arm seemed to increase in both strength and bone content. As explained in our results, several volunteers admitted exercising both arms. The correlation between grip strength and bone mineral content, however, remained across all groups and at all stages in both the volunteer and fracture studies.

The small loss between the values at the start and those after six months reflects the natural rate of bone mineral loss seen in aging women. Interestingly, there was a corresponding loss in grip strength.

We originally designed an experiment that was to run for a six week period of exercise. We chose to use the raw bone mineral content scores precisely because of the variation in bone mineral content measurements that subtract a supposed circumferential fat layer. They derive rather than measure fat content. In the region examined it is unreasonable to expect a net gain or loss in body fat in a mere six weeks of occasionally squeezing a tennis ball. The relatively large changes in grip strength and the associated significant gains in bone mineral content would in fact outweigh any changes in fat content. We used a routine technique and daily phantom standardisation in our experiment. This reduces the variation in the subject's grip technique and positioning errors, which are probably the greatest source of error in other series.

We agree with Dr Stevenson and Ms Lee's last point: further work is certainly needed on the role of brief periods of skeletal stress in the reversal of osteoporosis. This free and physiological augmentation of the skeleton may, however, be more effective than some current expensive and unproved remedies.

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Cricoid pressure during cardiopulmonary resuscitation

SIR,—We would like to comment on one aspect in the excellent article by Mr A K Marsden[1]: the use of cricoid pressure (Sellick's manoeuvre) during two rescuer cardiopulmonary resuscitation by health care professionals.

This procedure requires the operator to use both hands.[2] It is often used during induction of anaesthesia to prevent pulmonary contamination by gastric contents. Once applied, cricoid pressure must not be relaxed until the trachea is protected by a cuffed endotracheal tube. Any period without cricoid pressure allows passive regurgitation into the pharynx; hence pulmonary soiling is possible with positive pressure ventilation, rendering the technique pointless. During two rescuer cardiopulmonary resuscitation it is not possible for one person to apply and maintain cricoid pressure continuously while performing chest compression. Thus we believe that a third person must be available to apply cricoid pressure, as recommended in the American standards for cardiopulmonary resuscitation.[3]

Training of health care professionals in this manoeuvre must be thorough because poorly applied cricoid pressure distorts the anatomy, making ventilation and intubation more difficult.[4] To our knowledge no suitable training manikin exists to teach this manoeuvre. Adequate training in the application of cricoid pressure can be given in anaesthetic rooms. We believe that this should be encouraged: it would give trainees opportunities to practise airway management skills under experienced supervision in a safe and controlled environment.

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1 Marsden AK. Guidelines for cardiopulmonary resuscitation. Basic life support. *Br Med J* 1989;**299**:442-5. (12 August.)
2 Sellick BA. Cricoid pressure to control regurgitation of stomach contents during the induction of anaesthesia. *Lancet* 1961;ii: 404-6.
3 Standards and guidelines for cardiopulmonary resuscitation and emergency cardiac care. *JAMA* 1986;**255**:2905-84.
4 Churchill-Davidson HC, ed. *A practice of anaesthesia*. 4th ed. London: W B Saunders, 1979:1364.

Advanced life support

SIR,—A serious flaw in Dr D A Chamberlain's article on the updated guidelines for treating different modes of cardiac arrest[1] is the absence of any mention of hypoxic cardiac arrest, which is undeniably the cause of death in 1500 asthmatic patients every year in Britain.

It is known from animal experiments and from observing brain dead patients disconnected from ventilators that the most common course of cardiac events in profound hypoxia, such as occurs in severe acute asthma, is not the sudden onset of ventricular fibrillation but progressive sinus bradycardia followed by slow idioventricular rhythm and cardiac arrest in asystole. A vital measure in resuscitating a dying asthmatic patient is therefore to correct hypoxia by immediate intubation and ventilation with 100% oxygen. If the patient has not progressed beyond the stage of sinus bradycardia or if idioventricular rhythm has only recently supervened this measure will usually return the patient to sinus tachycardia and restore an adequate circulation. Even if cardiac arrest has occurred a sharp blow on the sternum will often restart cardiac action, provided the arrest has been of short duration and the lungs are being adequately ventilated with oxygen. If, however, the treatment of such patients does not include reoxygenation and is restricted to the measures shown on the apparent asystole algorithm a fatal outcome will be inevitable.

