

be the rule that the patient is not discharged home until the patient's own general practitioner has been consulted and has agreed that home treatment is suitable.

It could well be that if the above principles were followed the burden on the psychiatric services would be lessened.—I am, etc.,

Strood, Kent.

W. A. PRITCHARD.

Cancer and the Nervous System

SIR,—In a recent leading article (26 July, p. 193) entitled "Cancer and the Nervous System" you point to our lack of knowledge concerning the mechanism of production of the carcinomatous neuropathies. However, the leader postulates the existence of a remote and specific effect of the neoplasm on the nervous system.

If one considers the general manifestations of cancer in man, one can distinguish two general classes. The first class, represented, for instance, by loss of weight, anaemia, and infections, can usually be explained by factors which are not specific for cancer. Quite different is the second group of manifestations, the so-called paraneoplasia. These are specifically caused by an abnormal function of the cancer tissue such as production of hormones. The question is, to which of these groups do the carcinomatous neuropathies belong?

In our experience here it has been possible to explain most of the neurological disorders observed in patients with cancer other than by a remote effect of the tumour.

In particular, nutritional factors seem to play an important role in producing the neuromuscular disorders observed in these patients.¹ Some of the characteristics of the so-called carcinomatous neuropathies, such as their failure to respond to a complete removal of the neoplasm and the lack of correlation in the clinical evolution of the cancer and the neuropathy,² are in contradiction with the idea that the former is directly responsible for the production of the latter. In this respect I disagree with the statement that the effects of neoplasm on the nervous system are reminiscent of hormonal disturbances. The mechanism of production of endocrine paraneoplasia has been elucidated: they are caused by the cancer tissue and they are completely suppressed by removal of the tumour.

Since many of the syndromes forming the group of carcinomatous neuropathies are also observed in patients without cancer, the physiopathological basis of these disorders was based on alleged frequency of the association of certain forms of cancer with certain neurological disorders. But even on that point divergent results have been published. Thus it was found, in a recent study, that the rate of unexplained neurological disorders was at least as high in patients with chronic lung diseases as in patients with lung cancer.³

I have previously expressed my opinion that unless a specific effect of the cancerous tissue on the nervous system can be demonstrated the majority of the so-called carcinomatous neuropathies should be considered as non-specific complications.⁴ For instance, the progressive multilocal leucoencephalopathy, which is probably viral in origin,⁵ is frequently found in patients with lymphoma. Other infections of the nervous system, such

as herpes zoster or cryptococcal meningitis, are also frequently found in that group of patients, but unlike leucoencephalopathy are not usually included among carcinomatous neuropathies. It may be reasonable to consider all these diseases as the result of the well-known decreased resistance to infection of patients with lymphoma.

I do not deny the necessity of further research on this subject, as suggested in your leading article. However, I am not convinced on the basis of clinical observations referred to that there is a specific and remote effect of cancer on the nervous system.—I am, etc.,

J. HILDEBRAND.

Service d'Investigation Clinique et de Médecine Interne, Institut Bordet, Brussels, Belgium.

REFERENCES

- ¹ Hildebrand, J., and Coërs, C., *Brain*, 1967, **90**, 67.
- ² Croft, P. B., and Wilkinson, M., *Brain*, 1965, **88**, 427.
- ³ Wilner, E. C., and Brody, J. A., *Neurology*, 1968, **18**, 1120.
- ⁴ Hildebrand, J., *European Journal of Cancer*, 1967, **3**, 159.
- ⁵ Zu Rhein, G. M., and Chou, S. M., *Science*, 1965, **148**, 1477.

An Ingenious Munchausen

SIR,—The "ingenious Munchausen" described by Dr. D. R. Seaton (3 August 1968, p. 317), Drs. J. M. Malins and P. J. Watkins (31 August 1968, p. 554), and Dr. A. C. Young and colleagues (5 July, p. 56) recently arrived in Belfast.

