In fact there exists a very simple clinical test distinguishing between pain originating from a joint and from adjacent muscle. The function of muscle is to contract; if it does so fully and painlessly it cannot be causing the patient any pain, however tender or nodular it happens to be.¹ If a muscle is made to contract against such resistance that no movement takes place the tension on the muscle alters, but the joint remains stationary. If this attempt at movement causes or aggravates pain a muscle lesion is present. Since it is clearly desirable in any case of backache, before proceeding to palpation, to find out whether the lesion is muscular or articular, the joints and muscles should be tested separately first. This is the more important since referred pain, referred tenderness, myalgic spots, and trigger points are all commonly present at the site of pain in the dural and nerve-root lesions set up by disk protrusion. Trigger points, etc., result from the lesion causing pain; they are not primary.²

The clinician's only safeguard is to test each muscle individually against resistance; this singles out the muscle at fault—if there is one. This is the only way to find out whether or not to disregard the tender fat lobules that Copeman and Ackerman's own dissections, no less than everyday clinical experience, have shown to exist in many normal subjects without interfering with muscle action.

I have spent many years examining muscles and joints in this way and warmly commend it to those who would like to know at what structure to look for the lesion in cases of pain in the trunk or indeed about any part. Another virtue of this method is the quick detection of functional symptoms that it provides. When the active, passive, and resisted movements at a joint are tested, patients who have no local lesion must answer at random, and a self-contradictory pattern soon emerges. This system, so quick and simple to use by the examiner, confuses only those patients whose pain possesses no organic basis. We have found it, in reverse, a valuable criterion for detecting genuine local lesions in patients obviously neurotic; a common injustice is thus avoided. Dr. Tegner and his colleagues (July 23, p. 201) have done us a great service in emphasizing the frequency of functional symptoms in patients sent for physiotherapy.

Every layman knows what "rheumatism" is, but there exists no acceptable medical definition. If it is to mean anything at all (except of course in rheumatic fever) it must indicate some non-specific lesion of moving parts, chiefly of muscles and joints. My disagreement with Dr. Tegner is therefore only on nomenclature. Psychogenic "rheumatism" is self-contradictory, for if the lesion lies in the patient's sensorium the word rheumatism becomes inappropriate. "Functional" or "psychogenic pain" avoids all attempt at fusion of two incompatible words and provides an unambiguous alternative.—I am, etc.,

London, W.1.

JAMES CYRIAX.

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- ¹ Cyriax, J., *British Medical Journal*, 1948, 2, 251.
- ² *Quart. J. Med.*, 1944, n.s. 13, 37.
- ³ Cyriax, J., *Rheumatism and Soft Tissue Injuries*, 1947, London.
- ⁴ ———, *St. Thomas's Hospital Reports*, 1949, in the press.

Strains of *Bact. coli*

SIR,—It seems quite likely that whenever a new publication appears on the subject of *Bact. coli* in relation to gastro-enteritis it will be found that the authors have coined a fresh name for the bacteria or the strains which they have isolated, although these may in fact be *Bact. coli* of the same serological type. Giles and Sangster¹ have stated that the strains they isolated in Aberdeen were identical serologically with those originally isolated at Hillingdon by Bray.² Drs. Joan Taylor, B. W. Powell, and Joyce Wright (July 16, p. 117) say that their strains are identical serologically with those of Giles and Sangster. It follows therefore that the D 433 strains of Dr. Joan Taylor and her colleagues are identical with Bray's original strains—a point which they did not make in their paper.

A summary of the nomenclature up to date is as follows.

