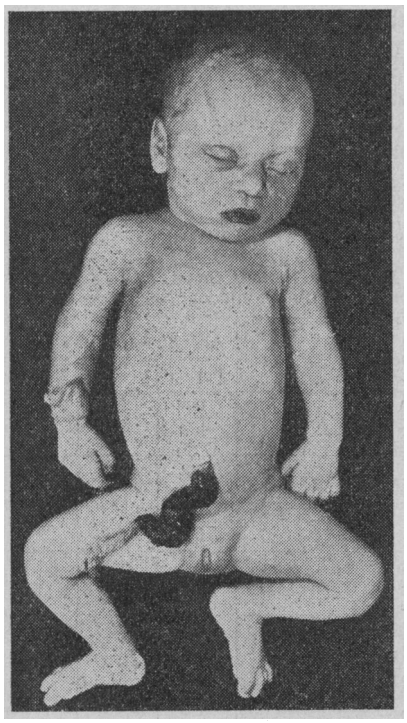
Case 3

A woman aged 29 had an uneventful antenatal period and was admitted in the first stage of labour on her expected date of confinement. Foetal movements had ceased early in labour, and on her admission six and a half hours before delivery no foetal heart was heard. The cervix was dilated three fingerbreadths and no cord was felt. Labour lasted 14 hours and the patient was delivered of a female stillborn infant weighing 6 lb. 12 oz. (3,060 g.). The cord was coiled loosely once round the baby's neck and showed a swelling 5 cm. long near the umbilicus (see Fig.). Again necropsy showed the swelling to be a haematoma associated with rupture of the umbilical vein, and again the aetiology was obscure.



Case 3. Photograph showing swelling of the umbilical cord.

The patient has since had two normal children.

Discussion

The incidence of haematoma of the umbilical cord is approximately 1 in 5,500 deliveries (Toland *et al.*, 1959). Various aetiological factors have been suggested: syphilis, mechanical trauma, anomalies of the umbilical veins or arteries (degenerative or congenital), short cords, cord around the neck, torsion of the cord, and ruptured membranes have all been incriminated. There is, however, no really adequate explanation.

According to Dippel (1940) haematomas of the cord are usually single and arise from the umbilical vein. They vary in size from "1.3 cm. to the size of a child's arm," and, although usually situated near the umbilicus, may be located anywhere along the cord. Browne (1925) remarks that "foetal death is as a rule not due to the amount of haemorrhage but to pressure by the clot upon the umbilical vessels, especially the umbilical vein, or to the formation of a coagulum in the lumen of the latter." While this is probably true for foetal death associated with small haematomata there is little doubt that with larger haemorrhages the foetus may be seriously exsanguinated.

The foetal mortality rate is reported to be 47%, the majority of deaths occurring in the first or second stage of labour.

Summary

Three cases of haematoma of the umbilical cord are described. This rare condition is often associated with foetal death, as in two of these three cases.

I am indebted to Mr. J. A. Price for permission to report the second and third cases, and to Professor J. H. M. Pinkerton for permission to report the first case.

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Preliminary Communications

Assessing the Comparability of Mortality Statistics

Brit. med. J., 1964, **2**, 1437–1439

Large differences exist between the reported death rates from coronary disease and bronchitis of middle-aged men in the United States of America, the United Kingdom, and Norway (Reid, 1960). Mortality from bronchitis is allegedly very much higher in Britain than in the United States, where it is higher than in Norway. Conversely, the death rate for arteriosclerotic heart disease (A.S.H.D.) among middle-aged men in the United States is almost double the British rate, while the Norwegian rate is again the lowest of the three. If these differences are real they may provide important clues to the causes of these two important diseases. It may be, however, that they only reflect international differences in diagnostic habits and in concepts about the classification of the various forms of cardio-respiratory disorder. If this is so, then it is clearly important that the presence of such sources of confusion be recognized before any misleading conjectures about possible causes are based on these reports.

A number of studies have already been undertaken to test the accuracy of death certification in individual cases; for example, by defining the clinical and post-mortem evidence underlying the statement of the cause of death (Swartout and Webster, 1940; Pohlen and Emerson, 1942, 1943; James *et al.*, 1955; Moriyama *et al.*, 1958; Heasman, 1962).

From the point of view of international comparisons, however, even if equal skill in the eliciting of clinical data of all kinds be assumed, there may remain differences between physicians, trained and practising in different countries, in their classification of these findings into diagnostic groups and their habits in certifying the underlying cause of death. Thus the underlying cause of death in a patient dying because of heart failure might be entered as bronchitis in one country but as arteriosclerotic heart disease in another. There is no published evidence on the diagnostic transfers to which such habits might lead. This note gives the results of an inquiry designed to develop and test a method by which future investigation of this problem might proceed.

