Letters to the Editor

Infection and infectious diseases

Sir,

Dr Critchley's criticism¹ of my 'condensed review'² contains errors when he states that it contained statements that could be misconstrued and do not reflect experience gained from three recent outbreaks. He quotes three references. The first³ refers to his analysis of 27 patients with one type of botulism seen by a number of physicians in a small area of one small country. The second and third references^{4,5} each refer to outbreaks in one patient. The world is larger than one country and writing for a journal that has an international readership demands a more than parochial view. My review started by mentioning 'his' outbreak but it is obvious that I thereafter took a broader look at botulism.

In individual outbreaks symptoms differ. I said 'symptoms of gastroenteritis are usually notable by their absence.' Christie⁶ states 'unlike other forms of food poisoning, botulism produces almost no signs of gastrointestinal irritation' and this world view is held by others 'there may be little in the earliest symptoms that suggests foodborne disease.'⁷

Critchley objects to my comment that patients might be found dead. Whilst all patients survived and no unexplained deaths occurred in the outbreak described by Critchley, in areas of the world other than Britain the mortality rate may be higher. Schaffner⁷ gives a figure of 25% given antitoxin, and intensive respiratory support – all of which may not be available in developing countries where the incidence and mortality of botulism would be higher. Whole families of Eskimos have been found dead⁶ and in infant botulism *Cl. botulinum* or its toxin were found in 10/280 of unexpected sudden infant deaths in one study⁸ and in 9/70 in another.⁹

Critchley states correctly that the presence of fever or drowsiness does not exclude botulism. However, the presence of *anything* does not exclude *any* diagnosis. Schaffner states 'patients are *afebrile*' (his italics) and 'development of fever signifies complicating nosocomial infection.'⁷ Critchley states 'antibiotics are best reserved for secondary complications except in the presence of infantile or other toxico-infective forms of botulism' – so his recommendation is that antibiotics are *not* to be given. However, I said that 'some physicians give penicillin to eliminate gut carriage.' Opinions do differ – no one knows – so that the proper question that should be asked is, in our current state of uncertainty, 'Would prescribing antibiotics be likely to do more good than harm?'

I said that treatment includes urgent administration of antitoxin. Critchley states, correctly, that antitoxin treatment is only of *proven* (my italics) value in type E intoxication. Reading the literature it is apparent that antitoxin might be beneficial in other types – but how does a clinician know which type he is dealing with unless there is, at the time when individual patients present, an obvious outbreak with rapid accurate clinical and botulinum type diagnosis? Indeed, in the one well described and recent outbreak, botulism caused by type B toxin, all new patients and those whose condition had yet to stabilize received toxin.³ If I had botulism, on balance, I

would want antitoxin without waiting for type identification.

P.D. Welsby

Department of Infectious Diseases,
City Hospital,
51 Greenbank Drive,
Edinburgh EH10 5SB, UK.

References

1. Critchley, E.M.R. Botulism. *Postgrad Med J* 1991, 67: 401.
2. Welsby, P.D. Reviews in Medicine. Infection and infectious diseases. *Postgrad Med J* 1990, 66: 807–817.
3. Critchley, E.M.R., Hayes, P.J. & Isaacs, P.E.T. Outbreak of botulism in North West England and Wales. *Lancet* 1989, ii: 849–853.
4. Jones, S., Haugh, C., Starer, F., Huma, Z., Young, Y. & Sinclair, L. Central nervous system involvement in infantile botulism. *Lancet* 1990, i: 228.
5. Colbatch, J.G., Wolff, A.H., Gilbert, R.J. *et al.* Slow recovery from serious foodborne botulism. *Lancet*, 1989, ii: 1216–1217.
6. Christie, A.B. Botulism in Infectious Diseases. *Epidemiology and Clinical Practice*. Churchill Livingstone, Edinburgh, 1987, pp. 338.
7. Schaffner, W. *Clostridium botulinum*. In: Mandell, G.L., Douglas, R.G. & Bennett, J.E. (eds). *Principles and Practice of Infectious Diseases*, Chapter 22, Churchill Livingstone, Edinburgh, 1990.
8. Arnon, S.S., Midura, T.F., Damus, K. *et al.* Intestinal infection and toxin production by *Clostridium botulinum* as one cause of sudden infant death syndrome. *Lancet*, 1978, ii: 1273.
9. Sonnabend, A.R., Sonnabend, F.F., Krech, V., Molz, G. & Sigrist, T. Continuous microbiological and pathological study of 70 sudden and unexpected infant deaths: toxigenic *Clostridium botulinum* infection in 9 cases of sudden infant death syndrome. *Lancet* 1985, i: 237–241.

Intraocular pressure

Sir,

With tons of journals received weekly, I usually read the table of contents of each to decide which articles I should read in depth. I was immediately attracted to the paper by Al-Sereit *et al.*¹ when I read the title 'Intraocular pressure and papillary responses in patients with diabetes mellitus' in the Contents of the March issue of the *Journal*. I was curious to know if the papillary responses referred to the optic papilla or the renal papillae.

I, of course, was very surprised to find that it was the pupillary responses, not papillary responses, the article was about. Thanks to this printing error, I was glad that I learned something about the autonomic neuropathy in patients with diabetes mellitus.

Tsung O. Cheng, M.D.

Division of Cardiology,
The George Washington University,
2150 Pennsylvania Avenue, N.W.,
Washington, DC 20037, USA.