and the doctor's general assessment and opinion.

Dr. Sanazaro is experienced enough to anticipate criticism of the scheme. The criteria could be restrictive; they could become rigid rules which are difficult to modify or change, they could limit judgement and retard innovation. His qualifying comment that such things have not happened after medical auditing is not entirely reassuring and the cynic might reasonably suggest that even more committees are needed to pronounce on this. In general, bureaucratic methods seldom encourage initiative and are prone to political expediency. They tend to encourage mediocrity and overemphasize the importance of mistakes. Doctors, like many of their fellow men and women, certainly make mistakes. But to assume that medical auditing might render them infallible would be naive as long as medicine is recognized to be far from a pure science.

The main stimulus to medical auditing, rightly or wrongly, is financial, and indeed the major insurance companies and the federal government in the U.S.A. require hospital accreditation by medical auditing before reimbursement. Such a scheme is not, and is not likely to be, applicable to hospitals in the United Kingdom unless there is a breakdown in the hospital service as a result of disaffection on the part of the doctors, nurses, and technical and other staff. More than a few of the professional staff feel that a growing frustration and a tendency to build up an ever-increasing and costly administration, unless checked, could lead to a significant decline in hospital standards. Others maintain that this has already happened. But whatever the facts may be it is to be hoped that the traditions of British medical and nursing training will not be forgotten and that responsibility and service will continue to be acknowledged as attributes.

A good case, no doubt, could be made for developing a bureaucratic system of medicine in a society that is wedded primarily to materialism. But in our own environment we have already learnt that it is unwise to assume that bureaucracy will add much, if anything, to well-tried and evolved systems even if such systems are not perfect. If medical auditing is ever tried in the United Kingdom let us hope that it will be conducted as a properly controlled experiment and that the results will be evaluated by reputable representatives of all, not just a section, of those people concerned with medical care.—I am, etc.,

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SIR,—I have just read with great interest the symposium on medical audit (16 February, p. 271).

The principle, of course, is good. Self-examination is an age-old discipline propounded by Dr. A. K. Thould's medieval church even before it was medieval. I agree with Dr. Thould that rigidity is the greatest danger, especially in general practice, which by its very nature must be flexible if it is to survive in its true and necessary state. One of my greatest fears is that with the imminent reorganization of the Health Service general practice will be submerged in a welter of irrelevant statistics poured out by a top-heavy, over-powerful, and inexperienced administrative body and that when it re-emerges it will be an emasculated service, part mini-specialist, part public health, and in its entirety not general practice in its true sense. Doubtless my consultant colleagues have similar fears for their particular specialties.

There are certain basic principles of medical practice which must be held constantly in mind when a medical audit is being held:

(1) The healing of the sick and prevention of disease. A platitude, one may well say, but is there any one of us who could truly deny that when bogged down under the administrative minutiae of medicine today he tends to forget this, even if only for a short moment?

(2) Complete liaison between consultant and G.P. If (1) above is to be effective, this principle is essential. It has happened in the evolution of medical practice that the consultant treats the disease whereas the G.P. treats the patient. In fact the G.P. must, like the Catholic priest, be oriented to treat the whole man. If there is to be this essential liason, the present Cogwheel system must be greatly improved and include the G.P. as an equivalent cog to the consultant, each being a principal in his own right and master of his trade.

(3) The physician or surgeon must be master in his own house. The doctor is the head of the house in the medical family. He is the principle worker and "bread-winner" in the sense of providing healing, so he must also be the maker of decisions, the ultimate authority. He must not for the sake of an easier life or for convenience relinquish this authority to other members of the family, particularly to the administrative organization. An ideal concept, one may well say, already lost, but the basic principle remains.

There are many other basic principles that one could enlarge upon, such as adequate time, adequate facilities, time for recreation, etc., but most of these occupy our waking thoughts perpetually and time need not be spent on them here. One basic principle, however, will bear repeating ad nauseam—unity in the profession. It is tragic how we let ourselves be fragmented into smaller dissident groups each fighting for our own little kingdom, whereas united we would be an effective body in improving the state of medical practice, an authority to be reckoned with and not subjugated to political or bureaucratic expediency.—I am, etc.,

DAVID HOOKER

Truro

Current Practice in Tetanus Prophylaxis

SIR,—With the free availability of human antitetanus serum there will be renewed interest in the methods of tetanus prophylaxis.

The widespread use of antitetanus serum (A.T.S.) prepared from the horse dates from the first world war and resulted in a considerable reduction in the incidence of tetanus. Since 1963, when Cox and her colleagues[1] in Sheffield wrote their paper, the emphasis has changed and we were of the opinion that current practice in England followed her views. However, discussion with our colleagues indicated that this did not seem to be universally accepted. It was therefore decided to find out the current methods of prophylaxis, in the non-immune patient, in use in casualty departments in Britain.

