Some of the mental changes are suggestive of cerebral ischaemia.

Depression and loss of libido were found to be more frequent with strongly progestogenic low oestrogen compounds, and these have a high level of endometrial monoamine oxidase activity for most of the cycle.[3] Of the recommended pills, Anovlar, Volidan, Gynovlar, and Minilyn are probably the most progestogenic and therefore likely to have a high incidence of depression. However, the dose of oestrogen is important, as Lyndiol 2·5 and Minilyn cause more depression than Lyndiol, 5 mg.

While the depression per se could often be ameliorated by changing to a high-dose oestrogen product, this is no longer advisable because of the increased thrombosis risk.[4] It also does not seem wise to resort to antidepressant drugs and even, as happens, E.C.T., without first making a serious attempt to provide an acceptable alternative method of contraception. This is not simple, as the young unmarried student has based her way of life on a "safe" method of contraception, while the older woman may be reluctant to make the effort to revert to mechanical methods or consider sterilization. Nevertheless, we cannot ignore the responsibility which is inherent in prescribing oral contraceptives because of the fear of an unwanted pregnancy.

Baumblatt and Winston advocate further research following their encouraging results for treating the mood changes with pyridoxine,[5] and it seems important to find out if pyridoxine can also help the vascular aspects of the syndrome.—I am, etc.,

ELLEN C. G. GRANT.

New Malden,
Surrey.

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Herpes Infection after Transplantation

SIR,—We write with reference to the paper by Dr. E. S. Spencer and Dr. H. K. Andersen on infections with herpes virus following renal transplantation (1 August, p. 251). It would be interesting to know whether any of the patients developing a rise in cytomegalovirus complement-fixing antibody received fresh blood preceding, or at the time of, transplantation—as transmission of cytomegalovirus by fresh blood is well documented.[1-4] Further, in a series of 56 patients receiving fresh blood a fourfold rise of cytomegalovirus complement-fixing antibody was demonstrated in 38%, usually 6-8 weeks after the blood was given.[5]

The statement "that five out of seven patients thought to have cytomegalovirus infections had virus-neutralizing antibodies in their serum before the onset of symptoms" suggests that two of the patients had not previously been infected with cytomegalovirus—and therefore these could not have been due to reactivation of the virus, but must be primary infections. Our own series showed that 55% of the infected patients were seronegative to cytomegalovirus complement-fixing antibody prior to transfusion, while 45% were seropositive. Therefore though reactivation may be responsible for rising cytomegalovirus complement-fixing antibody following immunosuppression or fresh blood transfusion our work suggests that many cases are due to primary infection. Furthermore infection with cytomegalovirus of a different strain may occur in patients with pre-existing complement-fixing antibody.

Another point of interest is the fact that these patients on immunosuppressive and steroid therapy were capable of producing antibody in response to viral infection. This no doubt explains why their symptoms were those of a healthy adult with a similar infection, and not the disseminated type of infection that tends to occur in immunosuppressed patients.[6]—We are, etc.,

G. R. CAIRD.
R. S. E. WILSON.
United Bristol Hospitals.

E. O. CAUL.
Public Health Laboratory, Service, Bristol.

T. G. M. PERHAM.
Frenchay Hospital, Bristol.

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Hypotension caused by L-Dopa

SIR,—The hypotension produced by L-dopa during treatment of Parkinson's disease (21 February, p. 474) has been attributed by Professor J. H. Burn (7 March, p. 629) to a replacement by dopamine of some of the noradrenaline in the sympathetic fibres. This reduces the effect of the vasoconstrictor activity of the sympathetic nervous system on the peripheral resistance vessels. Recently, Dr. R. C. Duvoisin (4 July, p. 47) has described the use of propranolol in the treatment of the hypotension produced by L-dopa, on the basis that propranolol would antagonize the effect of the dopamine since "dopamine appears to have predominantly beta-adrenergic properties."

This hypothesis is not correct, and does not explain the mechanism by which propranolol might counteract the hypotension induced by L-dopa. The actions of dopamine are complex. It stimulates adrenergic beta receptors in the heart but not in the peripheral resistance vessels. The fall in systemic vascular resistance produced by dopamine results largely from renal vasodilatation. The mechanism of this is not understood but it is not antagonized by a beta-receptor blocking drug. Dopamine increases femoral vascular resistance.[1] Consequently it would not appear possible for propranolol to antagonize the hypotension produced by L-dopa through blockade of adrenergic beta-receptors.

Before the acceptance of propranolol for the treatment of the hypotension produced by L-dopa, more detailed clinical pharmacological studies on this subject are required.—I am, etc.,

R. G. SHANKS.

Department of Therapeutics and Pharmacology,
Queen's University,
Belfast.

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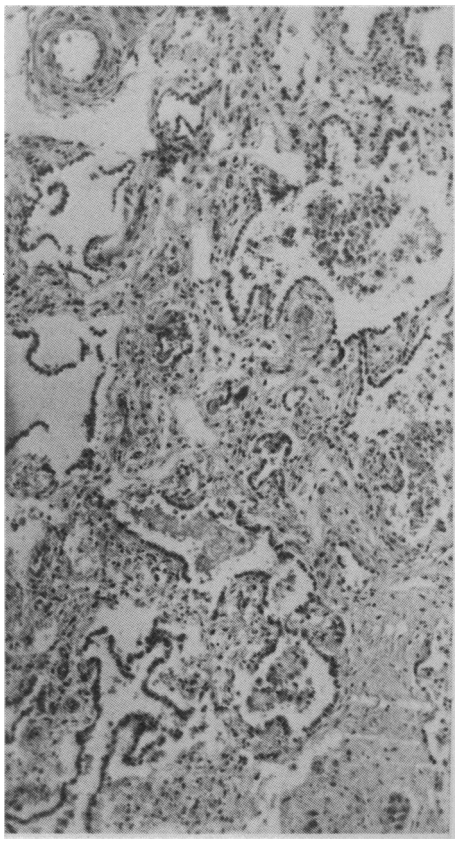
Pulmonary Veno-occlusive Disease

SIR,—In 1966 Brown and Harrison[1] found eight reported cases of pulmonary veno-occlusive disease. They added a ninth, later the subject of a clinico-pathological conference.[2] In 1967 Liebow *et al*,[3] presented 16 cases to the American Heart Association. We wish to report an additional case.

A 40-year-old female gave a history of childhood bronchitis and asthma. Skin tests in 1953 revealed a wide range of sensitivity. Chest x-ray was normal. In 1963 the right middle lobe collapsed. Bronchoscopy was normal and the lobe re-expanded; subsequently she noted increasing exertional dyspnoea. A bronchogram in September 1967 showed mild fusiform bronchiectasis in the right lower lobe, and by December 1967 she was admitted to hospital with severe dyspnoea. Chest x-ray showed a right pleural effusion, a generalized increase in pulmonary vascularity, and a normal heart size and shape. E.C.G. revealed right atrial and ventricular hypertrophy. Examination of the pleural effusion was



unhelpful, and all significant tests were normal. The patient remained very breathless, even after aspiration of two litres of fluid. Steroid therapy produced rapid general improvement and clearance of the fluid.

She was discharged on steroids but one month later (January 1968) fluid had re-accumulated. For the first time some definite bronchospasm