On examination he was pyknic in build, about 5 ft. 6 in. tall (162.5 cm.), and balding. His abdomen was obese, and there were very marked

striae in both loins. The bedclothes were heavily blood-stained, and examination of the urine revealed large numbers of red blood cells but no ova or crystals. Intravenous pyelography was normal and a telephone conversation with Dr. A. C. Young in Aberdeen confirmed that we were dealing with the same patient.

It was possible to keep him here long enough to be interviewed by a psychiatrist (W. A. N.). Eventually he admitted that his cover had been broken. He became much more communicative and was informally admitted to a psychiatric unit. Under questioning he denied trauma to the urethra, but the pulp of each finger bore white healed linear scars which he said were produced by cutting with razor blades to obtain blood. There were two recent stab wounds with bruising on his left thumb. He also claimed to suffer from paroxysmal tachycardia.

He knew he was the subject of correspondence in medical journals and claimed he could recognize in hospital others who, like himself, were going from hospital to hospital. He said that after working in the Congo he was ostracized by his countrymen, so he left home and has been in Britain for two years doing various jobs. Lack of money seems to be his main reason for seeking hospital admission, as at no time did he show signs of drug withdrawal.

He discharged himself five days after his admission to the psychiatric unit and we believe went to Eire.

We wish to thank Mr. G. W. Johnston for permission to publish details of this case.

—We are, etc.,

W. A. NORRIS.

TREVOR C. TAYLOR.

Royal Victoria Hospital, Belfast, N. Ireland.

Nail Changes in Gout

SIR,—During the past 15 months while working in a country town in New South Wales, Australia, I have observed an interesting physical sign in relation to atypical gout. This sign is briefly mentioned in Sequeira's textbook on *Diseases of the Skin*—namely, "reeding and breaking of the nails." I can find no other references to this condition in combination with a raised serum uric acid.

The appearance of the nails is characteristic. Longitudinal ridging, or reeding, as it is called, can be seen, starting at the cuticle and running the entire length of the nail. The longer the duration of the illness the more marked the reeding, and in long-

standing cases it was very marked and could easily be felt as well as seen. Often the nails are broken at the ends.

Nineteen cases have been seen and all had reeding of the nails and a raised serum uric acid. Extreme brittleness and breaking of the nails were present in most.

The degree of reeding appears to bear a direct relation to the duration of the illness, and in fact two further cases have been seen in a first attack where no nail changes had occurred.

The atypical type of gout, presenting with shoulder, chest, or finger pain, is the most likely to be missed, and the reeding and

Presenting Symptom	No. of Patients	Age Range	Degree of Reeding	Duration of Symptom	Uric Acid
Backache	1	42	Mild	3 years	6.8
Hypertension	1	43	Mild	4 years	7.3
Swelling of ankles and painful feet	4	32-70	Moderate	2-4 years	6.9 8.8 8.2 9.8
Upper abdominal pain	1	43	Mild	2 years	6.6
Retrosternal pain	2	56-58	Mild	3 years	8.0 8.9
Auricular fibrillation	1	54	Moderate	1 week	7.1
Pain in shoulder	1	60	Marked	7 years	8.6
Pain in elbow	1	42	Mild	1 year	6.2
Pain in feet	2	42-46	Marked	6 years	9.5 8.9
Pain in fingers	4	54-58	Marked	6-8 years	6.2 7.7 8.1 8.6
Occipital headache	1	59	Moderate	5 years	7.0

splitting of the finger-nails provided me with a clue to the diagnosis in these cases. The coexistence of nail changes and a raised serum uric acid in 19 cases would appear to suggest that reeding and splitting and brittleness, either individually or together, may be a frequent physical sign in hyperuricaemia and might warrant further investigation.—I am, etc.,

G. A. RAIL.

Casino Memorial Hospital,
New South Wales, Australia.

Detection of Hyperparathyroidism

SIR,—The most important biochemical finding in the detection of hyperparathyroidism is a raised plasma calcium level. However, in the differential diagnosis from other forms of hypercalcaemia additional biochemical findings are necessary. The following case report points to the plasma chloride level and so may be of interest.

