Letters to the Editor

Death during prostatectomy

In a recent report from this hospital,¹ three cases, one fatal, were described, of cardiovascular collapse due to absorption of irrigation fluid, occurring during transurethral resection of the prostate. However, the implications for pathologists conducting necropsies in such cases were not emphasised. Since then a further possible fatal case has occurred, as well as several "near misses" that were successfully treated. We now report the necropsy findings in these two fatal cases and stress the role of the pathologist in identifying the cause of death in such cases.

The first case, as described in the original paper, was a man aged 76 who collapsed and died 30 minutes after the start of a routine transurethral resection of prostate (TUR). The anaesthetist, who had diagnosed and successfully treated other cases, suspected the diagnosis and took a blood sample for estimation of the serum Na^+ , during attempted resuscitation. This revealed a concentration of 86 mmol/l. At necropsy, there was evidence of pulmonary oedema and congestion and small effusions in all the body cavities. There was no evidence of significant cardiac disease, and the remainder of the necropsy was essentially negative. Further confirmation of absorption of irrigating fluid was obtained by estimation of the glycine concentration in the same blood sample as was used for the Na^+ estimation. A level of 80 mmol/l (600 mg/100 ml) was reported. The concentration of glycine in the irrigating fluid is 1.5 g/100 ml of water. Glycine, a low molecular weight amino acid, is presumably distributed throughout the extracellular fluid, given time. However since death can occur rapidly, the degree of equilibration at the time of collapse or death is difficult to assess. Despite these limitations, one can calculate that at least three litres and probably more, was absorbed.

The second patient was a man aged 77 who was admitted to a medical ward for investigation of drop attacks. Two days after admission he developed acute retention. Cardiological studies revealed evidence of ischaemic heart disease, and a systolic murmur was also apparent. A prostatectomy three years earlier had been uneventful. Two weeks after admission, a TUR was performed. However, 10 minutes after the start of the procedure bradycardia developed followed by asystole, and resuscitation was unsuccessful. At necropsy, there was evidence of severe pulmonary oedema (right lung 850 g; left lung 770 g). The heart (430 g) showed evidence of left ventricular hypertrophy, patchy myocardial fibrosis and severe calcific atheroma involving particularly the right coronary and left circumflex branches. In addition, there was evidence of aortic stenosis due to degenerative calcification of a congenitally bicuspid valve. These findings were thought to be sufficient to explain death. However a sample of blood taken at necropsy showed a serum Na^+ of 123 mmol/l despite an infusion of 200 ml of 8.4% sodium bicarbonate administered by the anaesthetist, who suspected hyponatraemia.

The interpretation of the results in this second case is much more difficult due to two factors. The necropsy was performed 65 hours after death, and there is evidence that Na^+ concentrations fall after death, although there is considerable individual variation in the rate of fall.² In addition, the patient was given 200 ml of 8.4% bicarbonate during resuscitation. In retrospect, we should have estimated the Na^+ level in vitreous humour, since the concentrations at this site are much more stable post mortem,³ and estimated the blood glycine. Only then would we have been able to assess the degree of absorption. The amount of fluid absorbed and the degree of hyponatraemia was undoubtedly much less than in the first case, but with significant cardiac disease, it seems reasonable that lesser degrees may be fatal.

In conclusion, the message seems clear. When conducting a necropsy on a man who has collapsed and died during or soon after a prostatectomy, the possibility of absorption of the irrigation fluid should always be considered, and steps taken to confirm or refute the diagnosis. If the anaesthetist has taken a blood sample during the collapse and attempted resuscitation, one should estimate serum Na^+ and glycine concentrations from this specimen. If the only material available is that obtained at the time of necropsy, a sample of vitreous should be taken for Na^+ estimation and blood sent for glycine estimation. In our opinion, it appears likely that this condition is under-diagnosed, largely due to pathologists being unaware of it. Certainly since it has

been recognised by the urologists and particularly the anaesthetists, more cases are being diagnosed, although fortunately the majority are not fatal.

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Resistance of urine pathogens to trimethoprim in Finland

Since 1973, trimethoprim has been recommended to be used as the sole antimicrobial drug for the prophylactic treatment of chronic and recurring urinary tract infection (UTI) in Finland.¹ Unfortunately, there is no baseline against which to compare the present findings of trimethoprim resistance² and no sufficiently extensive surveys have so far been published. The epidemiological information would also be extremely useful in other countries which start using trimethoprim alone for prophylactic treatment of UTI. We therefore analysed consecutive urine isolates of *E. coli*, *Klebsiella* sp and *Proteus* sp at Rovaniemi Public Health Laboratory, the Clinical Laboratory of Savonlinna Central Hospital and the Department of Medical Microbiology, University of Oulu. Samples analysed at the first two laboratories were obtained from the Central Hospital and its sur-

roundings, including treatment centres for the chronically ill. Samples analysed at the third laboratory were from the University Hospital with 1100 beds. The sampling area represented half of Finland; the samples were collected in March-May, 1981.