A questionnaire was sent to 63 members of the Casualty Surgeons' Association in Great Britain. Forty-nine of these replied Forty-five stated that they never used A.T.S. (horse) in tetanus prophylaxis but advocated antibiotic prophylaxis, usually penicillin associated with wound toilet. Twenty-two of the doctors stated that they did not see a use for A.T.S. (human) even if it was readily available, but the four who used A.T.S. (horse) said they would use A.T.S. (human) if it was freely available. Twenty-one others declared an interest in A.T.S. (human) and felt that there might be a place for its use in selected cases.

A recent leading article in the *Lancet*[2] announces the availability of this human serum but gives only general indications for its use. It is evident from the above survey that there are no generally accepted indications for the use of A.T.S. (human) in tetanus prophylaxis and that the most common prophylactic procedure is surgical debridement with chemoprophylaxis.—We are, etc.,

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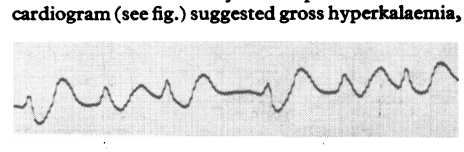
1 Cox, C. A., Knowelden, J., and Sharrard, W. J. W., *British Medical Journal*, 1963, 2, 1360.
2 *Lancet*, 1974, 1, 51.

Death from Accidental Potassium Poisoning in Childhood

SIR,—I should like to report the fatal poisoning of a small child by Slow-K tablets. Overdosage by intravenous potassium is a well-known hazard, but there are few reports of fatal poisoning by any form of oral potassium, and none hitherto of death due to ingestion of Slow-K.[1 2]

A 15-month-old boy was admitted soon after midnight almost moribund. He came from a chaotic family and had been left for the evening in the care of a bed-ridden and demented great-grandmother who had not noticed that her Slow-K tablets had got mixed up with the Smarties he was eating. He swallowed at least eight tablets and sucked the sugar coat off about 30 more that were found scattered around him, together with more unsucked tablets and with Smarties. He vomited and became unconscious and lay for several hours before being taken to hospital. He was then flaccid and cyanosed, with shallow respiration, quiet heart sounds, and peripheral circulator failure. He was dirty but well-nourished and normally developed. An electrocardiogram (see fig.) suggested gross hyperkalaemia,



which was confirmed by a serum potassium level of 9.9 mEq/l., with sodium 138 mEq/l., chloride 120 mEq/l., and urea 22 mg/100 ml. He was treated with intravenous dextrose, calcium gluconate, and sodium bicarbonate, with intragastric sodium polysterene sulphonate, and by peritoneal dialysis, but he died 75 minutes after admission. His serum potassium had by then risen to 14 mEq/l. At necropsy eight Slow-K tablets, partly dissolved, were recovered from his stomach; the kidneys appeared normal. There was no evidence in the home or at necropsy that he had taken any other type of tablets, and there was no salicylate in his serum.

Excess ingestion of potassium rarely produces lethal serum levels,[3] probably because it is counterbalanced by the vomiting and

diarrhoea that are often provoked and by increased elimination of potassium by the normal kidney,[4] though of course if the kidney is diseased, dangerous hyperkalaemia may ensue. Though this child vomited and apparently had normal kidneys he evidently absorbed enough potassium to overwhelm the excretory mechanism. The final serum level was probably inflated by the effects of anoxia and acidosis. The electrocardiogram shows an advanced stage of hyperkalaemia[5] where atrial activity has stopped and there is an irregular ventricular rhythm with grossly elongated complexes. Asystole or fibrillation is imminent.

This sad case again illustrates how dangerous pills that look like sweets can be for children,[6 7] and re-emphasizes that all medicaments, even those as apparently innocuous as Slow-K, should be kept securely out of reach.— I am, etc.,

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2 Committee on Safety of Medicines, personal communication, 1973.
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Breast Cancer Regression under Oestrogen Therapy

SIR,—We read with great interest the provocative article by Dr. B. A. Stoll (25 August, p. 446) as well as the letter from Drs. P. Garcia-Webb and M. H. Briggs (17 November, p. 419) raising several points in debate. One major issue in the discussion was the question whether high doses of oestrogens increase blood prolactin levels in postmenopausal breast cancer patients, as stated by Dr. Stoll quoting our unpublished data. We wish to report these data, as well as others which are pertinent to the discussion.

