defibrillation attempts [3]. Such evidence must now seriously call into question the continued role of lignocaine as the recommended second-line drug treatment for ventricular fibrillation.

Second, in the past few years the effectiveness of the class III antiarrhythmic drugs (namely amiodarone, bretylium and sotalol) has been investigated. In particular, the effectiveness of amiodarone [4] and the ineffectiveness of bretylium [5] to reduce the defibrillation threshold in the chronically instrumented dog model has been highlighted. Such evidence might have been usefully included in the review to assist in the future identification of suitable drugs during cardiopulmonary resuscitation.

Sir,—I am grateful to you for the opportunity to reply to the letter by Drs Quiney and Allen.

I believe that lignocaine has not proven to be of substantial value in the management of cases of ventricular fibrillation because of, not only its potential to increase the defibrillation threshold, but also its negative inotropic activity [1]. Its use is probably best reserved for cases of persistently recurring ventricular fibrillation after electrical defibrillation, particularly in association with reperfusion occurring after open heart surgery [2].

Neither bretylium nor amiodarone have attracted sufficient support in the clinical field as yet to enable them to be recommended confidently [3]. Of the two, amiodarone appears to have the brighter future, on present evidence.

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USE OF LIGNOCAINE IN VENTRICULAR DEFIBRILLATION?

Sir,—We read with interest the recent review “Advances in Cardiopulmonary Resuscitation” [1]. In the review of drug therapy, we were surprised by the omission of a discussion of the role of lignocaine for the treatment of ventricular fibrillation. Despite its theoretical advantages, it is well recognized as causing an increase in defibrillation threshold [2]. More recent evidence also has pointed to a three-fold increased incidence, after lignocaine administration, of asystole following subsequent defibrillation attempts [3]. Such evidence must now seriously call into question the continued role of lignocaine as the recommended second-line drug treatment for ventricular fibrillation.

Sir,—I read with interest the study by Rabey and colleagues [1] demonstrating a reduction in lower oesophageal sphincter pressure (LOS P) during halothane anaesthesia with a laryngeal mask. Comparison with a Guedel oral airway. When LOS P is measured continuously, the end-expiratory pressure can vary greatly over a 5-min period [2]; I am concerned that when LOS P is measured for a moment in time with the pull-through technique used by Rabey and colleagues, it may be subject to a significant sampling error and may miss brief reductions in pressure that could be clinically important. Furthermore, as decreased LOS P values of 5–6 cm H2O greater than gastric pressure are sufficient to prevent gastro-oesophageal reflux, even during...