- (1) Bray, 1945, and Bray and Beavan,³ 1948, referred to these bacteria as *Bact. coli neapolitanum*, and the two strains in circulation among bacteriologists are called Lindsay and G. R.
- (2) Giles and Sangster, 1948, and Giles, Sangster, and Smith,⁴ 1949, refer to the bacteria as *Bact. coli neapolitanum* type *alpha*. The *beta* type described by them, also in association with gastro-

enteritis, is a serologically distinct type. (3) Taylor, Powell, and Wright, 1949, refer to the bacteria as *Bact. coli* D 433. I shall also immodestly add (4): in Holland, where these bacteria are being found (personal communication by Dr. ten Seldam), they are even referred to as *Escherichia coli* bray.—I am, etc.,

London, N.W.1.

JOHN BRAY.

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- ¹ *J. Hyg., Camb.*, 1948, 46, 1.
- ² *J. Path. Bact.*, 1945, 57, 239.
- ³ *Ibid.*, 1948, 60, 395.
- ⁴ *Arch. Dis. Childh.*, 1949, 24, 45.

Zoster and Varicella in the Same Patient

SIR,—I was interested to read letters from Dr. A. Courts (June 11, p. 1052) and Dr. C. E. Taylor (July 9, p. 100) on the coexistence of herpes zoster and varicella. This combination cannot be considered rare and is in fact not uncommon in a hospital admitting infectious diseases from a considerable catchment area.

I have had five such patients under my care at the Neasden Hospital during the last three months. In one case the two manifestations appeared concurrently. In four cases the varicella developed in periods of 2–15 days after the appearance of herpes zoster and before it had healed. The nervous lesions involved the area of the first, second, third, and fourth lumbar roots in one case, and the second, third, and fourth sacral roots in another; in the remaining three cases it was the ophthalmic division of the fifth cranial nerve.—I am, etc.,

London, N.W.10.

W. F. TWINING McMATH.

SIR,—Further to the letters on this subject in the issues of June 11 (p. 1052) and July 9 (p. 100) we record particulars of another instance of this occurrence.

A male patient aged 84 years was recently admitted with well-marked herpes zoster involving the distribution of the posterior root of the second cervical nerve, accompanied by a typical attack of mild chicken-pox with characteristic distribution. No contact history with either complaint could be established, and he had no recollection of a previous attack of chicken-pox. The herpes zoster had preceded the onset of the varicella by six days.

As some of the immature varicellous lesions presented a haemorrhagic appearance and the base of some of the mature lesions was unusually hyperaemic, the possibility of vitamin-C deficiency was considered likely, especially as the patient admitted he seldom partook of fresh fruits. The ascorbic acid saturation test showed a considerable deficiency of vitamin C, although no other clinical evidence of deficiency was apparent.

Apart from the relative infrequency of this concomitance of chicken-pox and herpes zoster the case is additionally interesting in view of the very advanced age of the patient.—We are, etc.,

J. H. WEIR.

G. LAING BROWN.

Ilford, Essex.

Eclampsia and Peritoneal Haemorrhage

SIR,—I have read with much interest the article entitled "Rupture of Liver Associated with Parturition" by Dr. Jean R. C. Burton-Brown and Dr. J. A. Shepherd (May 28, p. 941). The prophetic suggestion of Rademaker that sudden death during eclampsia may sometimes be due to a ruptured liver was rejected by the writers of the article. The following brief notes of a case may prove of interest to your readers.

An East Indian woman of 30, para 8, was admitted to hospital with a history of having had a fit at 6 a.m. on Feb. 4. In the ward she had two fits—the first at 7.30 a.m., and the second at 9.30 a.m. At 4 p.m. her condition was satisfactory. The patient died suddenly at 5.15 p.m. Post-mortem examination revealed that the peritoneal cavity was full of fluid blood and blood clot. This had come from a very large subcapsular haemorrhage on the upper surface of the left lobe of the liver, which had ruptured the capsule at one point. The liver showed advanced eclamptic changes. The kidneys were pale and the cortex swollen. Nothing else abnormal was noted. Death was attributed to peritoneal haemorrhage and eclampsia.

—I am, etc.,

Port of Spain, Trinidad.

JAMES A. WATERMAN.