published.[3] The failure to demonstrate higher levels of this activity in our mouse test—injecting the animals with *chronic* idiopathic thrombocytopenic purpura plasma—needs to be further elucidated.

It was a pity that Dr. Penington did not classify his positive cases. Although deficiency in feed-back inhibition could be considered in thrombocytopenia, my colleagues demonstrated that the giant-cell content of the protected areas of *x*-ray treated mice can be increased with "positive" plasma (received after irradiation) even in animals adjusted to normal platelet levels by means of syngeneic thrombocyte transfusions.[4]

Comparing the thrombopoietic effects of from 0·4 to 2·0 ml. subcutaneously administered plasma samples using his isotope bioassay method Dr. Penington states that "the results presented are the first in which a dose-response relationship has been established." We investigated dose-response curves in several cases.[2] There were five different doses from 0·015 ml. to 0·240 ml. given intravenously. Platelet count increases higher than those quoted by Dr. Penington were produced five to eight days after the plasma injection. Bone marrow magakaryocytes were also investigated. At least some pathologically active human plasma samples initiated a significant bone marrow and splenic megakaryocytosis, with a shift to the left in recipient mice already one day after the appropriate stimulus. The same pattern of changes followed the administration of "positive" mouse plasma. Several conditions of platelet physiopathology were, later, investigated and bone marrow changes, in general, were in accordance with expectations, except post-splenectomy thrombocytosis.[5]

It is surely unfair to disparage extensive previous work because some preliminary results with a seemingly more precise method diverged; especially as Penington's new method, used independently by others,[6] appears to be a real advance.—I am, etc.,

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Glaucoma

SIR,—I feel sure that practitioners who have read Mr. S. Romano's article on primary glaucoma (16 May, p. 407) will be grateful for his conciseness and clarity. He has, however, confined the article to glaucoma in the adult.

I think a description of primary glaucoma ought to remind the reader of the congenital form, which is not an uncommon condition. It may be classified apart from glaucoma associated with rarer diseases occurring in fetal or neonatal life, and which diseases exhibit either purely ocular defects or these together with systemic abnormalities.

Most cases of congenital glaucoma are sporadic but a small percentage show an autosomal recessive trait. Mechanical impairment of aqueous outflow, with a resultant rise in intraocular pressure, produces early symptoms and their recognition is important. They appear before the physical signs and are an intolerance of bright light, blepharospasm, and epiphora. Later the signs appear of a hazy cornea in an enlarged eye which feels hard when palpated, becomes somewhat injected, and in which the optic disc may be seen to be cupped. The treatment is essentially surgical, supplemented where necessary by miotics, and the correction of refactive errors.

Since the prognosis depends on not only early treatment but also early recognition of the disease, one should therefore consider the possibility of congenital glaucoma when one is asked to see an infant who is persistently photophobic and irritable, and an ophthalmic opinion may be very welcome.—I am, etc.,

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Illustrated Lectures

SIR,—Having just returned from a medical conference at which the majority of the speakers used slides during their talks, I wonder if I might have the courtesy of your columns to draw attention to the uselessness of the bulk of these slides because of their illegibility.

Since many participants at such conferences travel far and at much—usually public—expense in order to attend, it would seem desirable that as much as possible of what is addressed to them should be assimilable.

High quality slides require the specialist medical illustrator's skill. But if the following points are borne in mind, any speaker can produce useful slides.

Make as *few* points per slide as possible; more than five is probably too many. Use several slides instead.

Represent the information in diagrammatic form if at all feasible, rather than in words and numbers. Simple histograms and graphs are most effective, using thick lines. Omit everything that is not essential.

If using a typewriter, the area to be photographed should be no larger than 3·5 in. × 5 in. (appr. 9 cm. × 12·5 cm.)—assuming a pica typeface with 10 characters to the inch. An electric typewriter with carbon ribbon is most desirable.[1]

Lettering on larger originals than the size mentioned should be clearly legible when the original is viewed from a distance equal to eight times its largest (that is diagonal) dimensions—for quarto paper, from over 4 yards (3·7 m.).

Positive—that is black on white—slides are on the whole more legible than negative.[2]—I am, etc.,

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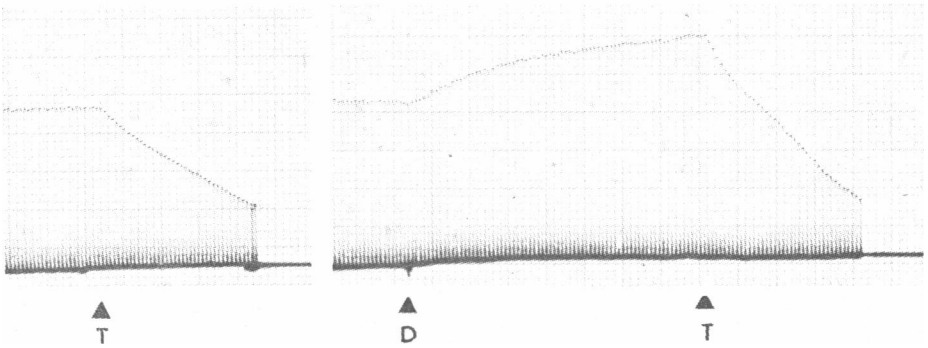
Diazepam and Neuromuscular Transmission

SIR,—Drs. S. A. Feldman and B. E. Crawley have recently reported that diazepam potentiates the neuromuscular blockade produced by gallamine (9 May, p. 336). This is in contrast to the findings of Hunter[1] who investigated diazepam and tubocurarine.

In order to determine whether diazepam affects transmission at the neuromuscular junction we have investigated its activity upon the rat phrenic nerve-diaphragm preparation in vitro. Diazepam in concentrations between 2 and 16 x 10^{-5}M potentiated tage inhibition was compared with the original control contraction height prior to diazepam pretreatment. However if this percentage inhibition was expressed in terms of the contraction height after diazepam pretreatment a slight potentiation of neuromuscular blockade was seen (13%).

In order to determine whether diazepam increased contraction height pre- or post-



Neuromuscular blockade produced by tubocurarine before and after pretreatment with diazepam. (T = D-tubocurarine 10^{-4}M; D = diazepam 4 × 10^{-5}M.)

contractions elicited by supramaximal stimulation of the phrenic nerve. This effect was dependent upon concentration and, at the highest concentration examined, a 50% increase in contraction height was seen.

Neuromuscular blockade produced either by tubocurarine or by gallamine was not potentiated by diazepam when the percen- synaptically diazepam was added to the tissue bath with a high concentration of tubocurarine, and the muscle was stimulated directly. A sustained increase in contraction height was observed as before.

It is concluded that diazepam does not potentiate the effects of competitive neuromuscular blocking agents in vitro in