	Senior	Junior	No Consensus Diagnosis	1970 Diagnosis
Total no. of diagnoses	876	698	8	292
Major difference (A)	18 (2·1%)	56 (8·0%)		2 (0·7%)
Minor difference (B)	41 (4·7%)	77 (11·0%)		12 (4·1%)
Total A + B	59 (6·8%)	133 (19·0%)		14 (4·8%)

been obtained in all but eight of the first 300 cases reviewed. Differences between the consensus diagnosis and the participants' diagnoses are placed in two categories; (*a*) a diagnosis that gives a misleading prognosis or one that would lead to inappropriate treatment, and (*b*) a minor diagnostic difference of no clinical relevance. The consensus diagnosis has been compared also with the original 1970 diagnosis. Nine pathologists participated. These have been divided into two groups, senior pathologists who have obtained the M.R.C.Path. diploma and junior pathologists without this diploma. The results of the first 300 cases are shown in the table. Since clinical information was minimal and since consultation between pathologists before diagnosis was not permitted, the figures may be interpreted as the maximum for incorrect diagnosis.

The benefits of this on-going system are as follows. (1) It has proved to be a valuable educational exercise for consultant and trainee pathologists. (2) Uniformity of nomenclature and diagnostic criteria have been promoted. (3) It is a valuable guide to the suitability for delegation of responsibility for reporting. We can see that a comparable system may have advantages in the clinical field.—We are, etc.,

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1 Royal College of Pathologists of Australia, Board of Education. *Report of Surveys:* 1969, 1970, 1971. Sydney, R.C.P.A.
2 Penner, D. W., in *Pathology Annual*, vol. 8, ed. S. C. Sommers, p. 1. New York, Appleton-Century-Crofts, 1973.

Disclosure of Medical Records

SIR,—Since August 1971 the Rules of the Supreme Court have provided (Order 24 Rule 7A made under the Administration of Justice Act 1970) that before commencing proceedings a potential plaintiff in a personal injury case may apply to the court for an order requiring a medical practitioner who is a potential defendant to disclose his clinical records. Similarly, once proceedings have been started to which a medical practitioner is not a party (for example, a factory accident case) a medical practitioner who would be a material witness in the case can be required by order of the court to disclose his clinical records at an early stage in the proceedings rather than delaying this until he is in the witness box. It is worth pointing out in general terms that an order of the court is not made automatically and that if the practitioner chooses not to submit his records voluntarily (as is his absolute right) the court will consider the merits of each application and restrict the disclosure of records to that which justice requires.

This subject has been dealt with in detail on more than one occasion in the medico-legal columns of your journal[1-3] as well as in a letter from Dr. P. H. Addison, past Secretary of the Medical Defence Union,[4] and there is no need to go over the same ground again. What we wish to do now is to bring to the notice of members of the profession a particular aspect of the problem.

Two recent cases[5 6] heard by the Court of Appeal and supported by the M.D.U. have established that when a court orders disclosure of medical records they shall normally be produced only to another medical practitioner, acting as medical adviser to the party that obtained the order for disclosure, and not to solicitors. A practitioner's records may well include letters written to him by another practitioner—for example, a general practitioner may have had letters from a hospital consultant about the patient. The precise description of the documents the production of which may be ordered will be set out in the order, but it should be assumed that when the order specifies "all the medical records of Dr. X relating to . . ." this will include not only the practitioner's own notes but all consultants' letters and other clinical documents which are relevant to the case.