A woman age 35 years with a history of renal calculi four years and seven years previously, and whose serum calcium was 12.9 and 11.9 mg./100 ml. and inorganic phosphate 2.05 and 2.5 mg./100 ml. with normal alkaline phosphatase (13.8 and 14.4 K.A. units), was admitted for further investigation in March 1968. For about 2½ years she had had dyspnoea on moderate exertion, pain in her left upper arm, and paraesthesiae of the fingers of her left hand. More recently she had been feeling irritable with marked tiredness, loss of energy, loss of weight, and blurring of vision.

Physical examination of this healthy looking female with a pulse rate of 80 and B.P. 150/90 was normal. Full blood count, E.S.R., blood urea, x-ray of chest, skull, hands, and barium swallow were also normal. Urine specific gravity was 1017 and reaction acid. Serum sodium and potassium were normal, but serum chloride was elevated to 115, 120, and 121.5 mEq/l. In a specimen of arterial blood the pH was 7.36, bicarbonate 22.8 mEq/l., and PCO₂ 43 mm. of mercury. Serum calcium was 12.6 mg./100 ml., inorganic phosphate 2.4 mg./100 ml., and alkaline phosphatase 12.3 King-Armstrong units. The E.C.G. showed a shortened QT interval (0.36 second) compatible with hypercalcaemia.

Because of the progressive symptoms with the persistently raised serum calcium, lowered inorganic phosphate, and the hyperchloraemia, the neck was explored, and a parathyroid adenoma, proved by histology, was removed on 14 March. Post-operative progress was satisfactory and her serum calcium was 9.7 and 9.8 mg./100 ml. on the first and fourth postoperative days. On this latter day the serum inorganic phosphate was 3.2 mg./100 ml., and alkaline phosphatase 10.9 King-Armstrong units. Serum chloride decreased post-operatively and was 109, 102, and 104 mEq/l. on the first, second, and fourth post-operative days; other serum electrolytes (Na and K) remained normal.

At follow-up one year postoperatively (April 1969) the patient was very well and her symptoms had disappeared. Her plasma calcium was 9.7 mg./100 ml. and her plasma chloride 107 mEq/l.

The plasma inorganic phosphate level is the additional biochemical finding generally thought to be of value in the differential diagnosis of hyperparathyroidism from other forms of hypercalcaemia. According to Thomas, Connor, and Morgan¹ and Howard² the plasma bicarbonate might be useful. Wills and McGowan³ found that 32 of their 33 patients with proved primary hyperparathyroidism had plasma chloride levels of not less than 102 mEq/l. Furthermore, they believed that the plasma chloride level was

superior to the plasma inorganic phosphate level for differentiating hyperparathyroidism from other conditions associated with hypercalcaemia.

This case report shows that an elevated plasma chloride level may be of value in the differential diagnosis of primary hyperparathyroidism.—We are, etc.,

ANDREW HEFFERNAN.
HELEN CARTY.

Mater Misericordiae Hospital,
Dublin 7.

REFERENCES

- 1 Thomas, W. C., Connor, T. B., and Morgan, H. G., *Journal of Laboratory and Clinical Medicine*, 1958, 52, 11.
- 2 Howard, J. E., *Transactions of the College of Physicians of Philadelphia*, 1962, 30, 55.
- 3 Wills, M. R., and McGowan, G. K., *British Medical Journal*, 1964, 1, 1153.

Abortion Act Amendment

SIR,—I would like to draw the attention of general practitioners to the rather subtle attack on their status implied in the recent failed amendment, and the lobbying and manœuvring continuing now, in connexion with the Abortion Act.

The Act as it stands simply requires the opinion of two doctors, who may therefore be general practitioners. While claiming that there is no suggestion that any general practitioner be excluded from assessing that an abortion is necessary or desirable, what is being sought by the opponents of the Act in its present form is that one of the two doctors should be a consultant gynaecologist holding office in the N.H.S.