Method

The case histories of a representative sample of hospital patients whose deaths had been ascribed to various cardio-vascular-renal and respiratory causes were submitted to groups of doctors in three different countries with the request that they "certify" the cause of death according to their usual conventions.

Two London hospitals (one a teaching hospital) were chosen as the source of the cases; between them they provide fairly complete hospital cover for their particular area. The hospitals were visited and the counterfoils of all death certificates issued in 1958 were examined. A note was made of each male death in the age range 40–64 where death had been certified as due to hypertension, cerebrovascular accident, arteriosclerosis, non-valvular heart disease, nephritis, bronchitis, emphysema, or bronchopneumonia (unspecified). A sample of 10 of these deaths was then chosen randomly from this list. The hospital records of these patients were obtained and abstracts were prepared of all the available information thought relevant to the cause of death, including *x*-ray reports, electrocardiograms, and laboratory investigations. Where the original death certificate had been completed (or amended later) in the light of information obtained at necropsy the post-mortem findings were appended.

Since some of these patients were moribund on admission, investigation was often limited and the available clinical information inevitably scanty. In drafting the case histories only factual statements were included, and descriptions which implied particular diagnostic interpretations were avoided. A past history recorded in the original hospital notes as "bronchitis," for example, was changed to one of "recurrent productive cough." Brief versions of the 10 case histories are given in the Appendix.

The full case histories were circulated to 24 doctors in Boston, U.S.A. (through the help of Dr. Joseph Stokes III), to 16 doctors in Bergen, Norway (through the help of Dr. T. Mork), and to 30 doctors in London. The doctors concerned were nearly all working at teaching hospitals. In each sample about half were house-physicians, and the rest were mainly of registrar or equivalent status and engaged in clinical medicine. These grades were chosen because it is upon them that the onus of death certification in hospital patients mainly falls. For each of the 10 cases each doctor was asked to make out a death certificate in the standard international form, just as he would if the patient had been under his care.

The completed returns were then coded according to the *International Statistical Classification of Diseases, Injuries, and Causes of Death*. The coding for all three countries was done centrally by the General Register Office, London, and the 240 certificates from Boston, 160 from Bergen, and 300 from London were tabulated according to the categories used in vital statistical reports.

RESULTS

Table I gives the allocation of causes of death in the same 10 patients by the doctors in the three countries. In Table II these allocations are grouped more broadly. As a background to this information Table III presents the relevant mortality figures for the countries concerned, classified according to the *International Abridged List 1948* (W.H.O., 1959).

The totals for the major groupings are quite consistent between the three national sets of certifiers. Within some of these groups, however, there are suggestive divergencies. In the cardiovascular category, for example, rather more of the deaths were attributed by the American physicians to arteriosclerotic heart disease. This transfer was due in part to the rule whereby coronary disease mentioned in such cases only under section II of the certificate nevertheless takes precedence in the coding; for the American doctors appeared to list under section II all the conditions from which the patient suffered, even if they were not closely linked with death. British doctors, on the other hand, seemed to use the term "bronchitis" more readily than either of the other two groups, who preferred to attribute the corresponding deaths to either emphysema or bronchiectasis. This finding agrees with the impression of a group of American clinicians who had toured British centres (*J. Amer. med. Ass.*, 1960).

As Table III shows, these tendencies are in line with the pattern of prevailing mortality rates from these diseases in the three countries. This would suggest that some at least of the reported differences in mortality from specific causes such as bronchiectasis may be attributed to differences in national habits in disease classification. But the remarkable consistency in the allocation of deaths to major categories implies that such disparities are unlikely to be the whole explanation of the American and British excess in arteriosclerotic and respiratory disease respectively in the age group 45–64. In view of the unrepresentative nature of the samples and the fact that no test has been made of the consistency of coding practices in these countries, such speculation is premature. The method, however, has proved workable and instructive, and an extension of its use might well be profitable.

