of partial rhizotomies, therefore, should be examined to see whether in fact it is a mixture of the partial and the complete operations; if it is, its recurrence rate will be less than the true rate for partial rhizotomy. Of the only two series quoted in this connexion by Messrs. Clarke and Hankinson, both were mixtures in unspecified proportions. Olivecrona,[1] it is true, spared the ophthalmic division "in most cases," and so his recurrence rate for partial section, if he had given it, might not have greatly exceeded his 8.5% for all cases. Leriche[6] stated that *neurotomies électives* could be done "*si on le veut*," but not how many he had done. He had seen eight recurrences in "*quelques 250 cas*." Having no definite denominator, he quite properly gave no recurrence rate, and the "3%" ascribed to him by Messrs. Clarke and Hankinson is not in his book. In the two large series to which I have referred for the death rate,[7,9] the relapse rates for partial rhizotomy were 7.5% and 8.5%.

Injection of the trigeminal sensory root is bound to have some mortality. For example, a patient of mine died of pulmonary embolism one midday. If she had done so at any time between a few hours and a few weeks later her death would have been attributable to the injection which she was to receive that afternoon. This mortality, because of its very smallness, has not been satisfactorily estimated, but it is certainly well below a tenth of that from subtemporal rhizotomy (although the frailer or older the patient the less likely to be rhizotomized and the more likely to be injected). Mr. Taylor's "no mortality" is an inaccuracy, but, as the Red Queen would doubtless put it, *I've* seen inaccuracies, compared with which this would be a precision.—I am, etc.,

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REFERENCES

1 Olivecrona, H., *Acta psychiat. scand.*, 1947, Suppl. No. 46, p. 268.
2 Rowbotham, G. F., *Lancet*, 1954, **1**, 796.
3 Frazier, C. H., *Arch. Neurol. Psychiat. (Chic.)*, 1925, **13**, 378.
4 Horrax, G., and Poppen, J. L., *Surg. Gynec. Obstet.*, 1935, **61**, 394.
5 Cushing, H., *J. Amer. med. Ass.*, 1920, **75**, 441.
6 Leriche, R., *La Chirurgie de la Douleur*, 1949, 3rd ed. Paris.
7 Grant, F. C., *Res. Publ. Ass. nerv. ment. Dis.*, 1943, **23**, 408.
8 Peet, M. M., and Schneider, R. C., *J. Neurosurg.*, 1952, **9**, 367.
9 Grant, F. C., *Ann. Surg.*, 1938, **107**, 14.

Difficulties of Doctors in Industry

SIR,—On 4 October 1968 Mr. Justice Swanwick delivered judgement in the High Court in the case of Stokes v. G.K.N. Bolts and Nuts Ltd. The action was brought by the widow of Mr. Stokes on the grounds that his death on 13 February 1966 was caused by epithelioma of the scrotum due to exposure to mineral oil whilst in the employ of the defendants when he was working as a machine toolsetter. Damages of £10,000 were awarded to Mrs. Stokes. Leave to appeal was granted to G.K.N., who after careful consideration, and not without consultation with the Medical Defence Union, decided against further action. I ceased medical supervision of G.K.N. Bolts and Nuts Ltd. in March 1965, but continued as chief medical officer to Guest, Keen and Nettlefolds Ltd. until the end of 1966, and since then acted as part-time consultant to the company's London office.

I am not using your columns to complain against the comments of the learned judge, who held me to be negligent on two counts: firstly, that I did not arrange for sufficient warning of the employees after the death of another man, Ward, on 20 July 1963 from an epithelioma of the scrotum; and secondly that after Ward's death, the first case I had discovered during 20 years of supervision of the health of 10,000 people, I had failed to carry out six-monthly inspection of the scrota of all employees exposed to mineral oil. I cannot use your space to go further into the case, but I have listed some points which have significance to doctors in general, and to those employed in giving advice to industry.

It is a legal duty for a doctor seeing a case of epithelioma of the scrotum, if there has been exposure to mineral oil, to report the case to the Chief Inspector of Factories on Form 303. The disease is notifiable. It is the doctor's moral responsibility to tell the man that he can claim under the Industrial Injuries Act.