Our purpose is to point out to all practitioners in the United Kingdom that letters to other doctors about patients should always be written in the knowledge that they may be subject to detailed scrutiny by other practitioners prior to any court hearing, as well as by the judge and lawyers when the case gets to court; and that accordingly their tone should be serious and precise, even though this may mean the loss of the "personal touches" which have in the past lightened correspondence between colleagues.—We are, etc.,

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J. LEAHY TAYLOR
Secretary,
Medical Protection Society

J. W. BROOKE BARNETT
Secretary,
Medical Defence Union
London W.1

1 *British Medical Journal*, 1972, **1**, 577.
2 *British Medical Journal*, 1973, **1**, 623.
3 *British Medical Journal*, 1974, **1**, 652.
4 Addison, P. H., *British Medical Journal*, 1972, **1**, 565.
5 Davidson *v.* Lloyd Aircraft Services Ltd., *The Times*, 15 May 1974, p. 20.
6 Deistung *v.* South West Metropolitan R.H.B., *The Times*, 26 October 1974, p. 24.

Retinitis Pigmentosa

SIR,—The leading article on this subject (17 August, p. 429) is misleading with regard to the genetic advice to be given to a family with affected members and also the visual prognosis to be given to an affected individual.

You state that the disease is usually transmitted as a recessive condition without differentiating between X-linked and autosomal recessive disease. In a family with X-linked disease the chances of affected individuals appearing in future generations is high, while in autosomal recessive disease it is low if cousin marriages are avoided. This differentiation is particularly relevant when a heterozygote seeks advice. You correctly point out that heterozygotes for the X-linked gene (female carriers) show some phenotypic expression of the abnormal gene by early adult life,[1 2] but it should also be emphasized that heterozygotes for the autosomal recessive gene rarely if ever have recognizable ocular changes. Therefore these two forms of the disease, which are equally common in south-east England,[2 3] must be distinguished one from another before genetic advice is given.

Your statement that retinitis pigmentosa, once recognized, leads to blindness within a few years is quite wrong. There is no doubt that patients with severe recessive forms of the disease may be blind in early life, though such cases are rare. Even males with X-linked disease who notice loss of dark adaptation in the first decade of life are not severely handicapped until the third decade and may retain some useful vision until they are 50 or 60 years old. Autosomal dominant retinitis pigmentosa, which represents 25% of all cases in our practice, is mild and may give rise to little disability even in late life.[2] —We are, etc.,

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1 Bird. A. C., and Blach, R. K., *Transactions of the Ophthalmological Societies of the U.K.*, 1970, **90**, 127.
2 Krill, A. E., *American Journal of Ophthalmology*, 1967, **64**, 1029.
3 Jay, B., and Bird, A. D., *Transactions of the American Academy of Ophthalmology Otolaryngology*, 1973, **77**, Op.—641.

** We did not expand on the various patterns of retinitis pigmentosa inheritance as this was not the aspect with which our article was primarily concerned, but we deemed it sufficient to refer readers to a recent genetic analysis—indeed, by the authors of this letter. We said that retinitis pigmentosa "usually . . . leads to blindness within a few years." We readily accept these authors' findings that this was unduly pessimistic.—ED., *B.M.J.*

Diagnostic Test for Multiple Sclerosis

SIR,—The degree of inhibition by linoleic acid of the response of human lymphocytes to antigens has been claimed by Field *et al.*[1] to be much greater in patients with multiple sclerosis (M.S.) than in other neurological disorders and could be used as a diagnostic test. Mertin *et al.*[2] failed to confirm that the test was diagnostically useful in double-blind trials on M.S. patients selected according to the criteria of Allison and Miller (see McAlpine *et al.*[3]). Without wishing to take sides we would like to draw attention to a new factor which we believe should be taken into account in patient selection if an effect of linoleic acid is to be tested on the patients' macrophages.

Low blood linoleate levels were demonstrated in 1966[4] in patients with M.S.; early in 1973 Millar *et al.*[5] published evidence that sunflower seed oil might act as a remission agent. At that time we were including M.S. patients in a study of blood fatty acids. However, considerable press and television publicity was given to these findings of Millar *et al.* and by the autumn of 1973 most of the M.S. blood samples we

analysed were remarkably rich in linoleic acid (see table), a finding which was explained by the fact that these patients, mostly on their own initiative, were taking four to six spoonfuls of sunflower seed oil a day; about 65% of the fatty acids of sunflower seed oil is linoleic acid. The trials by Mertin *et al.*[2] were carried out between October and December 1973.

