been because HBsAg was not present in the infected trainee's saliva at the time of the CPR class or because the infectivity of the saliva was low. Probably HBsAg was present in his saliva. It usually appears in the blood several weeks before the onset of clinical signs and symptoms. He developed jaundice eight days after the class. Infectivity may be related to the amount of virus present and the mode of transmission. The infectivity of a secretion cannot be determined by the presence of HBsAg, since most of this antigen is associated with subviral particles or defective viral particles.[4] The titre of HBsAg is lower in saliva than in the corresponding serum.[3] Bancroft *et al*[2] found that gibbons could be infected by subcutaneous but not by intranasal or intraoral administration of the same HBsAg-positive saliva inoculum, showing the importance of the mode of transmission.

The cleaning of our manikin heads between use was inadequate to decontaminate for hepatitis B virus. Thus the trainees probably had the maximum exposure that might occur in CPR training. We therefore believe that viral hepatitis type B is unlikely to be transmitted from oral contact with CPR training manikins or other fomites. Mandatory testing of all individuals for HBsAg before CPR training is unwarranted.

[1] MacQuarrie, M B, Forghani, B, and Wolochow, D A, *Journal of the American Medical Association*, 1974, **230**, 723.
[2] Bancroft, W H, *et al*, *Journal of Infectious Diseases*, 1977, **135**, 79.
[3] Alter, H J, *et al*, *Infection and Immunity*, 1977, **16**, 928.
[4] Gerin, J L, Ford, E C, and Purcell, R H, *American Journal of Pathology*, 1975, **81**, 651.

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Interaction between disopyramide and practolol

Practolol and disopyramide are commonly used together in treating supraventricular tachycardia, and no adverse interaction has been described. We report two cases in which after practolol and disopyramide were used sequentially to treat supraventricular tachycardia the patients suddenly deteriorated and one died.

Case reports

Case 1—A 56-year-old man was admitted to hospital in March 1979 after 48 hours of chest pain. He had had myocardial infarctions in 1966 and 1974, but had since been well and was receiving no treatment. He was found to have a supraventricular tachycardia of 180 beats/min; carotid sinus massage had no effect. He was given 20 mg practolol intravenously with no response, and then 20 minutes later a slow intravenous injection of 150 mg disopyramide. Within two minutes he developed a sinus bradycardia of 25/min, lost consciousness, and became profoundly hypotensive. He was given 0·6 mg atropine intravenously without an improvement in his heart rate. Later, while a temporary pacemaker was being inserted, his heart rate increased to 60/min. Electrocardiography showed sinus rhythm, right bundle-branch block, left axis deviation, and an acute anteroseptal myocardial infarction. After 24 hours he again developed a supraventricular tachycardia of 180/min; with the pacemaker in position, he was given 150 mg disopyramide intravenously alone, producing a sinus rhythm of 100/min. Thereafter he remained well on treatment with oral disopyramide.

Case 2—A 67-year-old man with widespread vascular disease presented with palpitations, and was found to have a supraventricular tachycardia of 180/min, which did not respond to carotid sinus massage. He was given 10 mg practolol intravenously with no effect and 20 minutes later a slow intravenous injection of disopyramide. After 80 mg had been injected his heart rate reverted to a sinus rhythm of 80/min; this then slowed to 20/min despite intravenous atropine and asystole occurred. He was resuscitated with intracardiac adrenaline, but he remained unconscious and died five hours later.

Comment

In both cases the patients' deterioration was closely related to injection of disopyramide. This drug is now widely used to treat supraventricular tachycardia, apparently with safety,[1] although temporary slowing of the atrial rate by up to 30%, widening of the QRS complex, and transient hypotension have occurred.[2] When given disopyramide without having previously been given practolol, our first patient suffered no ill effects. We therefore suspect an adverse interaction between practolol and disopyramide, and suggest that intravenous disopyramide should not be given after practolol.

[1] Hillis, W S, *et al*, *Journal of International Medical Research*, 1976, **4**, suppl No 1, p 74.
[2] Mizgala, H F, and Huvelle, P R, *Journal of International Medical Research*, 1976, **4**, suppl No 1, p 82.

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Vancouver style

All manuscripts submitted to the *BMJ* from now on should conform to the uniform requirements for manuscripts submitted to biomedical journals (known as the Vancouver style).

The *BMJ*, together with many other international biomedical journals, has agreed to accept articles prepared in accordance with the Vancouver style and will be introducing the system from January 1980. The style (described in full in *BMJ*, 24 February, p 532) is intended to standardise requirements for authors and covers text format, presentation of methods and results, use of SI units, and the form of tables and illustrations. All the participating journals have also agreed to introduce a standard form of references.

In future references to papers submitted to the *BMJ* should include: the names of all authors if there are fewer than seven or, if there are more, the first three followed by *et al*; the title of journal articles or book chapters; the titles of journals abbreviated according to the style of *Index Medicus*; and the first and final page numbers of the article or chapter.

Examples of common forms of references are:

[1] International Steering Committee of Medical Editors. Uniform requirements for manuscripts submitted to biomedical journals. *Br Med J* 1979;**1**:532-5.
[2] Soter NA, Wasserman SI, Austen KF. Cold urticaria: release into the circulation of histamine and eosinophil chemotactic factor of anaphylaxis during cold challenge. *N Engl J Med* 1976;**294**:687-90.
[3] Weinstein L, Swartz MN. Pathogenic properties of invading microorganisms. In: Sodeman WA Jr, Sodeman WA, eds. *Pathologic physiology: mechanisms of disease*. Philadelphia: W B Saunders, 1974:457-72.

Up to the beginning of October some 100 journals had agreed to accept articles in the Vancouver style, and a full list will be printed early in 1980.