Where a doctor advises a company and a case of epithelioma of the scrotum occurs involving exposure to mineral oil the doctor would be well advised to inform the employer in writing of the company's duty to notify the occurrence to the local inspector of factories and to the appointed factory doctor.

Any action the doctor intends to take about prevention is better put in writing to the Factory Inspectorate.

Make sure that all the statutory notices concerning any hazard are displayed and maintained. This duty devolves upon the factory occupier, who is also responsible for the distribution of individual leaflets. Where a factory uses mineral oil it is statutory to display the cautionary notice "Effects of Mineral Oil on the Skin," form S.W.W. 397, price 6d. The individual leaflet "Effects on the Skin of Mineral Oil," S.H.W. 295, must be distributed to every worker subject to exposure. These leaflets are a free issue, but experience has shown that until recently they were in very short supply. A further point to be remembered is the language barrier, since many non-English speaking immigrants are employed in such factories.

The court was more impressed with the literature than with personal experience, although mine was of 27 years' standing.

The judge ruled that everybody in contact with mineral oil must have his scrotum examined by a doctor at six-monthly intervals, and presumably this must continue after he leaves employment. Garage hands, for example, would fall into this category.

There are no statutory regulations about routine inspections. Some advice from the factory department is needed on this point. When do we start them? Has anybody realized what medical manpower is involved? Who has pronounced that these can stop the incidence of the disease, which at the time I had to make a decision after the discovery of the case of Ward was 23 cases in 1961? There must be some 10 million people exposed to mineral oil in this country. Incidentally I have found that many men (and educated ones) do not know what the scrotum is. Why not drop secrecy and put "private parts" on the official notices?

Far more research is needed in the oil industry on the dangers of routine medical inspections creating a false sense of security, and on types of guarding and individual protection. I believe we should be doing a disservice to medicine if we take panic measures using valuable medical man hours, when I believe that the answer is personal cleanliness.

Finally, I would like to recognize the backing given to me by my former company, and by those on the shop floor, whose interests matter above everything else.—I am, etc.,

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Frusemide and Urinary Cell Loss

SIR,—We were interested to read the paper by Dr. A. E. Gent and his colleagues (2 November, p. 294) on the effect of frusemide and other substances on urinary cell excretion.

These authors suggest that the increased urinary white cell excretion which we reported[1] in patients who had undergone surgery might be explained by administration of such drugs before or after operation. We do not think this is likely, for several reasons. Firstly, the increased white cell excretion we demonstrated was confined to the female sex, and one would not expect them to have enjoyed a monopoly in drug administration. Second, there was a correlation between increased postoperative white cell excretion and previous history of urinary tract infection. Finally, the increase in white cell excretion described by Dr. Gent after frusemide was about 25,000 white cells per hour; the increases we recorded were of the order of several hundred thousand cells per hour.—We are, etc.,

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REFERENCE

1 Lindsay, R. M., Lunan, B., McGeachie, J., and Linton, A. L., *Brit. med. J.*, 1967, **3**, 83.

SIR,—I was very interested to read the article by Dr. A. E. Gent and others (2 November, p. 294) concerning the effects of frusemide, lactose, and urea on urinary cell loss. I have also studied urinary cell excretion before and after treatment with frusemide and chlorothiazide. During a 40-minute period following the intravenous administration of 20 mg. frusemide, or 500 mg. chlorothiazide, there was a several-fold increase in the excretion rate of red blood cells, renal tubular cells, and leucocytes in three of four normal volunteers.

There seems little doubt that treatment with frusemide can result in increased excretion of renal tubular cells. This response is probably due largely to an increased rate of urine flow, as the authors suggest. I have previously reported a statistically significant correlation between urine flow rate and the excretion of red blood cells and renal tubular cells in healthy adults.[1] Thus during the normal day-to-day variation in urine-flow rates, the excretion of both cell types at a flow of 1.0 ml./minute was more than double that observed at 0.7 ml./minute. The toxicological significance of minor changes in renal

tubular cell excretion during the administration of drugs affecting rate of urine flow is therefore difficult to assess in the absence of other demonstrable nephrotoxic effects.

