Mr. Bevan Confuted

SIR,—Two years ago, in the early spring of 1946, I attended a meeting of practitioners of the Birmingham area at the Queen Elizabeth Hospital; there were about 600 present, and Dr. Dain was in the chair. I painted a picture of the political career of Aneurin Bevan, and I put forward a resolution worded thus, "That in the opinion of this meeting negotiations with the present Minister of Health are not possible." Dr. Dain was on his feet immediately; he remonstrated with me for proposing this, and he pointed out that if this resolution was carried it would embarrass him and other members of the Negotiating Committee who were on the platform. He moved that we pass on to the next business, and this was done. I am not writing this to prove how right I was in 1946 but because I feel very strongly that Dr. Dain's action that night gives the lie to the accusations that the Minister made about the B.M.A. "clique" in the Commons on Feb. 9.—I am, etc.,

Dudley, Worcs.

L. H. G. MOORE.

No Certification

SIR,—May I endorse the suggestion put forward by correspondents in your issue of Feb. 7 that in the event of a majority against entering the Service in its present form we do not wait for the appointed day but take immediate action by refusal of certification? Such action would bring matters to a head and necessitate amendment of the Act before the appointed day. This would nullify the Minister's main weapon—that of refusing compensation to doctors refusing to enter the Service.—I am, etc.,

Edinburgh.

G. S. M. WILSON.

SIR,—I agree with the various correspondents who have written to the *Journal* recently expressing the opinion that action should be taken at once before the National Health scheme becomes operative in July. I suggest that the Association should organize a scheme for the non-issue of medical certificates commencing, say, in April rather than leave the matter until concerted action may be too late. I understand that many doctors are in agreement with some such policy and only require a lead from some organization such as you represent.—I am, etc.,

Dartford, Kent.

F. S. D. THOMPSON.

Test of Death

SIR,—The interesting phenomenon of segmentation or "railway truck" appearance of the blood column visible in the retinal vessels occurs when the blood flow is sufficiently reduced either in death, on account of the absence of the cardiac impulse, or in obstruction of the retinal artery from various causes. In the latter type of case the condition is temporary. This segmentation was noted by Bouchut in 1874—possibly before then—and has been reported several times since (Kahn, 1913; Salisbury and Melvin, 1936), often as a new observation. Presumably the same process occurs in other parts of the body under similar conditions.—I am, etc.,

Edinburgh.

H. M. TRAQUAIR.

REFERENCES

- Bouchut, M. E. (1874). *Gaz. Hôp., Paris*, 225.
Kahn, M. H. (1913). *Med. Rec., N.Y.*, 83, 801.
Salisbury, C. R., and Melvin, G. S. (1936). *British Medical Journal*, 1, 1249.

SIR,—Under this title Dr. E. A. Harris (Jan. 31, p. 226) describes a condition of segmentation of the blood column in the retinal vessels after death and says he is not aware that the sign had been described.

Probably the most complete account of this condition was given by C. H. Usher, of Aberdeen, in the *Ophthalmological Review* (1896, vol. 15, p. 339), where observations on the appearance of the retinal vessels in man, monkey, cat, and rabbit, just before, at, and just after death, are fully detailed.

A brief history of the subject is given also by E. Wolff and Francis Davies in the *Transactions of the Ophthalmological Society of the United Kingdom* (1928, vol. 48, p. 143), where the credit for first describing the condition is given to Bouchut in 1869. These writers, however, describe with illustrations a similar segmentation produced and abolished at will in the

living animal. They conclude that segmentation in the retinal vessels is not necessarily a sign of death. "Granted that in the large majority of cases it is a sign of death," the writers add. "we would suggest there must be exceptions."—I am, etc.,

The University, Aberdeen.

ROBERT RICHARDS.

SIR,—Bouchut in 1869 was the first to describe fragmentation of the blood stream and since then it has generally been looked on as a sign of death. In 1928, however, Francis Davies and I (*Trans. Ophthal. Soc. U.K.*, 1928, 48, 143) showed that by clamping the vessels to the head and neck in the cat we could produce and, by removing the clamps, abolish this phenomenon at will. I hope this answers Dr. F. Parkes Weber's query (Feb. 14, p. 315) as to what would happen if the heart was successfully induced to contract normally again. Fragmentation of the blood stream is thus a reversible phenomenon, and though it is a presumptive sign of death cannot be regarded as an absolute one. We suggested that the fragmentation was due to an aggregation of the red corpuscles—that is, a rouleaux formation such as occurs in shed blood. This is made possible by the slowing of the blood stream.—I am, etc.,

London, W.1.

EUGENE WOLFF.

