fully descended during infancy but where orchidopexies had been done after referral as a result of school medical examinations. One of these boys had had two operations for hypospadias and the other, a premature infant, was followed up by one of us (TML) for two years.

Comment

It is generally agreed that surgery for undescended testis should be performed by not later than 5 years of age. All but one of our consultant colleagues agreed with this, as did 75% of the general practitioners, yet only 32% of operations were done by the recommended age. Why does this happen and what does it mean?

The incidence of undescended testis after the age of 1 year is about 0·8%.[1 2] Between infancy and puberty, however, retractility of the testis may make diagnosis difficult; one survey found that 6% of boys between the ages of 5 and 11 appeared to have undescended testes but by the age of 14 this figure had fallen to 0·6%.[3] A paediatric surgeon mentioned this problem and said that he was reluctant to operate on boys in whom the diagnosis of undescended testis had not been documented during infancy.

Chilvers *et al* studied the increasing orchidopexy rate over the past 20 years and suggested that surgery on boys with retractile testes could explain the increase.[4] We have documented two cases where this occurred and believe this explanation to be correct. Hence about 10 000 unnecessary operations—75% of the total—are performed in England and Wales every year; these cause considerable discomfort, endanger fertility, and, with an average stay in hospital of 4·4 days, cost about £4m.[5]

This unsatisfactory state of affairs could be avoided by two measures. Firstly, neonates with undescended testis should be followed up by a consultant paediatrician for one year; if the testis remains undescended the boy should be referred directly to a surgeon. Secondly, an older boy thought to have an undescended testis and for whom no infant record is available should be examined by an experienced surgeon—if necessary under general anaesthesia—so that retractile testes can be identified.

We thank our colleagues who collected data for our survey and also D I Beeby, P G Girolami, R W Hoile, D G Jenkins, and P J Jennings for permission to study patients under their care. We are also grateful to Mrs T Redknap for secretarial help.

1 Scorer CG. The descent of the testis. *Arch Dis Child* 1964;**39**:605-9.
2 Baumrucker GO. Incidence of testicular pathology. *Bulletin of the US Army Medical Department* 1946;**5**:312-4.
3 Ward B, Hunter WM. The absent testicle. *Br Med J* 1960;i:1110-1.
4 Chilvers C, Pike MC, Forman D, Fogelman K, Wadsworth MEJ. Apparent doubling of frequency of undescended testis in England and Wales 1962-81. *Lancet* 1984;ii:330-2.
5 Department of Health and Social Security, Office of Population Censuses and Surveys. *Hospital inpatient enquiry*. London: HMSO, 1981. (Series MB4 No 18.)

(Accepted 2 July 1985)

All Saints' Hospital, Chatham, Kent ME4 5NG
B J COOPER, MRCP, paediatric registrar
T M LITTLE, FRCP, consultant paediatrician

Correspondence to: Dr Little.

Haemophilus parainfluenzae and H influenzae respiratory infections: comparison of clinical features

Haemophilus influenzae, although usually non-capsulated in the respiratory tract of adults,[1] is regarded as a possible pathogen when so isolated.[2] The role of *H parainfluenzae* is less clear and, despite causing invasive infection,[3] its effect as a pathogen has been doubted.[4] We report a study of 981 sputum isolates of haemophilus and relate the respiratory disease, clinical features, and antibiotic sensitivity to the organism isolated.

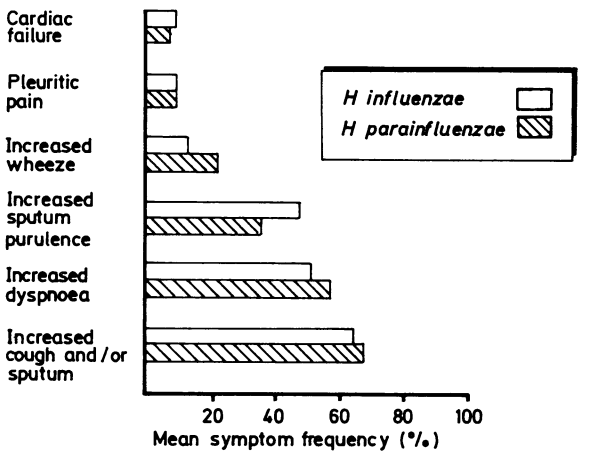
Materials, methods, and results

Using laboratory records to identify patients who had haemophilus isolated from the respiratory tract, we conducted a retrospective study of patients' case notes to record the clinical features at the time the specimen was produced. We also recorded bacteriological features of the isolate. Specimens were inoculated on to horse blood agar and heated horse blood agar containing 10 units bacitracin/ml and incubated for 18 hours at 37°C in 7% carbon dioxide. Non-haemolytic haemophilus colonies were identified by typical appearance and the organism confirmed by Gram stain and satellitism with *Staphylococcus aureus*. Differentiation between *H influenzae* and *H parainfluenzae* was achieved by their ability (*H parainfluenzae*) or failure (*H influenzae*) to metabolise δ-aminolaevulinic acid.