TABLE I.—*Allocation (%) of Deaths to Various Causes: Comparison of Boston (24 Doctors), London (30 Doctors), and Bergen (16 Doctors)*

International List Code No.	Disease	Allocation (%) of Deaths		
		Boston	London	Bergen
016	Renal tuberculosis	0·8	2·3	1·3
053	Septicaemia	0·4	0·3	
063	Gas gangrene			0·6
180	Renal cancer	0·8	1·3	0·6
199	Malignant neoplasm (unspecified site)	0·4		
241	Asthma	0·4		
252	Thyrotoxicosis	0·8	3·0	2·5
330–1	Cerebral haemorrhage	9·2	12·3	13·1
332	Cerebral thrombosis	8·8	7·0	4·4
334	Other strokes	1·3	0·3	4·4
416	Rheumatic heart disease	0·4		
420	A.S.H.D.	20·4	15·3	13·8
422	Other myocardial degeneration	0·4	0·3	0·6
431	Acute myocarditis (non-rheumatic)	0·4		
433–4, 782–4	Other heart disease	3·3	5·0	5·0
440, 442–3	Hypertension, heart disease +	5·4		4·4
444–7	" " " −	3·8	8·0	6·3
450	General arteriosclerosis	0·4	4·3	2·5
453	Buerger's disease		0·3	0·6
454–6	Other peripheral arterial disease	0·4	2·0	0·6
464–6	Phlebitis and pulmonary embolism	8·3	2·7	1·9
490–3	Pneumonia	1·3	1·3	0·6
500	Acute bronchitis		0·3	
502.0	Chronic bronchitis and emphysema	13·3	18·3	10·6
502.1	Chronic bronchitis	0·4	3·7	3·8
522, 525	Chronic non-specified pneumonia	1·7		
526	Bronchiectasis	2·5	1·3	5·6
527.1	Emphysema (no mention of bronchitis)	4·2	0·7	5·6
527.2	Other lung disease			1·3
541.0	Duodenal ulcer	0·4	0·7	0·6
590–3, 603	Nephritis	10·0	9·0	9·4

TABLE II.—*Allocations Grouped Into Major Categories*

Disease Category	Code Nos.	Allocation (%) of Deaths		
		Boston	London	Bergen
Strokes	330–1, 332, 334	19	20	22
Cardiovascular	416, 420, 422, 431, 433–4, 782–4, 440, 442–3, 444–7, 450, 453, 454–6	35	35	34
Respiratory	241, 490–3, 500, 502.0, 502.1, 522, 525, 526, 527.1, 527.2	24	26	28
Renal	016, 180, 590–3, 603	12	13	11
Other	053, 063, 199, 252, 464–6, 541.0	10	7	6

TABLE III.—*Comparison of Death Rates Per 100,000 From Various Cardiovascular-renal and Respiratory Causes. Males Aged 40–64 in the United States (White), England and Wales, and Norway (1956)*

Category	Cause of Death	U.S.(W)	E. & W.	Norway
B.22	Cerebrovascular accidents	73	89	42
B.26	Arteriosclerotic heart disease	480	286	192
B.27	"Other heart diseases"	17	15	15
B.28	Hypertension with heart disease	32	18	11
B.29	" without " "	5	15	4
B.38	Nephritis	12	14	13
B.22,24–29,38 A.85, 86	All cardiovascular + nephritis	*662*	*474*	*296*
B.31	Pneumonia	20	31	11
B.32	Bronchitis	2	87	2

SUMMARY

A method of studying death-certification habits in different countries is described. The results obtained in a pilot inquiry among hospital physicians in Norway, the United Kingdom, and the United States are discussed in relation to differences between these countries in the reported death rates from bronchitis and arteriosclerotic heart disease.

APPENDIX: CASE SUMMARIES

Case 1.—Aged 50. Cough for 10 years, worse recently and associated with increasing dyspnoea. Physical signs: stupor; cyanosis; jugular venous congestion, oedema; poor chest expansion, diffuse rhonchi, basal fine rales, and bronchial breathing. E.C.G.: pointed P in II. Hb 102%. Died 24 hours later. P.M.: right ventricular hypertrophy, moderate coronary atheroma; pulmonary congestion, oedema, and emphysema; stomach full of altered blood, acute duodenal ulcers.

Case 2.—Aged 64. Incipient gangrene of leg, with absent pulses. E.C.G.: Q and inverted T in II, III. No benefit from iliac thrombarterectomy. Leg amputation followed by gangrene spreading up from stump. No P.M.

Case 3.—Aged 47. Sudden onset of severe central chest pain. History of paroxysmal dyspnoea at age 44, when hypertension and proteinuria were noted. Physical signs: B.P. 150/105; cardiac enlargement. Heavy proteinuria. Blood urea 71 mg./100 ml. E.C.G.: recent infarction. Died 2 weeks later after further attack of pain. P.M.: old and recent infarction, and considerable hypertrophy, of left ventricle; right kidney small, with large area of cortical thinning; left kidney granular.