What this seemingly innocuous and apparently sensible suggestion amounts to in fact is a despicable attack on the competence of "the other" general practitioner to make the relatively simple obstetric assessment necessary and give the second opinion required by the Act. By restricting the operation of the Act to a minority of the profession, many of whom are opposed to it on religious and other grounds, will cripple it. Furthermore, I think it should be pointed out that, with the Act as it is at present, the gynaecologist who carries out the termination provides a third safeguard, if that is necessary.

I hope this threat to the Act and attack on the status of the general practitioner will be noted and acted upon by those concerned.—I am, etc.,

London N.W.3.

NORMAN CHISHOLM.

Elbow Disorders

SIR,—In his article on the elbow joint (16 August, p. 399) Dr. Malcolm Thompson refers to the possible origin of pain felt in the upper limb being in the cervical spine, especially when the pain is diffuse, with no localized tender points. I should like to draw attention to two causes of typical "tennis" or "golfer's" elbow, in which the cause is proximal, but in which there is well-localized tenderness and pain, because they frequently lead to failure of the usual treatment or to repeated relapse.

The first cause lies in the cervical spine, either as the common "stuck neck" with cervical discomfort and limitation of movement, but with normal x-ray findings, or as cervical spondylosis, often mild; in either case symp-

toms and signs in the neck may be minimal. In the first case manipulation of the neck produces a click and restores full movement with relief of the elbow pain sometimes in a few minutes, but more frequently within a few days; in the second case the usual treatments of cervical spondylosis, such as a soft collar, traction, and mobilizing exercises, and in selected cases gentle manipulation, produce relief.

The other, and more rare, cause of treatment-resistant tennis or golfer's elbow is a lesion of the acromioclavicular or occasionally sternoclavicular joint, or both. Again, this area may be virtually symptom-free, or there may be some discomfort in the shoulder, specially at the extremes of elevation, external rotation, or adduction of the arm. The affected joint is invariably tender to pressure. The cause of the lesion may be trauma, often minor, such as a fall or a jerk, or unaccustomed work—for example, gardening or carrying heavy cases on holiday; occasionally it is found to be early and sometimes previously undiagnosed rheumatoid arthritis. The elbow pain is sometimes improved dramatically following the injection of steroid with or without local anaesthetic into the tender joint; the total quantity injected should be less than 1 ml. to reduce the risk of a temporary increase in the pain owing to distension of the joint.—I am, etc.,

G. H. UNGAR.

National Spinal Injuries Centre,
Stoke Mandeville Hospital,
Aylesbury, Bucks.

SIR,—I should like to congratulate Dr. Malcolm Thompson on his excellent article on the elbow (16 August, p. 399). He stresses the importance of making certain that the patient is not suffering from gout, which applies equally to the female sex over the age of 50.

I should like to emphasize the question of strain of the forearm extensors in tennis elbow. The tender points are over the lateral epicondyle and the radio-humeral ligament which constitute the origin of the extensors. However, in the action of producing the forearm stroke or similar movements, as the dentist extracting molar teeth, the hand starts from the pronated position and goes to the supinated position with the elbow slightly flexed. Gradually, therefore, in tennis elbow the radio-humeral joint becomes involved, and, as Winchester¹ has shown, marked degenerative changes may occur in the head of the radius and the corresponding articular surface of the lower end of the humerus in patients with this condition as early as the age of 30. The biceps muscle is the main flexor of the elbow and supinator of the forearm, and becomes involved in almost all cases of tennis elbow. This is shown by tenderness in relation to the long head of biceps in the front of the shoulder.

Golfer's elbow is a similar condition in which, however, the movement takes place with the pain starting from the supinated position and going to the pronated position. The flexors and the pronator radii teres are activated and the biceps can still be involved because the elbow is always held slightly flexed.

My opinion is that many of these strains are the result of faulty head-neck-shoulder