3.6 g./100 ml.). Protein electrophoresis showed an increase in all the globulin fractions with mild decrease of the albumin fraction.

The most likely abnormality was therefore an increase in capillary fragility, although a qualitative defect could not be outruled. The pupura gradually faded; and on discharge four weeks later the tourniquet test, though still positive, was less marked. During her hospitalization she was transferred from insulin to chlorpropamide (Diabinese) 500 mg. daily, with good control of her diabetes and no deterioration in her purpura.

She was readmitted three weeks later with right hemiparesis due to cerebral thrombosis. Chlordiazepoxide 10 mg. was given on admission. The following morning the tourniquet test was negative and the platelet count was 200,000/cu. mm. The blood urea was 44 mg./100 ml., and urinalysis showed 60 mg./100 ml. protein only. Five days after admission she was noted to be depressed, and chlordiazepoxide 10 mg. t.i.d. was prescribed. Within 48 hours the purpura reappeared and the tourniquet test was strongly positive. The platelet count was 190,000/cu. mm. The chlordiazepoxide was discontinued; the purpura gradually faded, and the tourniquet test became steadily weaker. Nine weeks later it was negative and the platelet count was 162,000/cu. mm. It was found difficult to control her diabetes with chlorpropamide and insulin therapy was recommenced, with good control and no recurrence of the purpura. She was discharged with residual hemiparesis, has remained well, and has had no further episodes of purpura.

The only reported case of purpura occurring with chlordiazepoxide administration was at a clinicopathological conference,[1] at which concurrent chloramphenicol administration was considered responsible for the purpura. Thirty-four suspected cases have been reported to the manufacturers, but no cases have been reported in the literature. It is interesting to note that the purpura did not appear when 10 mg. was administered on the evening of her second admission, and only appeared when 30 mg. per day was given later. It seems, therefore, that there is a minimum dose needed to manifest the purpura.—I am, etc.,

Mercer's Hospital, Dublin 2. ISAAC J. COPPERMAN.

REFERENCE

[1] *Amer. J. Med.*, 1965, **39**, 260.

Salmonella in Tortoises

SIR,—I was most interested to read Dr. L. T. Newman's letter on the problem of salmonella in tortoises (4 November, p. 296). It is a well-known fact that tortoises and related terrapins are excreters and carriers of salmonellas, and, as such, are a constant hazard to their owners and handlers, in particular young children.

In 1966, in conjunction with Dr. C. D. Plows, of the Sheffield Public Health Laboratory Service, we were able to identify an Arizona strain from a case of gastroenteritis in a 3-year-old Sheffield girl. A symptomless excreter of the organism (her 5-year-old brother) was also found in the same family. The source of infection was traced to a pet terrapin—a type of small turtle (genus: *Graptenys*)—purchased by the family from a pet shop in Sheffield. Full details of this investigation are to be reported in the *Journal of Hygiene* (in press).

Terrapins, like tortoises and turtles, are excreters of both salmonellas and Arizona. If the habit of keeping terrapins as pets increases, then these small innocuous animals may well become an important source of human salmonella infection. This is especially so in young children. Terrapins are more likely to act as a source of human infection than tortoises for the following reasons: terrapins are smaller and therefore easier and more tempting for young children to handle; they are usually kept indoors and in a tank of water; it has been observed that children do play with them and are in the habit of transferring them in and out of the tank at frequent intervals. If the terrapins are excreters then the water in the tank will become infected, so increasing the possibility of spread to humans or to other pets.—I am, etc.,

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Paraquat Poisoning

SIR,—The recent report of Dr. Ch. Almog and Dr. E. Tal (16 September, p. 721) of the result of a subcutaneous injection of paraquat (Gramoxone), with the development of a right facial paresis, prompts me to record another case of facial palsy associated with Gramoxone.

This was in a male horticultural worker of 49 who presented in May this year. He had rolled a cigarette with unwashed hands after washing spray nozzles contaminated with Gramoxone. He developed a burning sensation of the tongue and examination four days later revealed nothing abnormal. Two days later, he awoke with headache and nausea, sore throat, fever, aching of the limbs, and general malaise, and by mid-afternoon had marked muscular weakness and nearly collapsed. His temperature was 101.5° F. (38.5° C). Neck stiffness was present, and there was a thin line of vesicles on the right fauces. There was a horizontal nystagmus which the patient claimed had been present for years.