Case 4.—Aged 64. Productive cough and dyspnoea for years. Attacks in past 4 days of sudden faintness, sweating, and dyspnoea. Physical signs: cyanosis, dyspnoea, clubbing; barrel-shaped chest with many rhonchi. Serum potassium 7.6 mEq/l. Died 2 days later. No P.M.

Case 5.—Aged 42. Sore throat followed 1 month later by oedema; also by hypertension and proteinuria, which persisted. Readmitted 2 years later with heart failure. Hb 41%. Blood urea 216 mg./100 ml. P.M.: bullous emphysema; left ventricular hypertrophy; granular kidneys with thin cortices.

Case 6.—Aged 40. Collapsed at work. Hypertension first noted 3 years previously, when tuberculous kidney was removed. Physical signs: stupor, hemiparesis; B.P. 250/150; papilloedema and retinal exudates. Died in coma. No P.M.

Case 7.—Aged 45. Three-months history of lassitude and dyspnoea. Physical signs: prominent eyes, sweating; auricular fibrillation, cardiac enlargement, jugular venous congestion; basal rales. E.C.G.: fibrillation, T waves all flat. B.M.R.: +55%, +27%, +13%. ^{131}I uptake normal. Given carbimazole; improved for 6 months, then heart failure relapsed. Died suddenly. P.M.: pulmonary embolism and infarction; heart weight 735 g.; some pericardial adhesions.

Case 8.—Aged 51. Recurrent cough and dyspnoea since pneumonia 18 years earlier. Recent pleural pain. Physical signs: fever, cyanosis, dyspnoea; jugular venous congestion and oedema; fixed chest, scattered rhonchi, fine rales over right lower lobe. E.C.G.: tall pointed P in II. Proteinuria ++. Improved for 1 month, then died after sudden collapse with tachycardia and unrecordable blood-pressure. No P.M.

Case 9.—Aged 50. Ten-days history of headache, vertigo, vomiting. Cardiac infarction 5 years previously. Physical signs: stupor, aphasia, left facial weakness and hemianaesthesia; B.P. 165/115. Died in coma. P.M.: old and recent cardiac infarction; recent pontine infarction.

Case 10.—Aged 57. Sudden onset of headache and loss of speech. Proteinuria and hypertension discovered at age 51. Nephrectomy for renal carcinoma at age 56. Hemiplegia at age 56. Physical signs: dysphasia, confusion, hemiparesis; B.P. 200/140; left ventricular enlargement. Urine: protein ++. Died in coma. No P.M.

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Antimalarial Effect of Sulphorthodimethoxine (Fanasil)

Brit. med. J., 1964, **2**, 1439–1440

The new long-acting sulphonamide sulphorthodimethoxine[1] (Fanasil) was used in a preliminary trial to test the potentiating effect of sulphonamide with pyrimethamine (Hurly, 1959; McGregor, Williams, and Goodwin, 1963) against pyrimethamine-resistant malaria. The drug is closely related to sulphadimethoxine[2] (Madribon), of which it is an isomer, but the rearrangement of the molecule gives it different properties. These include a more prolonged antibacterial action, a dose of 1 g. weekly being therapeutically equivalent to 1 g. daily of sulphadimethoxine or other long-acting sulphonamides such as sulphamethoxypyridazine[3] (Lederkyn, Midicel) or sulphaphenazole[4] (Orisul). Sulphorthodimethoxine was chosen because of this sustained concentration in the plasma, so matching better than other sulphonamides the persistent plasma concentration of pyrimethamine.

The work was done among schoolchildren in the Muheza district of Tanga, north-east Tanganyika, where malaria is holoendemic and the children have a high degree of acquired immunity (asexual parasite rates of 60–80% are the rule). Pyrimethamine-resistant falciparum malaria was found in this and adjacent areas by Clyde and Shute (1957), and recent surveys have shown that it is still present.

Prior to giving sulphorthodimethoxine, all children were given 25 mg. of pyrimethamine (the recommended adult dose) weekly for two months to eliminate drug-sensitive parasites. A survey done at the end of this period showed an asexual parasite rate of 27% (all *Plasmodium falciparum*). Sulphorthodimethoxine in 500-mg. doses was then given weekly, together with pyrimethamine at the same dosage as before, to one group of 191 children; another similar group (208 children) was given 500-mg. doses of the sulphonamide alone, while a third group (192 children) continued with pyrimethamine as before. A blood survey after six weeks showed that asexual parasitaemia