The monthly frequency of isolations of *H influenzae* and *H parainfluenzae*, the underlying diagnosis and presenting symptoms, the antibiotic sensitivities, and the frequency of mixed growths were recorded and compared using χ^2 statistical analysis.

There were 689 sputum isolates of *H influenzae* and 292 of *H parainfluenzae*; *H influenzae* had its peak in March and *H parainfluenzae* in November. The patients were aged 12-98 years (mean 60 (SD 18) years).

The most frequently recorded symptoms associated with isolation of haemophilus were cough (with or without sputum production), dyspnoea, increased sputum purulence, and wheeze, and these varied little among the diagnostic groups of chronic bronchitis, asthma, pneumonia, bronchial carcinoma, and bronchiectasis. There was no difference in the symptoms produced by *H influenzae* or *H parainfluenzae* in these groups or in total (figure). In patients with chronic bronchitis 35% of *H influenzae* and 37% of *H parainfluenzae* isolates were obtained when there was no clinical evidence of exacerbation of the disease (NS).



Mean frequency of symptoms recorded for all biotyped hospital episodes. (No of episodes with symptom recorded/total No of episodes × 100%.)

Other organisms were isolated from 20% of specimens yielding *H influenzae* and 13% of those yielding *H parainfluenzae*, but this difference was not significant ($0·1 > p > 0·05$). Mixed growths were most common in bronchial carcinoma ($p < 0·001$); and in patients with pneumonia, 10 out of 38 had mixed growths, seven with *Streptococcus pneumoniae*, all but one of the serotypes (type 33) having been recorded in association with pneumonia in this hospital.[5]

Antibiotic sensitivities were determined in 954 isolates. Ampicillin resistance occurred in 9% of *H influenzae* (60% of these producing β-lactamase) and 7·5% of *H parainfluenzae* (58% β-lactamase producers). Fourteen per cent of *H influenzae* isolates were resistant to tetracyline, 1·5% to co-trimoxazole, and 0·3% to chloramphenicol, the corresponding figures for *H parainfluenzae* being 17%, 1·5%, and 0·4%.

Comment

We have shown that *H parainfluenzae* is associated with the same clinical spectrum of illness as *H influenzae* and that symptoms produced, occurrence in mixed growths, and the antibiotic sensitivities are the same for the two organisms. It is difficult to define an exacerbation in chronic bronchitis, as sputum purulence may not be a factor, and we relied on clinical impression at the time of presentation; again there was no difference in presentation whether *H influenzae* or *H parainfluenzae* was isolated. Whether haemophilus initiates infection or is merely a secondary infection is uncertain but there is no evidence that *H influenzae* and *H parainfluenzae* are different in this regard. In patients with pneumonia haemophilus may not have been solely responsible in all cases, since in seven *Str pneumoniae* was isolated.

H parainfluenzae and non-capsulated *H influenzae* are clinically indistinguishable; hence we suggest that *H parainfluenzae* should be reported by bacteriology departments and be considered pathogenic in a patient who has evidence of a respiratory tract infection if the organism is obtained in the presence of pus cells and in pure growth from the respiratory tract.

1 Holdaway MD, Turk DC. Capsulated Haemophilus influenzae and respiratory tract disease. *Lancet* 1967;i:358-60.
2 Turk DC. The pathogenicity of Haemophilus influenzae. *J Med Microbiol* 1984; **18**:1-16.
3 Reddy CM, Rao KW, Thomas FE, Anderson ER. Haemophilus parainfluenzae bacteremia with meningitis. *NC Med J* 1978;**39**:165-6.
4 Smith CB, Golden CA, Kanner RE, Renzetti AD. Haemophilus influenzae and Haemophilus parainfluenzae in chronic obstructive pulmonary disease. *Lancet* 1976;i:1253-5.
5 Morgan AD, Rhind GB, Connaughton JJ, Calder MA. Pneumococcal serotyping and antigen detection in pneumococcal pneumonia of adults. *Journal of Infection* 1984;**9**:134-8.

(*Accepted 2 July 1985*)

Departments of Respiratory Medicine and Bacteriology, City Hospital, Edinburgh EH10 5SB
GEORGE B RHIND, MRCP, research fellow in respiratory medicine
GRAHAME A GOULD, MRCP, research fellow in respiratory medicine
FAREEDUDDIN AHMAD, MB, MSC, registrar in bacteriology
MICHAEL J CROUGHAN, FIMLS, senior chief medical laboratory scientific officer, department of bacteriology
MARGARET A CALDER, MD, consultant bacteriologist

Correspondence to: Dr Rhind.

Effect of coroners' rules on death certification for alcoholic liver disease

In England and Wales the coroner is required by law to inquire into violent or unnatural deaths, including those resulting from poisoning. Until July 1984, when the coroners' rules were amended, chronic alcoholism was considered in this category and was a proper verdict at a coroner's inquest.

We were interested to examine the effect of this legislation on the accuracy of death certification in patients dying with alcoholic liver disease.

Methods and results

This retrospective study included all 43 patients who had initially presented to a general medical firm between 1976 and 1984 in whom investigations had established an unequivocal diagnosis of either alcoholic liver disease (21 cases) or non-alcoholic liver disease (22) and who were known to have subsequently died (26 at St George's Hospital, five at home, and 12 at other hospitals in south east England). Clinical diagnosis was compared with the information recorded on the death certificate.

