

TABLE A.—Abortions Notified in England and Wales, Second and Third Quarters, 1968, According to Marital State

Marital State :	Not stated	Single	Widowed, Divorced, or Separated	Married	Total
England and Wales	29	5,773(47%)	970(8%)	5,579(45%)	12,351
Cheltenham	—	7(21%)	4(11%)	23(68%)	34

TABLE B.—Total Number of Therapeutic Abortions at Two London Teaching Hospitals and at Cheltenham

	1963 (one year)	1968		Increase
		7 months	12 months estimated	
Chelsea Hospital for Women	30	148	254	8½ times
Guy's Hospital	10	81	138	14 times
Cheltenham	10	47	80	8 times

tenham in 1963 was the same as at Guy's Hospital (10), but the subsequent increase at Guy's has been appreciably greater. Presum-

ably this is partly due to the fact that many women seeking abortions gravitate to London, whereas the cases dealt with in Cheltenham come from the clinical area served by the hospital.

Already we are finding that the impact of the Abortion Act is making great demands on hospital beds and operating time, and we agree wholeheartedly with Mr. Lewis's statement to the effect that the whole character of the gynaecologist's outpatient work has altered because of the numerous requests for termination at almost every session.—We are, etc.,

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### Is Quinidine Outdated?

SIR,—“Be not the last to cast the old aside” must have come to many minds on reading your leading article on quinidine (8 February, p. 331).

I have used this drug for 20 years, without disaster, for two main purposes. First, in a small maintenance dose, 300–900 mg. daily, to control annoying but not dangerous arrhythmias (for example, paroxysmal atrial tachycardia or fibrillation) in those with otherwise normal hearts. Some have maintained control for over ten years without mishap, among them some of my colleagues or their wives, whose mishaps could not escape my notice. Second, to control dangerous arrhythmias (for example, ventricular tachycardia) after myocardial infarction. Five years ago, the reports that you quote, notably that of Oram and Davies,<sup>1</sup> led me to discontinue this last practice. The frequency of late, often fatal, recurrences in the third or fourth week, however, made me go back to quinidine with an improvement in results. Certainly D.C. shock can be repeated, though multiple shocks—60 in one record—must be trying to the patient. Lignocaine infusions can run for a while, but the patients described in your columns a week earlier (25 January, p. 213), who died in recurrent ventricular tachycardia some days after a “full course” of lignocaine, seem to me to cry out for quinidine.

Oram and Davies must be listened to with respect, and there are doubtless reasons for their different experience. Yet one patient who had two episodes of atrial fibrillation reverted by shock at their hospital was referred to me with a third recurrence and remains in sinus rhythm on quinidine. Finally, I must agree with your conclusion that we need a safe oral antiarrhythmic agent for long term use (can any depressant of myocardial excitability be quite safe?), but until we find one let us not discard quinidine.—I am, etc.,

London W.1.

C. P. PETCH.

#### REFERENCE

- <sup>1</sup> Oram, S., and Davies, J. P. H., *Lancet*, 1964, 1, 1294.

### Treating Shock

SIR,—The most interesting paper by Dr. J. F. Riordan and Dr. G. Walters (18 January, p. 155) comes at a time when many physicians are flirting with the idea of using phenoxybenzamine in cardiogenic shock and in intractable pulmonary oedema. It would, therefore, be a tragedy if these people were to be deterred from trying this regimen because of one series of failures.

McGowan and Walters<sup>1</sup> were among the first to demonstrate the importance of restoring flow rather than blood-pressure in the shocked state. It would be a great pity if Dr. Walters were now to contribute to perpetuation of the fallacy that there is some mystical fundamental difference between cardiogenic shock and all other forms of shock. Of course there is a difference in that a cardiac lesion is the primary factor in inducing the shocked state, but Lillehei's team<sup>2</sup> have shown clearly that shock can ensue where the initial infarct is very small, and that the extensive infarction found at necropsy may be the result of, rather than the cause of, the shocked state.

There are, therefore, good academic grounds for believing that phenoxybenzamine is worthy of trial in cardiogenic shock, and these are well supported by observations on experimental myocardial infarction in dogs. One of the most difficult problems, however, is that of definition in cardiogenic shock. Three separate syndromes tend to be confused: morphine-induced hypotension; vasovagal collapse; and true shock.

In the absence of complete heart block, we believe that true cardiogenic shock has tachycardia as one of its essential components, and that the group as defined by Drs. Riordan and Walters, but with the addition of tachycardia, has a mortality nearer to 100% than 80%. On the other hand, where there is bradycardia, the prognosis is probably very much better despite the other “shock” features. We would, therefore, have predicted a more favourable outcome for case 6, as was indeed the case. Indeed, if Drs. Riordan and Walters's paper proves anything, it proves the difference

between hypotensive coronary patients with bradycardia and those with tachycardia. It seems likely that a larger dose of atropine might have produced a more dramatic beneficial effect in this case.

Dietzman and his co-workers' observation that shock produces extension of infarction coupled with Drs. Riordan and Walters's statement that the signs of shock were present in all their cases “for at least four hours” would seem to hold the key to the failure of treatment in these six cases. Accepting that cardiogenic shock is ever reversible, it seems too much to ask of any regimen that it should reverse the condition when it has been present for such a length of time. Accordingly, failure of treatment is no proof of ineffectiveness of the drug used in such circumstances. Nor is any mention made of the use of inotropic drugs, correction of metabolic acidosis, and the possible place of blood-volume expanders, all of which must form an integral part of the treatment regimen.

More disquieting is the report of adverse respiratory effects following phenoxybenzamine. The workers cited above have advocated use of the drug for, or to prevent, pulmonary oedema. We have now used it for this indication in several patients with uniform success—the moist lungs in each case clearing completely where digoxin and frusemide had failed. We cannot understand why our experience has been so different from your contributors' in this context. We only wish we could claim equal success with cardiogenic shock, in which our survival rate so far has been identical with that which your contributors report. We believe, however, that our failures are the result of untoward delay—the hesitation to use an experimental approach before one is certain that conventional treatment has failed. We now believe that we are sufficiently familiar with this drug to justify our use of it immediately where shock is apparent, and we hope that this approach will enable us to make a better appraisal of its effect.—We are, etc.,

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#### REFERENCES

- <sup>1</sup> McGowan, G. K., and Walters, G., *Lancet*, 1966, 1, 611.  
<sup>2</sup> Dietzman, R. H., Lyons, G. W., Bloch, J. H., and Lillehei, R. C., *J. Amer. med. Ass.*, 1967, 199, 825.

### Awareness during Anaesthesia

SIR,—Awareness during operation, and at any other time, depends in the main on sensory input, a large part of which can be the proprioceptive impulses from muscles. When light anaesthesia is coupled with the administration of a muscle relaxant, if signs of arousal such as movement, attempts to open the eyes, pupillary dilatation, or lachrymation appear, these usually disappear as soon as more relaxant is given—probably owing to the reduction of muscle tone and consequent diminution of proprioceptive input. Furthermore, if an operation is performed under hypnosis, signs of arousal frequently parallel the increase of proprioceptive impulses produced by the stretching of muscle; painful stimuli, such as those arising