The bacteria were identified using routine microbial methods, including the API 20E procedure.³ The sensitivity of the bacteria was determined using sensitivity test discs (A/S Rosco, Denmark). The sensitivity test disc for trimethoprim contained 5.2 µg trimethoprim.²

Table 1 shows that the resistance of the bacteria to trimethoprim varied as follows: *E coli* 5-7%; *Klebsiella* sp 18-40%; *Proteus* sp 59-100%. Table 2 shows that the resistance to sulphatrimethoprim was lower than to trimethoprim alone. The resistance of the three pathogens to other antimicrobial agents was as follows (*E coli*; *Klebsiella* sp; *Proteus* sp): to nitrofurantoin 1-7%; 28-42%; 61-100%; to ampicillin 12-27%; 67-94%; 31-100%; and to cephalosporins 3-21%; 23-33%; 16-100%.

The results show that the resistance of the bacteria to all the antimicrobial agents

including trimethoprim varied at the three laboratories. In most cases the resistance rate was highest in Oulu. This reflects the problems of a university hospital without its own specialist microbiologists, but which treats patients with chronic UTI. Our results indicate that the increasing number of bacteria resistant to trimethoprim in Turku⁴ is a local problem and may be due to the intensive use of trimethoprim alone in the prophylaxis of UTI. In other parts of Finland trimethoprim is a safe alternative in the prophylaxis of UTI in open care, but it should not be used in institutions for the chronically ill.

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Table 1 Sensitivity of urine pathogens to trimethoprim in different parts of Finland

<i>No of urine pathogens</i>		<i>Sensitivity of pathogens (%)</i>			<i>Source of pathogens</i>
		<i>S</i>	<i>I</i>	<i>R</i>	
<i>E coli</i>	624	93	2	5	Savonlinna
	604	95	0	5	Rovaniemi
	200	91	2	7	Oulu
<i>Klebsiella</i> sp	69	73	9	18	Savonlinna
	65	60	0	40	Oulu
<i>Proteus</i> sp	49	29	8	63	Savonlinna
	70	41	0	59	Rovaniemi
	42	0	0	100	Oulu

S = Sensitive.

I = Insensitive.

R = Resistant.

Table 2 Sensitivity of urine pathogens to sulphatrimethoprim in different parts of Finland

No of urine pathogens		Sensitivity of pathogens (%)			Source of pathogens
		S	I	R	
<i>E coli</i>	624	93	3	4	Savonlinna
	604	96	1	3	Rovaniemi
	200	94	5	1	Oulu
<i>Klebsiella</i> sp	69	71	9	20	Savonlinna
	65	76	2	22	Oulu
<i>Proteus</i> sp	49	45	4	51	Savonlinna
	70	51	2	47	Rovaniemi
	42	4	0	96	Oulu

S = Sensitive.

I = Insensitive.

R = Resistant.

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- ² Hamilton-Miller JMT, Gooding A, Brumfit W. Resistance to trimethoprim in 1978-79 compared with 1973-1975. *Clin Pathol* 1981;34:439-42.
- ³ Apparcils et procédures d'identification imprimérie nouvelle, 01300 Belley France.
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Growth of *Neisseria Gonorrhoeae* at 25°C

In a previous report¹ it was recorded that contrary to general teaching, isolates of *Neisseria gonorrhoeae* grew well at a temperature below 30°C. The organisms yielded heavy growth in a semi-solid medium incubated at 28°C but no growth was observed at 24°C. The observations made at this lower temperature (24°C) however, were rather cursory and the tubes were observed for only 3-4 days as a general control for contamination and the inadvertent inclusion of *Neisseria* species which grow at temperatures at or below this level. Also it was not imagined that gonococci would grow at temperatures close to such a level but now it has been shown that many isolates do indeed grow slowly but profusely at a temperature of 25°C.

Thirty-five gonococcal strains were tested, several freshly isolated and the remainder from stored desiccates. Inocula for the tests were obtained from cultures grown in semi-solid agar incubated at 37°C.

The organisms were cultured on three media: semi-solid agar;¹ chocolate agar slopes; chocolate agar plates. Incubation was carried out in various constant temperature baths and cabinets. Slopes and semi-solid media were incubated in water baths (23°C, 25°C and 28°C) and the plates in cabinets (25°C and 28°C). All monitoring thermometers were calibrated against standard instruments. Plates were incubated in sealed containers but with no added carbon dioxide.

All isolates grew on plates, slopes and semi-solid media at 28°C, heavy growth being evident after 2-3 days. At 25°C,