Mean Linoleate Content (as %w/w of Total Fatty Acids ± S.E.M.) Fats in Plasma of Normal Subjects and Patients with M.S.

	Normal	M.S.1.	M.S.2.
Phospholipid	17·6±1·64 (n=11)	13·7±1·6 (n=7)	26·9±1·1 (n=12)
Triglyceride	11·0±0·8 (n=14)	7·86±0·71 (n=6)	26·8±3·1 (n=12)

M.S.1 refers to patients who claimed they were not taking sunflower seed oil. The M.S.2 group represents those patients who had been taking sunflower seed oil for at least one month (data from 1973).

Medication with sunflower seed oil can clearly be expected to increase the plasma linoleate. The macrophage electrophoretic mobility test is unlikely to give a high degree of inhibition by linoleic acid if the macrophages have been taken from blood which is rich in linoleic acid itself. Perhaps the only way to resolve the issue would be to relate electrophoretic mobility to plasma linoleate content.—We are, etc.,

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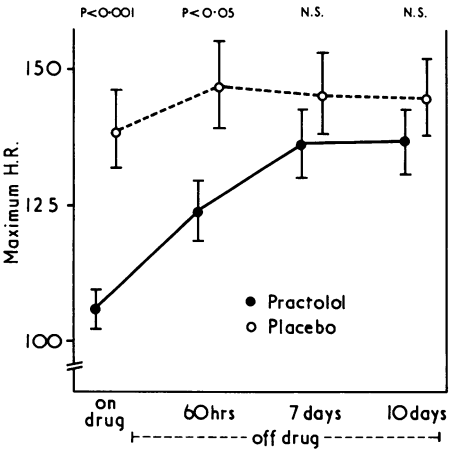
1 Field, E. J., Shenton, B. K., and Joyce, G., *British Medical Journal*, 1974, **1**, 412.
2 Mertin, J., *et al.*, *British Medical Journal*, 1974, **4**, 567.
3 McAlpine, D., Lumsden, C. E., and Acheson, E. D., *Multiple Sclerosis: A Reappraisal*. Edinburgh and London, Churchill Livingstone, 1972.
4 Baker, R. W. R., Thompson, R. H. S., and Zilkha, K. J., *Journal of Neurology, Neurosurgery, and Psychiatry*, 1966, **29**, 95.
5 Millar, J. H. D., *et al.*, *British Medical Journal*, 1973, **1**, 765.

Duration of Action of Practolol

SIR,—A current problem in cardiac surgery is to decide how long before surgery beta-blocking drugs should be stopped. As little information is available on the duration of action of practolol we have tried to assess this, using an exercise-induced tachycardia test, in a group of 19 patients who had completed a two-year period of study of the value of practolol after acute myocardial infarction. All patients had had definite acute infarction and were randomized into either a practolol (400 mg a day) or a placebo group. Exercise tests were performed on a stationary bicycle before and 60 hours, seven days, and 10 days after the tablets were stopped. The load was increased by 25 watts every minute starting from 50 watts and reaching 150 watts by the fifth minute.

There was no significant difference in the resting heart rates of the 11 patients who had received practolol and the eight who had received placebo (see fig.). There was a significant difference between the practolol and placebo patients while on the tablets and at 60 hours after cessation but no difference by the seventh day.

These results are very similar to those reported by Carruthers *et al.*,[1] who showed



Maximum heart rate (mean ±S.E.) after 5 minutes' exercise on 11 patients who had taken practolol and eight who had taken placebo.

that though the plasma half life of practolol was about 10-11 hours the pharmacological half life was of the order of 40-50 hours. It would seem reasonable therefore to stop practolol for at least three days before cardiac surgery to allow full recovery from its effects.—We are, etc.,

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1 Carruthers, S. G., *et al.*, *British Medical Journal*, 1973, **2**, 177.