Canine Hysteria and Agenized Flour

SIR,—We have recently had the opportunity for the first time of being present when Sir Edward Mellanby exhibited his film entitled "Canine Hysteria." In view of the prominence which has been given in the medical press (leading article *British Medical Journal*, 1946, 2, 903; *Lancet*, 1948, 1, 27) to Sir Edward's reports on the role of agenized flour and wheat products in the causation of canine hysteria (*British Medical Journal*, 1946, 2, 885; *Ibid.*, 1947, 2, 288) and the widespread deductions concerning the importance of agenized flours in the aetiology of canine hysteria which have been based on these reports, we feel that as clinicians closely concerned with canine disorders in our professional duties the attention of your readers should be drawn to certain matters relating to the diagnosis.

1. In several cases shown in the film the clinical signs were not typical of the condition which we have recognized clinically as canine hysteria for many years.

2. The disorder was not similar to the form of canine hysteria that we have observed in the London area and the provinces during the war years, when it has been markedly less common than formerly.

3. In particular, certain animals appeared to exhibit neurological signs, such as severe clonic choreic movements of the jaws and head, complete loss of vision, incoordination of gait, and hyperflexion of the hind limbs, strongly suggestive of localized nervous defects which are more usually associated with the infective encephalomyelitis and are certainly not seen in natural cases of uncomplicated canine hysteria. Furthermore, in the naturally occurring disease the severe epileptiform convulsion is not a common feature.

If the film is truly representative of the syndrome that Sir Edward observed—and we can only suppose that it is so—then there would appear to be grave doubts as to the precise nature of the disorder that Sir Edward was observing, and indeed whether it was in fact a single aetiological entity.

While this possibility does not detract from the interest in the action of the agene process on wheat proteins we would suggest that the term "canine hysteria" should not be applied to this condition until the disorder has been investigated more fully. For the present, therefore, perhaps the term "agene intoxication" might be a more suitable diagnosis for the disorder produced by feeding agenized proteins to dogs.—We are, etc.,

M. HARVEY CLARK.

HERBERT PARRY.

JOAN O. JOSHUA.

OLGA UVAROV.

S. F. J. HODGMAN.

Newmarket, Suffolk.

Curare in Spastic Conditions

SIR,—I read with interest the article on the above subject by Drs. C. Astley Clarke and R. D. Hotston (Feb. 14, p. 289).

In May, 1947, I treated with the same oily drug a woman aged 61, suffering from paralysis agitans of over four years' duration. There was generalized muscular rigidity in flexed position, the head and neck were moving together, the face was a typical Parkinsonian

mask with wrinkled forehead, there were tremors of the hands, the right worse than the left. She was walking with short shuffling steps, holding on to furniture indoors, and with a stick and the assistance of her daughter out of doors. Pro-, retro-, and lateropulsion were marked. She suffered from insomnia, and had great difficulty in turning over in bed. Her deep reflexes were increased. She was of high intelligence and spoke with a monotonous voice.

I began with intramuscular injections of 15 mg. of *d*-tubocurarine chloride in oil (30 mg. in 1 ml.), and worked up to 45 mg. On 15 mg. she had no reaction, apart from slight weakness which lasted for a day or two. On 45 mg. she had very severe side-effects, lasting up to seven days, when she was confined to bed, unable to get up owing to weakness, with a feeling of impending death, after which her condition was exactly as before the injection. On 30 mg. there was at times no reaction, but at others it was as severe as that obtained with 45 mg. The injections were always given in the same manner and into the same muscle. When the reaction was favourable (usually on 30 mg.) the clinical picture was as follows.

Four to five hours after injection her face became relaxed, she developed a bilateral ptosis and diplopia and a feeling of drunkenness which lasted from thirty minutes to two hours. Gradually a generalized weakness developed in all her limbs, which made it difficult for her to walk about even with assistance, so that she had to be confined to bed. She was off food, very drowsy, and slept well, and felt dispirited. This usually lasted for two days. At the end of this time her strength gradually returned and her walking improved. She felt better, started eating, and on the fourth day after injection she walked without support both in the house and out of doors, much to the amazement and wonder of her neighbours, who had not seen her walk without assistance for over four years. The pro-, retro- and lateropulsion phenomena were absent. I actually pushed her in various directions while she was walking and she did not lose her balance. She could turn over in bed with ease. There was, however, no change in her tremors. She was so delighted with herself that she walked about all day just for the sake of using her legs, like a child who for the first time discovers that it can perform some action and keeps on repeating it. This improvement lasted two to seven days.

I used the drug for three months, and at the end of that time the patient, although she had derived great benefit at times, refused the treatment, owing to the occasional severe side-effects.

I also treated a woman of 40 who had a residual right hemiplegia of four years' duration. Apart from walking better on the third day after injection, there was no change. Her side-effects were not as severe as those of the woman with Parkinsonism.

In both these cases my experience did not quite coincide with the findings of Drs. Clarke and Hotston in the following respects. (1) There was no diminution of side-effects after repeated injections. (2) The side-effects usually started within four to five hours, with one exception where severe reaction occurred in the Parkinsonian case within ten minutes. She developed bilateral ptosis and collapsed, remaining in that state for over two hours. (3) Both the unpleasant reactions and the beneficial effects lasted for days rather than hours. (4) Finally, I was impressed with the apparent unstableness of the drug, and with the unevenness of the reactions in intensity and duration both in the unpleasant and the beneficial effects obtained from an identical dosage, bottle, and technique.—I am, etc.,

Sheffield.

