported a rise in the frequency of diagnosis of oesophagitis from 6 to 18% of open access cases in 1987.¹ There may be many explanations for this change but there may well be a real increase in the disease.

The findings reported here resemble those on duodenal and gastric ulcer with increasing death rates in the elderly, especially in women.^{7,8} One proposed explanation for part of this phenomenon has been the increasing use of non-steroidal anti-inflammatory drugs for which evidence is very strong.⁸⁻¹¹ Evidence that such agents damage the oesophagus is less clear. Twenty per cent of patients receiving longterm indomethacin treatment have been reported to have oesophageal disease including ulcers,¹² a frequency similar to that of gastric erosions. Heller *et al* showed an increased risk of oesophageal stricture in users of non-steroidal anti-inflammatory drugs in a retrospective case control study.¹³ Further information is required but the frequent use of non-steroidal anti-inflammatory drugs in the elderly could also explain some of the increasing morbidity from benign oesophageal disease.

Another worrying possibility is that the use of endoscopy might be provoking the increased numbers of deaths. We attempted to assess the relation of endoscopic procedures to death in the cases reviewed. In the worst case analysis presented, endoscopic procedures (both rigid and flexible) were thought to have precipitated the complications that led to death in about a quarter. We do not know to what extent this proportion may have changed over the past 20 years, but endoscopic therapeutic manoeuvres are being used more frequently and it would be expected that the numbers of complications would have increased accordingly. The frequency of perforation of the oesophagus as the underlying cause of death has not changed over the period of study and this seems reassuring. An endoscopic perforation produced, however, during the diagnosis of severe oesophagitis or during dilatation of benign stricture would not necessarily be recorded as the underlying cause of death should the patient have died. In any event, a substantial proportion of hospital deaths from NMOD seem to be endoscopy related and it is unclear how much of the overall increased death rate this might explain.

Death from benign oesophageal disease is increasing in the elderly and is probably under recorded. The causes for this rise are not clear and their identification is warranted as they may be partly iatrogenic, related to endoscopy and drugs.

- 1 Gear MWL, Wilkinson SP. Open access upper alimentary endoscopy. *Br J Hosp Med* 1989; 4: 438-44.
- 2 Office of Population Censuses and Surveys. *Registrar General's statistical reviews 1968-1973*. Table 17. London: HMSO.
- 3 Office of Population Censuses and Surveys. *Registrar General's statistical reviews 1974-1988*. Table 2. Series DH2 no 1-15. London: HMSO.
- 4 Heasman MA, Lipworth L. *Accuracy of certification of cause of death*. Studies on medical and population subjects. General Register Office 1966; No 20 HMSO: London.
- 5 Office of Population Censuses and Surveys. *Registrar General's statistical reviews 1968-1973*. Table 1. Estimated populations: total home and civilian by sex and age. London: HMSO.
- 6 Office of Population Censuses and Surveys. *Registrar General's statistical reviews 1974-1982*. Table 1. Series DH2 no 1-9. and 1983-1988 Table 1, Series DH1 no 14-21. London: HMSO.
- 7 Walt R, Katschinski B, Logan R, Ashley J, Langman M. Rising frequency of ulcer perforation in elderly people in the United Kingdom. *Lancet* 1986; i: 489-92.
- 8 Holvoet J, Terriere L, Van Hee W, Verbist L, Fierens E, Hautekeete ML. Relation of upper gastrointestinal bleeding to non-steroidal anti-inflammatory drugs and aspirin: a case-control study. *Gut* 1991; 32: 730-4.
- 9 Somerville KW, Faulkner G, Langman MJS. Non-steroidal anti-inflammatory drugs and bleeding peptic ulcer. *Lancet* 1986; i: 462-4.
- 10 Beardon PHG, Brown SV, McDevitt DG. Gastrointestinal events in patients prescribed non-steroidal anti-inflammatory drugs: a controlled study using record linkage in Tayside. *Q J Med* 1989; 71: 497-505.
- 11 Gabriel SE, Jaakkimainen L, Bombardier C. Risk of serious gastrointestinal complications related to use of non-steroidal anti-inflammatory drugs. *Ann Intern Med* 1991; 115: 787-96.
- 12 Arnold JD, Swift GL, Williams GT, Wilkins WE, Morris JE, Rhodes J. Prevalence of oesophagitis in subjects on long term non-steroidal anti-inflammatory drugs. *Gut* 1991; 32: A1214.
- 13 Heller SR, Fellows IW, Ogilvie AL, Atkinson M. Non-steroidal anti-inflammatory drugs and benign oesophageal strictures. *BMJ* 1982; 285: 167-8.