Both routine and forensic necropsies were performed more often in patients dying with alcoholic liver disease than non-alcoholic liver disease (table). Forensic necropsy was followed by a coroner's inquest more often in patients with alcoholic liver disease. Despite the very high necropsy rate in patients with alcoholic liver disease, information on the death certificate was much more likely to be incomplete or misleading. In only seven of the 21 cases was alcoholic liver disease recorded (all the subject of a forensic necropsy and coroner's inquest), and nine had no mention of either liver disease or alcohol. In the remaining five cases various liver diagnoses were entered, including cirrhosis, micronodular cirrhosis, and nutritional cirrhosis.

By contrast, 18 of the 22 patients with non-alcoholic liver disease had a correct liver diagnosis and only three had no record of liver disease. In one patient the clinical diagnosis (sclerosing cholangitis) was found to be incorrect at necropsy (primary biliary cirrhosis) but the death certificate had not been amended.

Comment

Alcoholic liver disease is a main cause of illness and death in many industrial countries.[1 2] Official statistics suggest that this epidemic has bypassed England and Wales, since cirrhosis is certified in only 0·3% of deaths and alcohol is implicated in under one third of these.[2] Clinical experience suggests that this is a considerable underestimate.[1]

Inaccurate death certification has been attributed partly to the decline in frequency of postmortem examinations (now performed in under 30% of hospital deaths),[3 4] but in this study death certification in patients with alcoholic liver disease was inaccurate or incomplete despite a necropsy rate of over 90%. In only one third of deaths in patients with irreversible alcoholic liver disease was this diagnosis certified. By contrast, deaths associated with non-alcoholic liver diseases were accurately recorded.

The main factor responsible for this inaccuracy appears to have been legislation (in England and Wales) requiring alcohol related deaths reported to the coroner to be the subject of a public inquest, often with distressing publicity for relatives. Our experience suggests that forensic pathologists often make a non-controversial diagnosis such as "myocardial ischaemia." In other cases clinicians omit to inform the coroner; instead a hospital necropsy is performed and the findings recorded euphemistically as "nutritional cirrhosis" or simply "cirrhosis," to avoid an inquest.

Official statistics for the United Kingdom suggest that while consumption of alcohol and deaths from cirrhosis have doubled over the past 20 years, deaths due to alcohol still account for only one third.[2] If the increased mortality from cirrhosis is related to increased alcohol consumption the present proportion attributable to alcohol might be expected to be at least 50%. Our findings emphasise the problems in interpreting national statistics on alcohol related mortality and consequently the difficulty in making valid international comparisons. The recent amendment of the coroners' rules to exclude deaths due to chronic alcoholism is welcome and should encourage more accurate death certification, as the necessity for an inquest has now been removed.

Comparison of alcoholic and non-alcoholic liver disease: mode of death, necropsy, inquest, and accuracy of death certification

	Cases of alcoholic liver disease (n = 21)	Cases of non-alcoholic liver disease (n = 22)	Statistical significance (χ^2 test with Yates's correction)
Men/women	14/7	15/7	
Mean age at death (years)	60·5	60·6	
Liver biopsy (cirrhosis)	20 (19)	22 (10)	
Liver disease as direct cause of death	Hepatic failure (10); variceal bleeding (5); hepatoma (2)	Hepatoma (7); hepatic metastases (7); primary biliary cirrhosis (4); chronic active hepatitis (2); fulminant hepatic failure (2)	
Other immediate cause of death	Bronchopneumonia (1); miliary tubercle (1); asphyxia/burns (1); carcinoma pancreas (1)		
Necropsy	20/21	7/22	$p < 0·001$
Forensic necropsy	14/21	5/22	$p < 0·01$
Coroner's inquest	7/21	1/22	$p < 0·05$
Correct liver diagnosis on death certificate	7/21	18/22	$p < 0·01$
No mention of liver disease on certificate	9/21	3/22	$p = 0·07$

We are grateful for the help of Professor M J Davies and the Office of Population Censuses and Surveys.

1 Saunders JB. Alcoholic liver disease in the 1980s. *Br Med J* 1983;**287**:1819-20.
2 Morgan MY. Alcohol as a cause of liver disease in the United Kingdom. In: Hall P, ed. *Alcoholic liver disease: pathology, epidemiology and clinical aspects*. London: Edward Arnold, 1985:208-25.
3 Duffy GJ, Dean G. The reliability of death certification of cirrhosis. *Journal of the Irish Medical Association* 1971;**64**:393-7.
4 McGoogan E. The autopsy and clinical diagnosis. *J R Coll Physicians Lond* 1984;**18**:240-3.

(*Accepted 26 June 1985*)

Department of Medicine II, St George's Hospital Medical School, London SW17
J D MAXWELL, MD, FRCP, consultant physician

Westminster Coroner's Court, London SW1
PAUL KNAPMAN, MB, DMJ, barrister

Correspondence to: Dr Maxwell.