I. GOTTLIEB.

Louse-borne Relapsing Fever in Persia

SIR,—Drs. R. I. Bodman and I. S. Stewart (Feb. 14, p. 291) state that louse-borne relapsing fever has not previously been described in Persia. Actually louse-borne relapsing fever was described in Persia after the war of 1914–18 and the epidemic occurred about the same time as the then epidemic of typhus, also louse-borne. It is more than a coincidence that the first and second World Wars were followed by epidemic typhus and relapsing fever in that particularly lousy quarter of the globe. There was an epidemic of louse-borne relapsing fever in Shuster in 1942, which was diagnosed by my assistant Ali Hassan Assidi from blood films sent to Fields and later forwarded to me for confirmation.

In the case of tick-borne relapsing fever the tick—a species of *Ornithodoros*—lives in the dusty floors and cracked walls of mud dwellings and not on the person. Ticks are common enough in Abadan. The older medical missionaries—such as Dr. Shafter, of Ispahan—must have seen many cases of louse-borne relapsing fever in addition to the more common “Mianeh fever” the usual vector for which is *O. papillipes*.

Prof. Charles Oberling—author of the *Riddle of Cancer*—found cases of tick-borne relapsing fever in Tehran about 1942.

Generally the infective forms are numerous in blood films from louse-borne cases and are very scanty indeed in preparations from patients with the tick-borne disease. Blood films carefully stained with Leishman or Giemsa were adequate for diagnosis. It would be interesting to try the effect of penicillin on some of these cases.—I am, etc.,

Epping, Essex.

FRANK MARSH.

Injection Errors

SIR,—Mr. Harold Dodd (Feb. 14, p. 320) raises some interesting points regarding safety measures to be adopted with injection preparations. Undoubtedly much can be done to avoid errors by a rational and unambiguous system of labelling, and a committee set up by the Ministry of Health has recently published recommendations for the labelling of ampoules of anaesthetic drugs.¹

It appears to be generally agreed that it is desirable to avoid gummed labels on ampoules, and machines which will satisfactorily print directly on to glass are now available at reasonable cost. Use of this method would avoid the danger of labels becoming detached. A further advance would be made if commercial firms could be persuaded to use pharmacopoeial names in their labelling instead of coining new names for established preparations.

The suggestion of tinting a large number of injection preparations with different colours does not appear to be very practicable, since it would lead to the surgeon having to learn the colour of each preparation, and the method could not be applied to substances which have to be protected from the light by the use of amber bottles. Also, since the range of colour in suitable dyes is limited, the use of different shades might lead to confusion with those whose colour-perception is not acute.

With regard to the labelling of rubber-capped multiple-dose containers I suggest that the label should be headed “Injection Solution” or “Intravenous Solution” as the case may be, followed by the B.P. name in large letters. Solutions not intended for injection should never be placed in similar bottles to injection solutions, and local anaesthetics intended for topical use should be clearly marked “Topical Solution—Not for Injection.” In both the above cases the use of different coloured labels might be an additional safeguard.

I consider that the final essential in the avoidance of errors is to acquire the habit of deliberately checking the label of any drug used rather than relying on its appearance, and to discard any preparation when there is the slightest doubt about its identity.—I am, etc.,

University College Hospital,
London, W.C.1.

¹ *Pharm. J.*, 1948, 105, 54.

T. D. WHITTET,
Chief Pharmacist

Pulmonary Embolism

SIR,—I have read the article by Mr. Norman C. Lake on “The Aftermath of Gastrectomy” (Feb. 14, p. 285) with considerable interest, but I must join issue with him on the problem of pulmonary embolism. The author disposes of this matter in less than six short lines and he shows an extraordinary disregard for all modern knowledge of the subject. He states that embolism “remains a bugbear” and that he knows no effective method of preventing it. This, I submit, is an astonishing statement in 1948, when one would hardly have felt it necessary to commend the work of our Swedish and American colleagues to his notice.

Mr. Lake states that “thrombosis usually arises in the abdominal wall or, as in two of these cases, from extension of thrombosis of haemorrhoids possibly produced by the administration of an enema pre-operatively.” These suggestions are surely preposterous, and it is not surprising that anyone believing them remains unaware of any effective method of preventing embolism. I understand that the late Lord Moynihan used to teach that emboli originated from vessels in the abdominal wall, although, so far as I am aware, he never offered any evidence in support of this view. Such hypotheses should now be condemned to the scrap-heap, where their arrival is long overdue; and to those who still favour these views I would suggest a few visits to the necropsy room, where, with the aid of a scalpel, they can soon dispel any remaining scepticism about the paramount importance of the deep veins of the lower limb as a source of post-operative embolism.

Active movements, early rising, prompt recognition of thrombosis in the calves, and abortive treatment with heparin