Drug Combinations for Anaesthesia

SIR,—In a recent issue of the *B.M.J.* (9 November 1974) there were two reports concerning the use of methohexitone and diazepam in combination to produce anaesthesia. One was a coroner's report (p. 352) of a fatality occurring in a previously healthy patient in a dental chair. The other was a letter (p. 345) in which Dr. M. W. P. Hudson defended the use of this combination, which he used routinely to produce conditions suitable for intubation. He did stress that the patient should be horizontal at the time of injection and must remain so for at least one hour afterwards to avoid hypotension.

Any combination of drugs which produces conditions suitable for intubation is unsuitable for use in dental practice except by operators prepared to pass an endotracheal tube. During the inquest a dental surgeon with considerable experience of dental anaesthesia stated in court that "they [dental surgeons] were not trained in it [intubation]," and that "it was not a technique for dental surgery."

Dr. Hudson may be correct in his assertion that methohexitone and diazepam are a most valuable combination of drugs, but on the evidence he has presented we believe it can only be described as hazardous where the relevant apparatus and expertise are not immediately available.—We are, etc.,

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SIR,—There has been considerable recent controversy over the combination of methohexitone and diazepam as a possible cause of death in several of the recent dental anaesthetic fatalities. It is interesting to note that Dr. M. W. P. Hudson (9 November, p. 345) has been using this technique prior to intubation, the diazepam replacing suxamethonium as the relaxant drug prior to the placement of the tube. Naturally there must be considerable respiratory depression caused by the combination of these two drugs, with the possibility of stagnant anoxia, and it seems quite obvious that the reason Dr. Hudson, in over two years' safe usage of this technique, has had no problems is that the patient is immediately adequately oxygenated when inhalational agents are added.

I have personally used methohexitone and diazepam together (without intubation) for a considerable time. With a light sleep dose of the former, the slow additional injection of diazepam (maximum dose 10 mg), the immediate application of a 75/25% nitrous oxide/oxygen mixture, and correct attention to the airway I have seen no problem that has given me a moment's worry. The nitrous oxide, of course, provides the analgesia that the methohexitone, a hypnotic, does not.

As long ago as 1965, after some 6000 cases, I was one of the first dental critics of the intermittent methohexitone technique (which is in any case far from ideal for the majority of procedures) and it was pointed out at that stage that the smoothest and safest anaesthetic, despite the odd case of post-anaesthetic nausea, was methohexitone followed by a nitrous oxide/oxygen mixture. Additional increments of the drug were injected as necessary for operations of a maximum of 20 minutes' duration. Considerable respiratory depression can take place with methohexitone alone; but with the undoubted synergistic effect produced by the methohexitone combination it is imperative that oxygen be administered throughout all such anaesthetics (coupled, as stressed, with constant attention to the patency of the airway through correct mouthpacking technique and adequate suction).

The problem simply was that in each case which proved fatal inadequate oxygen had been reaching the brain due to either asphyxial or stagnant anoxia. Certainly from the facts one can assume that the Society for the Advancement of Anaesthesia in Dentistry, though naturally advocating that an oxygen machine should be present in working order in or near the surgery, do not advocate its use at all times during anaesthesia. By using a simple Goldman nosepiece secured by a McKesson harness the patency of the airway can at all times be appreciated owing to the equal inspiratory and expiratory sounds easily heard through the nosepiece valve. Any interruption of this sound (for example, a continuous gas flow) immediately signifies that normal breathing is not taking place and that there is some obstruction. This can then be speedily dealt with by changing or at least altering the position as necessary of mouthpacks, good suction, and/or forward movement of the mandible.

Dr. J. G. Bourne in the late 1960s emphasized the importance of the supine position to help prevent fainting before, during, or after an anaesthetic, this being a major breakthrough in general anaesthetic