Dr. Gent and his colleagues suggest that the effect of caffeine on renal tubular cell excretion is due to its diuretic action. This does not seem to be the case in the study referred to,[2] since the mean urine flow rate during the control period was 45.8 ± 5.4 (S.E.) ml./minute, compared with 44.2 ± 3.4 ml./minute during treatment with caffeine. Moreover, renal tubular necrosis associated with markedly increased renal tubular cell excretion is known to occur in rabbits and dogs receiving repeated daily doses of caffeine. These changes were observed in dogs treated with caffeine in doses of only 40 mg./kg. daily.[3]

I do not think that the effects of frusemide on urinary cell excretion can be compared with those of analgesic drugs such as salicylates for several reasons.

1. There was no significant change in the rate of urine flow when 3.6 g. of aspirin was given daily.

2. The renal tubular cell response to this dose of salicylate was very much greater than that observed with frusemide. Thus the five-day mean tubular cell counts rose by a factor of 10 when aspirin was given, whereas only a threefold increase was noted with frusemide. In one volunteer receiving aspirin the excretion of renal tubular cells rose to 8,192,000 cells an hour—a 120-fold increase.

3. The renal tubular cell response to aspirin follows a characteristic pattern. After a latent interval there is a sudden dramatic rise, followed by a slower fall toward normal values, even though the drug is continued. An identical response with a dose-related latent period occurs in rats treated with mercuric chloride in doses sufficient to produce tubular necrosis. The sudden rise in cell excretion coincides with widespread tubular necrosis, and the slow fall is associated with lesser degrees of necrosis and rapid regeneration, despite the continued administration of mercury.[4] This typical pattern of response did not occur when frusemide was given, though the effects of larger doses are unknown. The fact that frusemide produced an immediate response is more in keeping with a mechanism related to the diuretic effects of this drug.

4. I do not know whether frusemide can cause tubular necrosis, but there is no doubt that such lesions can be produced in experimental animals treated with caffeine or salicylates. Renal tubular cell excretion does not seem to be increased following therapeutic doses of probenecid, penicillin, alpha-methyldopa, or cephaloridine (2 g. daily). On the other hand, large numbers of renal tubular cells have been observed in the urine of patients receiving potentially nephrotoxic antibiotics such as bacitracin, kanamycin, and colistin methanesulphonate.

As Dr. Gent points out, there are many problems still unsolved, and it is hoped that further studies will clarify the situation. Dr. S. T. G. Butterworth (23 November, p. 517) suggested that some of the small peroxidase-negative cells in the urine might be lymphocytes rather than tubular cells. This might very well be the case, and lymphocytes have been described in the urine during the rejection of renal transplants. However, the cells of the urinary sediment are often degenerate, and conclusive identification becomes very difficult with a routine method. Nevertheless, it is impossible to avoid the conclusion that the masses of peroxidase-negative cells appearing in the urine during experimentally induced tubular necrosis are derived from the necrotic tubules.

I am glad that Dr. Butterworth has drawn attention to the possible carcinogenicity of the 2:7 diaminofluorene used in our stain. I was not aware of this hazard when the original method was published, and precautions to avoid contact with the stain must be taken.—I am, etc.,

