ized maculo-papular rash appeared, which was partly petechial on her legs. There was no lymphadenopathy and the spleen was not palpable. The results of blood counts are shown below.

Day of Illness	W.B.C. /cu. mm.	Neutrophils /cu. mm.	Eosinophils /cu. mm.	Lymphocytes /cu. mm.
1	11,000	4,400 (40%)	1,210 (11%)	4,950 (45%)
3	17,000	5,780 (34%)	1,050 (5%)	10,030 (59%)
11	12,000	4,440 (37%)	3,480 (29%)	3,360 (28%)
24	9,000	6,390 (71%)	270 (3%)	1,890 (21%)

On days 1 and 3 nearly all the lymphocytes were atypical, and about half of these resembled glandular-fever cells. By day 11 the latter had disappeared and only an occasional lymphocyte was atypical. On day 24 the leucocyte picture was normal. The haemoglobin level and platelet count were constantly normal. Paul–Bunnell tests were repeatedly negative, and toxoplasma dye tests gave titres of 256 during and for three months after the illness.

It seems most unlikely that glandular-fever or toxoplasma infection was the cause of the illness in either case. Both infections are rare over the age of 50.[2,3] Lymphadenopathy was not present, as it is invariably at some time in glandular fever[2] and in cases of toxoplasmosis with glandular-fever cells in the blood.[3] A toxoplasma dye titre of 256, as in Case 2, is found in 1% of adults, and the titre in the second case did not rise during or after the illness. On the other hand, the skin rashes and the marked eosinophilia, and the agranulocytosis in the first case, strongly suggest that both patients suffered a reaction to the drug phenylbutazone, and that the glandular-fever cells in the blood were part of this reaction. This was the conclusion of Dr. Lawrence as regards his case, which showed an even greater lymphocytosis of 17,820/cu. mm. with many glandular-fever cells present and an eosinophilia of 1,080/cu. mm.

I wish to thank Drs. J. Laurie, E. A. Murray, and I. D. S. Cowie for the clinical details and permission to publish these cases, and Miss J. Hutchinson for assistance.

—I am, etc.,

J. G. SELWYN.

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1 Lawrence, A., *Brit. med. J.*, 1960, **2**, 1736.
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3 Beverley, J. K. A., and Beattie, C. P., *Lancet*, 1958, **2**, 379.

Elastic Band Injuries

SIR,—Deliberate production of artifact ulcers by elastic bands around the lower third of the leg is described in the paper by Mr. I. D. Kitchin and others (22 April, p. 218) and in the letter of Dr. K. Dawson-Butterworth (20 May, p. 510). Accidental production of artifact ulcers by elastic bands around the fingers and around the arm is described by Dr. J. G. B. Thurston (6 May, p. 376) and Mr. J. P. Turney (13 May, p. 445), respectively.

A widow aged 77 presented in September 1967 with a deep circumferential ulcer of the lower third of the right leg and a chronic varicose ulcer distal to this. She reported that a tight bandage had been applied to the varicose ulcer; this was postulated as the cause of the circumferential ulcer. The day after admission to hospital an elastic band

was removed from the depths of the ulcer. On further questioning she admitted having used an elastic band to hold a dressing on the varicose ulcer some weeks before admission, and had been perplexed at its disappearance.

The photograph was taken by Mr. A. D. Ring 24 hours after removal of the band.

—I am, etc.,

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Nalidixic Acid and Intracranial Hypertension

SIR,—There have been letters in the *B.M.J.* this year describing intracranial hypertension in children treated with nalidixic acid (29 April, p. 310; 5 August, p. 370; and 9 September, p. 679).

We therefore think it of interest to report the case of a middle-aged woman, who in the third week of acute glomerulonephritis, while yet still febrile and slightly oedematous, but with good urine output and blood urea of 24 mg./100 ml., was given nalidixic acid in a dose of 1 g. four times a day for a superimposed coliform infection of the urine. At the time she was normotensive and had no papilloedema. Within 24 hours of commencement of the nalidixic acid she developed, in the night, a toxic confusional state of schizoid type with paranoid ideas and depersonalization. A day later the nalidixic acid was stopped and there was thereafter considerable improvement, although another week passed before she was completely normal. An E.E.G. at this time was essentially normal, but during the record the patient had a hallucinatory episode during which there was demonstrated an increase of theta activity, abolished by opening the eyes. A repeat E.E.G. at a later date was entirely normal, though it is probably significant that the frequency of the alpha rhythm showed an increase of one cycle per second.

It would seem possible that the administration of nalidixic acid during the oedematous phase of acute nephritis precipitated this psychosis. Such an occurrence bears a relation to the previous reports of intracranial hypertension, although in this case there was no direct evidence.—We are, etc.,

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M. WALTON.
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Goodpasture's Syndrome and Dialysis

SIR,—The following case history supports the use of peritoneal dialysis in Goodpasture's syndrome, as mentioned by Dr. J. F. Munro and others (14 October, p. 95).

A 36-year-old woman was admitted in August 1966 with pyrexia. She had previously enjoyed good health and had borne five children with no apparent medical complication. She had noticed haemoptysis for the previous two weeks.

A postero-anterior chest x-ray showed bilateral diffuse mottling in the mid and lower zones. Haemoglobin was 6 g./100 ml. and the R.B.C.s showed the features of an iron deficiency anaemia. There was no other site of blood loss. The W.B.C. was 9,600/cu. mm.; the E.S.R. 125 mm. in one hour, and the blood urea 175 mg./100 ml.; serum proteins were 4.6 g./100 ml. with an albumin of 2.9 and a globulin of 1.7. Lupus erythematosus and rheumatoid arthritis screening tests were negative. Her sputum contained many macrophages laden with haemosiderin but no mycobacterium. The urine contained a moderate amount of protein. R.B.C.s and casts could be seen on microscopy.

Despite her sex, these features are in keeping with Goodpasture's syndrome. She was started on prednisone. Over the ensuing days haemoptysis continued and she complained of increasing dyspnoea. Her urine output fell. The blood urea slowly rose to 390 mg./100 ml., by which time she had become semi-conscious. Peritoneal dialysis was performed with dramatic subjective improvement and her urea fell to 222 mg./100 ml.

During the next two weeks she ate a low protein diet and her blood urea fell still further to 42 mg./100 ml. Haemoptysis ceased.

She then started to develop considerable oedema despite a reasonable fluid output and was prescribed frusemide. She commenced a normal diet. Average proteinuria was 1 g. per day, using Esbach's method at the time of discharge. Since that time she has remained in reasonable health up to August of this year, although her blood urea has slowly risen to 100 mg./100 ml. Episodes of haemoptysis have been infrequent.

The original peritoneal dialysis was lifesaving during a critical part of her illness. She has recently been readmitted complaining of dyspnoea. Her haemoglobin is 10 g./100 ml. The blood urea has risen to 300 mg./100 ml.—I am, etc.,

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Cowpox and Paravaccinia

SIR,—I read your leading article "Cowpox and Paravaccinia" (November 11, p. 308) with great interest. How does the cow's udder become infected? What is the direct source of infection? Many country folk, and others, have quaint notions about this.