Aside from the special and crucial need for reoxygenation in treating cardiac arrest in asthma and in other conditions causing profound hypoxia, it is strange that the guidelines for prolonged resuscitation and post resuscitation care contain no mention of giving oxygen. Could the reason for that omission, and for the apparent disregard of the problem of hypoxic cardiac arrest, be that the guidelines are mainly orientated towards cardiac arrest in ischaemic heart disease and place insufficient emphasis on conditions in which severe hypoxia is either the primary cause of cardiac arrest or a major contributory factor?

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1 Chamberlain DA. Advanced life support. *Br Med J* 1989;**299**: 446-8. (12 August.)

AUTHOR'S REPLY,—The importance of hypoxia as a cause of asystolic cardiac arrest cannot be overemphasised, and Dr Grant's points are well taken. No member of the Resuscitation Council would disagree with them.

There is, however, an element of misunderstanding, which I am glad to clarify. My remit for the article on advance life support[1] was to provide an apologia for the changes from the 1984 guidelines; this is stated clearly in the introduction. The article was not intended to be a definitive account of advanced life support. The recommended procedures will be discussed more fully in the revised edition of the *ABC of Resuscitation*, due to be published in the autumn. The algorithms that were published in the recent article came from the new cardiopulmonary resuscitation poster published by the Resuscitation Council. The full poster has adequate emphasis on the importance of the airway, the need for airway adjuncts, the value of oxygen treatment, and the possible need for intubation. These were not included in the algorithms in the article because there were no major changes since 1984. Copies of the full poster may be obtained from the Resuscitation Council, c/o Department of Anaesthetics, Royal Postgraduate Medical School, Hammersmith Hospital, Du Cane Road, London W12 0HF.

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Ethics of clinical research

SIR,—The article by Professor Michael Baum and colleagues fails to address some of the more worrying questions provoked by the 1980 Cancer Research Campaign adjuvant breast cancer trial.[1] Several leading international authorities in the drug treatment of cancer were concerned about the ethics of the study before it started. Based on evidence available then, the cyclophosphamide only arm was considered inadequate treatment compared with a three drug combination of cyclophosphamide, methotrexate, and fluorouracil. They were concerned that suboptimal treatment was being given to uninformed patients. These criticisms were originally published in the *BMJ*[2,3] and cited again more recently in the *Institute of Medical Ethics Bulletin*.[4]

One justification for continuing the trial despite these concerns was that the ethics committee at King's College Hospital had decided that to proceed was ethically proper. As a committee can make judgments only on the evidence presented to it, this raises the question of what the committee members were told. For example, when they decided that informed consent was no longer necessary were they aware of the reservations about the ethics of the trial published in the *BMJ* or that cyclophosphamide alone would probably be of no benefit to 90% of the patients receiving it?

The implication that a requirement for informed consent might make patients reluctant to enter a randomised study is only a small part of the problem. Other reasons why patients are not entered into clinical trials generally include reluctance of surgeons and radiotherapists to inform patients that the option exists, inability to meet the trial protocol because of the lack of local facilities, and patients' insistence on adjuvant chemotherapy outside the setting of a trial. Even so, the original good news from Milan that mortality in high risk premenopausal patients with breast cancer can be significantly reduced with cyclophosphamide, methotrexate, and fluorouracil chemotherapy without unacceptable side effects has been confirmed by randomised studies conducted mainly in the United States in which informed consent was obtained.[9-11]

In my opinion most of the ethical controversy raised by the Cancer Research Campaign trial can be reduced to one simple question: Are patients entitled to know what treatment their doctors are giving them and why? I think the answer must always be "Yes." If the King's ethical committee was aware of the trial criticisms it seems to have decided that the answer to this question was "No."