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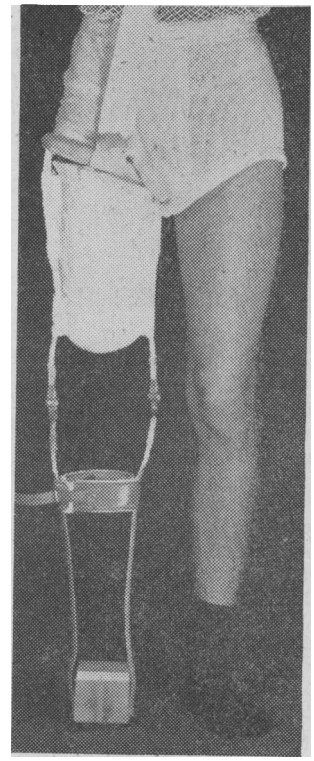
REFERENCES
1 Prescott, L. F., *Clin. Sci.*, 1966, **31**, 425.
2 Prescott, L. F., *Lancet*, 1965, **2**, 91.
3 Vinci, G., *Arch. ital. Biol.*, 1914, **61**, 401.
4 Prescott, L. F., and Ansari, S., *Toxicol. appl. Pharmacol.*, 1969, **14**, 97. In press.

Through-knee Amputation

SIR,—The article by Mr. P. F. Early (16 November, p. 418) on through-knee amputation shows that this technique is becoming more recognized as advantageous, though its use is still limited by the shortcomings of the artificial limb makers. It is, of course, true that in traumatic amputations the precious skin around the knee is too often mangled, or is cut too short by inexperienced emergency surgeons, so that proper cover of the femoral condyles is not achieved. At the same time, in the case of ischaemic limbs the flaps are likely to be ischaemic too, so that only a small proportion of people can enjoy this stump. It seems a pity, therefore, that more attention is not devoted to the provision of the appropriate limb. A friction-loaded knee



joint, placed 1 in. (2.5 cm.) below the condyles, should not really cause a great deal of mechanical or cosmetic disturbance, in spite of the difference in the level of the knee joints. However, should this indeed be an insuperable obstacle, it is a very simple matter to shorten the femur by 2 in. (5 cm.) by an oblique resection in the upper middle third, immobilizing the femur with an adequately thick Küntscher nail. With the obliquity of the resection controlling rotation, it is possible for such a patient to start walking within a week of operation. The elevation of the condyles by 2 in. (5 cm.) does away with all the prosthetic objections.

One of the universal complaints about limb makers is the unconscionable delay between the amputation and the fitting of the limb. Under the modern development of applying in the theatre a plaster stump sock, delay even in the supply of a pylon is very irritating. It is certainly not a common experience to find that pylons are supplied in a few days, as suggested in point (3) at the end of Mr. Early's discussion. The picture suggests a method of getting round this problem, using a preoperatively supplied walking calliper with a locking knee hinge. Weight-bearing is shared with the calliper ring and it is important to ask for heavy-duty side bars to the calliper.—I am, etc.,

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Systolic Murmurs in the Elderly

SIR,—May I clarify a possibly misleading reference to one of my papers in the leading article on systolic murmurs in the elderly (30 November, p. 530)?

The findings in fact confirmed that aortic valve or ring abnormalities were present in most of the cases with clinically definite aortic murmurs. However, these were only 37 of the 173 patients studied, and in the remainder the examining physicians had not considered the murmurs as having definite aortic characteristics. Such cases are, however, still commonly diagnosed as "aortic sclerosis," but apparently more from traditional habit than clinical findings. It was in the latter group only that the high proportion of mitral abnormalities was observed, and to which the conclusion in your leading article refers.—I am, etc.,

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Screening for Cervical Cancer

SIR,—Dr. A. J. Lucas and others (30 November, p. 578) make a timely point in drawing attention to the payment of general practitioners for the taking of cervical smears only from women over 35 years of age.

In the north-east region of Scotland the general practitioners have responded well to the advice to take cervical smears from their healthy women patients. In 1962 only 3% (292) of cervical smears were taken by practitioners, whereas in 1967 40% (6,044) of the smears coming into the laboratory from women screened for the first time were from general practitioners. Fortunately these doctors do not appear to be limiting their smear-taking to the group for whom they will receive payment. This makes the attitude of the Ministry unreasonable and the screening commendable. The postal service initiated by the Ministry is most helpful.

Since our screening programme started unsuspected preclinical cases of cervical cancer have been detected at a rate of 0.3% below 30 years of age, but 0.81% between 30 and 35 years, and 0.85% between 35 and 40 years. Perhaps there was a case for