The effect on blood prolactin levels of two oestrogen-related compounds, ethinyloestradiol and nafoxidine (U11, 100A) which are both active in the therapy of advanced breast cancer[1 2] was studied. Ethinyloestradiol 1 mg thrice daily by mouth, and nafoxidine 60 mg thrice daily by mouth, were given respectively to four and six postmenopausal women with advanced breast cancer. None was receiving any other drug known to alter prolactin secretion. Blood samples were collected between 8 and 9 a.m. after an overnight fast for several days before and during treatment. All samples from the same individual were run in the same assay for prolactin as previously described.[3 4] Since the distribution of prolactin values appeared to be log-normal, calculations and statistical analysis were made after logarithmic transformation.

Ethinyloestradiol in doses therapeutically active in advanced breast cancer significantly increased basal prolactin secretion (see table). This observed increase seems thus to refute the generally held theory explaining the therapeutic effect of high-dosage oestrogens in breast cancer by an alleged inhibition of prolactin secretion.[5 7] On the other hand nafoxidine failed to stimulate prolactin secretion and even had an inhibitory effect in one case.

Nafoxidine is an oestrogen antagonist[8] that binds to tissue-specific "oestrogen receptors," acting at this level as a competitive inhibitor of oestrogens.[9] Nevertheless, this compound also behaves as a weak oestrogen in castrated animals[10] and in postmenopausal women.[2] It inhibits growth of the dimethylbenzanthracene (DMBA)-induced rat mammary carcinoma[11 12] yet it counteracts to a limited but definite extent the ovariectomy-induced regression of this tumour, possibly by its weak oestrogenic properties.[13] In postmenopausal women with advanced breast cancer nafoxidine has been found to induce objective remissions in approximately 35% of the patients treated.[2] The question, however, is not entirely settled whether the therapeutic effect of nafoxidine in postmenopausal breast cancer patients is caused by oestrogen antagonism or by oestrogen action. The latter hypothesis finds some support in the observation that both ethinyloestradiol and nafoxidine seem active in the same category of patients—namely, those in whom cancer tissue contains "oestrogen receptors."[14]

If nafoxidine and ethinyloestradiol produce tumour regression in breast cancer through their oestrogenic properties, it is necessary to postulate that they do so independently of their divergent effects on prolactin secretion. In the DMBA-induced mammary cancer of the rat, in the development and growth of which prolactin plays a crucial part,[15] it has been suggested by Meites[16] that the inhibitory effect of large doses of oestrogens is exerted by interference with the peripheral action of prolactin on tumour cells. If this were also the case in human breast cancer, then greater antitumour efficacy may theorically be expected from oestrogenic-type compounds, such as nafoxidine, which are devoid of stimulating effect on pituitary prolactin secretion.

Blood Prolactin Levls before, after and during Treatment

Case	Age (years)	Type of treatment	Control prolactin* Mean (± 1 S.E.M.)	n	Treatment prolactin* Mean ± 1 S.E.M.)	n	P (Student's *t* test)
1	51	Ethinyloestradiol	357 (308-746)	2	673 (603-746)	3	<0·05
2	54	Ethinyloestradiol	89 (87- 93)	5	202 (180-227)	7	<0·001
3	72	Ethinyloestradiol	542 (499-510)	9	893 (766-1042)	6	<0·02
4	89	Ethinyloestradiol	423 (399-449)	6	1302 (900-1157)	5	<0·001
5	61	Nafoxidine	74 (40-138)	3	50 (37- 67)	5	N.S.
6	69	Nafoxidine	642 (630-654)	2	720 (667-777)	6	N.S.
7	72	Nafoxidine	552 (500-610)	9	484 (429-545)	2	N.S.
8	74	Nafoxidine	166 (134-205)	5	160 (113-226)	4	N.S.
9	75	Nafoxidine	47 (38- 58)	4	64 (52- 77)	4	N.S.
10	80	Nafoxidine	480 (469-490)	6	318 (297-341)	5	<0·001

*Expressed as mU/ml by reference to the laboratory standard. Calculations and statistical analysis after logarithmic transformation. n = number of determinations.
N.S. = not significant.

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—We are, etc.,

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Geriatric Orthopaedics

SIR,—In the section on hip fractures in his otherwise admirable article (2 February, p. 190) Mr. M. B. Devas states that "there is no contraindication to a geriatric patient receiving either general anaesthesia or hypotension, each of which materially benefits both patient and surgeon."

We have over 25 years' experience of anaesthesia for these cases and only occasionally have either we or the surgeon considered it desirable to induce hypotension. We make extensive use of induced hypotension in suitable patients of all ages for all types of surgery. In our view, however, it is undesirable and possibly dangerous to advocate the unrestricted use of this technique in a group of patients many of whom, perhaps the majority, are in such poor physical and mental shape that hypotension would be an unjustifiable risk. Further, these patients are not always operated on by surgeons of sufficient experience to make use of hypotension safely.

We believe that these patients should be investigated in the manner described by Mr. Devas and operated on at the earliest convenient moment without hypotensive