phrenia had the following blood picture: Hb 150 g/l (cytochrome oxidase method); P.V.C. 70% (neutrophils 65%, lymphocytes 6%, monocytes 3%); platelets 450,000/mm$^3$. There was a good response to venesection and 20P. In March 1972, when severely paranoiac, he became unconscious on his feet. Examination of the central nervous system was normal: the Hb was 121 g/l; P.V.C. 68%; platelets 267,000/mm$^3$; leucocyte alkaline phosphatase score 115 per 100 neutrophils (normal 30-100). Soon after venesection num-
ness developed down the left side: it improved
in May. Lymphadenopathy was absent. The flexes and an equivocal left plantar response were later apparent. By June the left lower limb was paralysed with ankle clonus and an equivocal plantar response, the left arm was weak, and
there was urinary incontinence. A cerebral tumour was thought possibly to be causing a progressive hemiplegia. Chest and skull radio-
graphs and a brain scan were normal. After in-
creasing loss of consciousness he died in July.

Postmortem examination showed no evidence of neoplastic disease nor any endocrine, respira-
tory, cardiovascular, or renal abnormality asso-
ciated with a secondary polycythaemia. The spleen weighed 270 g and histological examina-
tion showed only venous congestion. The bone
marrow was hyperplastic and all elements were increased in proportion to the erythrocytes. The
brain was generally oedematous with recent thrombotic occlusion of the superior cerebral veins on both sides and a patent supr
rior sagittal sinus. Cut sections showed petechial haemorrhages within the white matter of the
right and left parietal lobes. The occlusion of the
superior sagittal sinus. Cut sections showed petec
hial haemorrhages within the white matter of the
right and left parietal lobes. The occlusion of the
right frontal and left occipital poles were old infarcts with cyst formation.

Malignant Lymphoma and Acute S.L.E.

Sir,—The interesting report by Drs. A. G. Cudworth and A. Ellis (29 July, p. 291) of
a case of malignant lymphoma and acute systemic lupus erythematosus prompts me to re-
iterate that I have seen two cases of Hodgkin's disease with clearly defined lupus erythematosus syndromes—one in a man aged 24 and the other in a man aged 41. We have also seen a number of other malignant reticuloses which were preceded by various conditions of an allergic or auto-
imune nature, including arthritis.1 In most cases the connective tissue disorder of essentially non-malignant character has pre-
ceded the irreversible, or malignant, lymphoma, but nearly all our cases of reticuloses, including chronic lymphocyti
leukaemia, lymphosarcoma, and reticulosa-
coma, and the paraproteinaemias, have had histories of recurrent or chronic inflamma-
tion. Whenever reactive arthritis or reticulo-
stimulus to the reticuloendothelial system. In
addition, high antibody titres against toxoplasma were found in approximately
90% of these patients (compared with an inci-
dence of 56% at the mass in controls matched for age$^2$). Such a stimulus, be it chemical, viral, bacterial, protozoal, or allergic, will lead to
a reactive proliferation of the target tissue, with or without the production of anti-
odies. The number of mitoses in a prolifer-
ing tissue is increased and with it the number of spontaneous mutations will rise. Mutations may also be promoted by some of the drugs given or even by the stimulating agent itself. Though the majority of
mutants have no chance of survival, with an increased total number the number of survivors will also increase. Some of these mutants will multiply and grow to become an abnormal cell clone.—I am, etc.,

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1 S. Chester-Karpin, Rose, and d'Heureux-Gerhardt,
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Israel Journal of Medical Sciences, 1965, 1, 819.
2 S. Chester-Karpin, Rose, and d'Heureux-Gerhardt,
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1960.
3 S. Chester-Karpin, Rose, Thesis, Humboldt Uni-
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R., Deutsche Gesellschaften der, 1967, 22, 139.

Lumbar Disc Problems

Sir,—I am surprised that one of the calibre of Mr. A. H. G. Murley should expect us to ac-
cept his beliefs (26 August, p. 529) in this
scientific age when nothing is acceptable unless it can be retested proved by adequate
demonstration or experiment. In the case of
disc lesions this has long since and very ade-
quately been done by James Cyriax and also by many others who have contrib-
uted adequate evidence to show that dis-
c lesions are indeed commonplace rather than rarities.

Whether damage to the posterior joints predisposes to disc prolapse is another ques-
tion. Cyriax has proved that these joints are
rarely capable of being the cause of the pain. It is a great misfortune that people I have
come across who have had their back-
ache cured after more than 20 years either
by manipulation or traction should have had
to suffer so long without effective treatment simply because they had been told that they
were suffering from chronic muscle or joint
spasticity. Finally, I am astonished to see Mr.
Munrley confuse Cyriax's findings with his
own and claim that he did not spring his
ankle, but when my knee locks owing to my
torn cartilage I should be delighted to visit
him for manipulation, which is the appro-
priate treatment for an internal derange-
ment of a joint, be it meniscus or disc.—I am,
etc.,

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Sheerness, Kent

1 Cyriax, J., Textbook of Orthopaedic Medicine, 5th

2 Chester-Karpin, Rose, and d'Heureux-Gerhardt,

Dysphagia and Sarcoid Granulomas

Sir,—In the unusual patient described by Dr. R. J. Davies (2 September, p. 564), who had had sarcoidosis for 14 years, the Tong-
silon test was negative but the muscle atrophy around the lesions of the oesophagus was reminiscent of that seen in myasthenia
gravis.