He was admitted under the care of Dr. J. Campbell, and four days later developed a right lower motor neurone facial palsy with no other neurological findings. The chest, skull, and mastoid *x* rays were normal. The cerebral spinal fluid the day after admission showed R.B.C.s 41/cu.mm., W.B.C.s 5/cu.mm. protein 110 mg./100 ml., sugar 100 mg./100 ml. chlorides 760 mg./100 ml. A repeat lumbar puncture one week later showed R.B.C.s 8/cu. mm., W.B.C.s 39/cu.mm. neutrophils 11%, lymphocytes 89%, protein 40 mg./100 ml., chlorides 750 mg./100 ml., sugar 104 mg./100 ml; the pressure was 150 mm. with a free rise and fall Blood count, E.S.R., blood urea, liver function tests, aspartate transaminase, and alanine transaminase were normal; the urine contained a trace of protein, many pus cells, and follow-up intravenous pyelogram suggested old right-sided pyelitis.

A follow-up chest *x* ray five months later showed no evidence of any active pathology in the lungs, and the heart was of normal size (Dr. Maxwell).

This sequence of contact with paraquat (Gramoxone) and facial palsy may be entirely coincidental; the minute dose of paraquat involved might suggest a hypersensitivity response in this case. It would be interesting to know of facial or other palsies occurring in others using or manufacturing paraquat.—I am, etc.,

K. A. MOURIN.

Dereham, Norfolk.

Low Back Pain

SIR,—"Medicine Today" (11 November, p. 341) presents what is presumably a résumé of eminent authoritative exposition of the symptoms, signs, diagnosis, and treatment of this most common of complaints. Reading this, for I have not yet seen the film, it truly amazes me that these are the practices still in vogue.

What is new? What does "Medicine Today" have to offer? Let us be reasoned, and, if you like, be with it. Let us read, learn, digest, and practise, if not all, some of the teachings of Dr. James Cyriax. He after all (and a great number of his enlightened colleagues) has been thundering out for 20 years or more the signs, symptoms, and treatment of derangement of intervertebral joints (D.I.J.).

Whether you like it or not, his whole concept works—and very well at that. The problem is for the average doctor to grasp, interpret, and treat the condition.

The essence of treatment of D.I.J., no matter how severe, is immediate manipulation (apart from S.4). The acute severely immobilized patient with lumbago or sciatica is gently manipulated, not once, but daily, on alternate days, or every third day. This, together with a rigid bed, trunk extension exercises (as soon as the patient can start them), and no lumbar flexion when up and about, has been constantly found to get the patient back to work within a week or two.

Lying doggo in bed, swallowing hundreds of painkilling tablets, sweltering under a heat-tunnel, etc., is just not good enough when immediate reduction of the D.I.J. and mobilization is all that is required.—I am, etc.,

T. HAMBLY.

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Intestinal Spirochaetosis

SIR,—Lieutenant-Colonel C. O. Burdick (11 November, p. 357) asserts that we have "misinterpreted normal intestinal microvilli cut tangentially as spirochaetes." He presumably bases this view on the single low-powered electronmicrograph published with our paper.

We wish categorically to deny this suggestion. There can be no doubt that the condition we described (16 September, p. 718) is an infestation of the colonic mucosa by small, spiral micro-organisms. These are much larger than microvilli but smaller than bacteria. Axial filaments are clearly visible in our own material and in negatively stained preparation of the organisms prepared by Dr. J. Gordon. The organisms are motile on dark-ground microscopy.

It is of interest that this condition is quite common and can be diagnosed from routine histological preparations. The lesions must have been seen frequently by pathologists, but it seems the appearances have not been interpreted correctly in the past.—We are, etc.,

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SIR,—Dr. S. C. Dyke (21 October, p. 176) has referred to my work on intestinal spirochaetosis in terms which I deeply appreciate.