and dicoumarol should eradicate more than 90% of our yearly toll of deaths from embolism, besides reducing the other morbid effects of thrombosis. Although there are occasional cases where even the most expert may fail to detect deep venous thrombosis, a daily scrutiny of the temperature and pulse charts and examination of the patient's calves should be our ideal. With extension of this practice I trust that the day is not far distant when all surgeons will regard a fatal embolism as a reflection on their standard of post-operative care.

Finally, may I quote from a letter I received only a few days ago from a foreign authority with an international reputation on this subject? "In fact, I have for a long time expected that Britain should awake to conviction of the usefulness of anticoagulant therapy, but your people, which I like more than other nations, are stubborn and conservative."—I am, etc.,

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Tuberculosis of the Great Trochanter

SIR,—In Mr. B. McMurray's article on "Tuberculosis of the Great Trochanter" (Sept. 27, 1947, p. 492) the author suggests that "where there is radiological evidence of peripheral erosion in tuberculosis of the great trochanter the bony lesion is secondary to tuberculous infection of the trochanteric bursa, and begins only after secondary infection has taken place." Two of the reasons which he produces in support of this are: (1) the five patients in his series who showed no erosion of the bone radiologically were the only ones (except the one with the encysted form) who had not yet developed sinuses and were therefore free from secondary infection; (2) in all the radiographs of peripheral erosion there is definite evidence of sclerosis, and this is undoubtedly due to the secondary infection which is present.

As the total number of cases reported in the literature is so small the following case is of interest as it disproves Mr. McMurray's suggestion that secondary infection must be present before erosion of the base takes place. It does not of course affect his conclusion that the original site of the tuberculous lesion is the trochanteric bursa.

The case history was as follows. The patient was a stoker in the Royal Navy, aged 18, who was running after a football on Sept. 18, 1947, when he first noticed that he could not flex his right hip properly owing to a pain on the outer side of the hip-joint. He had previously noticed no trouble in the joint; there was no history of injury and no other relevant medical history.

He was admitted to the Royal Naval Hospital, Chatham, on Sept. 25 and examination revealed a cystic swelling 2-3 inches (5-7.5 cm.) in diameter situated just anteriorly and above the right greater trochanter. When he stood up the swelling disappeared behind the tensor fascia lata and iliotibial tract. X ray showed a fluffiness of the upper surface of the greater trochanter, but no sclerosis. There was no evidence of tuberculosis elsewhere. E.S.R. was 20 mm. in 1 hour (Wintrobe), W.B.C. 7,800 cells per c.mm., and the skin over the swelling was intact. A provisional diagnosis of tuberculous subgluteal bursa was made.

On Oct. 10, 50 ml. of viscid straw-coloured fluid was aspirated from the bursa and sent to the laboratory. It was found to contain 480 leucocytes per c.mm., of which 55% were lymphocytes and the rest polymorphs. No tubercle bacilli were found in a direct film and no organisms seen with Gram stain. Agar culture was sterile after 24 hours.

On Oct. 21, nearly five weeks after the onset of symptoms, the patient was operated upon and the bursa with all its ramifications excised. It was found to contain numerous melon-seed bodies. A caseous area of bone at the upper end of the greater trochanter was curetted out. The wound was closed without drainage and a fortnight later it had healed by first intention. The E.S.R. had fallen to 2 mm. in 1 hour, and the patient was placed in a plaster-of-Paris hip spica.—I am, etc.,

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Brachial Neuralgia

SIR,—The account of a discussion on brachial neuralgia (Nov. 29, 1947, p. 882) brings to light the weaknesses of the general belief that pain is due to pressure on nerve fibres. If pain of uncertain origin is felt in the arm we are permitted to assume that nerves are being subjected to pressure or tension. Then it remains only to discover or to invent a possible

mechanism by which pressure or tension might be generated. For long the theory of the cervical rib held sway; and, when this proved inadequate, the "pseudo-cervical rib," or fibrous band that could not be revealed by x ray. Later, spasm of the scalene muscles was suggested, and more recently abnormal approximation of clavicle to first rib. Higher up, osteophytic outgrowths or intervertebral disks have been supposed to press on nerve roots.

When pressure on nerve trunks was found inadequate to explain the symptoms, pressure on arterial trunks or on sympathetic fibres was advanced to take its place. From France recently two new explanations of the pain have come: (1) excitation of sympathetic fibres by venous stasis in the scalene tunnel;¹ (2) spasm of the subclavius muscle.²

From the advent of the cervical rib theory its inadequacy has been admitted by even its strongest advocates. Thus the signs often extend beyond the field of the root or trunk that is pressed upon. To meet this Oppenheim³ invoked a genetic deficiency; skeletal and neuropathic defects, he said, are both stigmata of degeneration and may occur in the same subject. Many authors have reported cervical ribs combined with syringomyelia or with disseminated sclerosis.

Before agreeing that pain means pressure on nerves we should ask if pain is prominent in disorders known to cause pressure on nerves. The following evidence is available.

1. Experimental. Pressure on the ulnar nerve causes numbness and tingling, but no pain, in its distribution. "The excitation of a skin nerve in its course (e.g., faradization of ulnar trunk at elbow) it was at one time taught evoked pain only, not sensations of touch. To refute the teaching, the experiment has only to be repeated. The sensation obtained is usually not painful, though always strange."⁴

2. Injury. (a) Excluding a small number of causalgic cases, gunshot wounds involving nerve trunks are not especially painful (b) Paralysis of nerves by external compression often is painless—e.g., "Saturday night paralysis," tourniquet paralyses, etc.

3. Tumours. (a) Pain is not especially characteristic of tumours of nerves, malignant,⁵ or benign.⁷⁻⁹ (b) Of 47 benign tumours arising from nerve roots 25 were painless.¹⁰ Dumb-bell tumours of the cord which grow through the intervertebral foramina and press on nerve roots are often completely painless.¹¹⁻¹³ (c) Of 53 extramedullary meningiomas pain was felt in only 22.¹⁰ A meningioma that compressed the first dorsal and the lower cervical roots, producing a triplegia, was painless.¹⁴ Extradural cysts that enlarge the spinal canal by erosion usually cause no pain.^{15 16} (d) Of spinal cord tumours in general Lambert Rogers¹⁷ wrote: "Although for many years pain has been regarded as one of the characteristic features of spinal tumour, several of our cases have run an almost painless course." Of 42 neoplasms reported by Spurling and Mayfield¹⁸ pain was entirely absent in 11 and was an early symptom in only 21.

We shall have to look elsewhere than to pressure on nerves to explain pain in the arm.—I am, etc.,

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Treatment of Subacute Bacterial Endocarditis

SIR,—Dr. Florence M. E. Davies (Feb. 14, p. 317) discusses the use of penicillin in this condition and states that she thinks that "penicillin merely clears off invaders for the time being," due to the fact that insufficient penicillin may have been given for the numbers of bacteria present. Were this statement in fact true, all that would be necessary to effect a complete and final cure (with the proviso that penicillin cannot restore an injured valve to normal anatomy) in all cases of subacute bacterial endocarditis due to penicillin-sensitive organisms