Four reported cases of myasthenia had sarcoidosis,1 and at least 30 more had giant-
cell myocarditis, which possibly may arise from subclinical sarcoidosis.2 Myocardial sarcoidosis is more common than was previously thought, particularly as the infiltration in the late stages cannot be identified with any certainty. Possibly idiopathic cases of cardiomyopathy may begin as sarcoidosis. Probably autoimmunity is a factor in this process. Sarcoidosis is rare in those with unexplained cardiomyopathies. Even when there are no initial symptoms or signs occur the long-
term effects of sarcoidosis can be severe. Much time has been spent defining the
Aflatoxins and Reye’s Disease

Sir,—The syndrome of encephalopathy and fatty degeneration of the liver (Reye’s syndrome; RS) has been reported in several countries as a cause of death or serious illness in young children. The aetiology remains unknown.1 One of us3 has suggested that contamination of foods by aflatoxins, the toxic products of strains of Aspergillus flavus, was a possible cause. This suggestion was based on the strong suspicion from pathological appearances that the liver lesions were toxic in origin, the similarity of the lesions to those of experimental aflatoxin poisoning, the special sensitivity of young children to toxicants, and the limited possibilities of exposure of some affected children to toxins except through foods. Recently circumstantial evidence has been presented that a similar syndrome occurring frequently in northern Thailand might be caused by aflatoxins from fungal-contaminated rice.4 5 This evidence includes the identification of aflatoxins in the organs of affected children.6 Aflatoxins have been sought in the tissues of two children dying in Auckland recently.

Case 1.—A 22-month-old Polynesian boy whose illness was typical of the syndrome as previously defined.3 There was a prodromal illness, drowsiness progressing to coma and then to death in 4 months. In the last week of life there was progressive weight loss and hyperpyrexia. Hepatomegaly, an elevated serum transaminase, and, in the post-mortem specimen, fibrosis were noted before her death in liver failure. A wide range of negative investigations included studies for inborn errors of metabolism and heavy metal poisoning.

Assay Methods.—Liver tissue in approximately 10-g samples was extracted by the method of Brown and Poss6 using dichloromethane as the solvent. The extracts were chromatographed on silica gel (Merck No. 5662/0001) and developed in chloroform-ethanol 98:2. Visual comparisons were made by a standard light with an aflatoxin B1 standard (Markov Chemicals Ltd., Israel) and two mixed aflatoxin standards (M.R.C. "Porton 41," and U.S. Dept. of Agriculture, "SA 14/4/3"). The derivatizations-formations method8 was used for the further identification of fluorescent materials.

Results.—Four separate extractions were made of liver tissue from Case 1 and three extractions of the liver of Case 2. Tissue had been stored at -4°C for from 3 to 4 months before study. All seven extracts contained a blue fluorescent material with the same Rf as aflatoxin B1 and, variably, a green fluorescent material with the same Rf as aflatoxin G1. Estimates of the former material as aflatoxin B1 in each extract ranged from 5 to 50 μg/kg weight of tissue and were considered insufficient material for further identification by derivatization methods. All fluorescing materials presented in each extract and reacted with antiserum to aflatoxin standards and non-identity was confirmed by derivatizations. Though inconclusive, these results encourage a further search for aflatoxin-like materials in the syndrome of encephalopathy with fatty degeneration of the viscera. All reported cases of Reye’s syndrome may not have had a single aetiology, particularly those in which the liver pathology has not conformed to the data laid down in earlier reports.1 3 A search for aflatoxins is indicated where, as in the cases of Reye’s disease recorded in Sydney between 1950 and 19631 5 and in Auckland between 1959 and 1966,6 the incidence of the condition in the area was low and presumably single environmental factor.9

The findings in Case 2 support the original hypothesis of an aetio-pathological link between a chronic liver disease and the acute encephalopathy. The material in the liver was aflatoxin then this has persisted longer than has been reported experimentally.10 It is of interest but probably coincidental that Case 1 was from an immigrant family with, for New Zealand, a large daily consumption of rice, and that the symptoms of Case 2 began after the introduction of solid foods of which "baby rice" was the first given. For the New Zealand is mostly imported from Australia and the United States with about 10% coming from Thailand in 1971. This work was supported by a grant from the Medical Research Council of New Zealand.—We are, etc.,

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References