and potentially a number of pharmacological parameters could be represented.

We have modified the logistic curve to a more general form in order to define the parameters in terms of a number of useful practical pharmacological entities:

$$R(X) = \frac{A + DX^B}{1 + CX^B} \qquad (2)$$

This curve has the right characteristics, namely sigmoid shape. Two theoretical dose-response curves are shown in Figure 1 for drugs working by a similar mechanism but of different potency.

For the two response curves to be parallel they must both have the same asymptotic value and the same value of B. The asymptotic value of R(X) indicates the maximum achievable response and is where

$$R(X) = R(\infty) = \frac{D}{C}.$$

The control response is where $R(X) = R(O) = A$.

$$\left(\frac{1}{C}\right)^{1/B}$$

is the ED_{50} and if B is the same value for the two curves the relative potency RB is

$$\left(\frac{C_1}{C_2}\right)^{1/B}.$$

Examples are shown for data having a Poisson distribution (from the acetylcholine writhing test), to data having a negative-exponential distribution (from the Roto-rod test), and for estimating potency of inhibitors of histamine release where the data was intractable by ordinary methods.

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A postsynaptic action of prostaglandin E_1 on sympathetic responses in guinea-pig ileum

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Prostaglandins inhibit the response of many tissues to sympathetic stimulation. It is now well established that one possible mechanism involved is a reduction of transmitter release (Hedqvist, 1969), but a postsynaptic action may sometimes also play a part (Clegg, 1966).

We have investigated the actions of prostaglandin E_1 on the responses of isolated guinea-pig ileum to stimulation of the perivascular nerves. The ileum was stimulated transmurally with just maximal stimuli at a frequency of 0.1 Hz. The perivascular nerve was stimulated with 8 s trains at a frequency of 1–32 Hz, immediately before each coaxial pulse (Watt, 1971). The frequency of perivascular stimulation required to produce a 50% inhibition of the coaxial twitch was increased by a factor of 1.5 ± 0.15 (mean ± s.e. mean $n = 10$ $P < 0.005$) in the presence of prostaglandin E_1 (57 nM). In similar experiments in which the coaxial twitch was inhibited by exogenous noradrenaline, prostaglandin E_1 (28 nM) increased the dose of noradrenaline necessary to produce a 50% inhibition by a factor of 2.3 ± 0.4; $n = 8$ ($P < 0.003$).

The effects of perivascular stimulation and of noradrenaline were also compared before and after treatment with indomethacin (7 μM). The frequency of perivascular stimulation then required to inhibit the coaxial twitch by 50% was 0.5 ± 0.2 of the frequency required to produce an equal inhibition of the untreated preparation. This effect of indomethacin was significant ($P < 0.015$).

Indomethacin also significantly reduced the concentration of noradrenaline necessary to inhibit the coaxial twitch by 50%; the concentration required being 0.3 ± 0.1 as great as that required in the untreated preparation ($P < 0.0002$). The results obtained with noradrenaline indicate that in the isolated guinea-pig ileum prostaglandin E_1, as well as any presynaptic action, has a postsynaptic action. The relatively greater effects both of prostaglandin E_1 and of indomethacin on the responses to noradrenaline than on the responses to perivascular stimulation, suggest an action of prostaglandin on adrenoreceptors

which are not accessible to neurally released noradrenaline. One site for this action may be the β-adrenoceptors situated on the longitudinal muscle (Kosterlitz & Watt, 1965). Most of the sympathetic fibres terminate in Auerbach's plexus (Norberg, 1964) and the response to perivascular stimulation is hardly affected by β-adrenoceptor antagonists (Watt, 1971).

Possible explanations for the greater part of the postsynaptic action of prostaglandin are potentiation of the response to acetylcholine (Harry, 1968, Kadlec, Masek & Seferna, 1974) or an increase in the acetylcholine released from the Auerbach's plexus. However, in our experiments the response of guinea-pig ileum to acetylcholine was not affected by indomethacin (7 μM) confirming the findings of Bennett, Eley & Stockley (1975) and indicating that the effects observed in the presence of indomethacin were not due to inhibition of the response to acetylcholine.

G.J.S. is a Science Research Council scholar.

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The effect of subcutaneous injections of adrenaline on platelet MAO activity

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Gentil, Greenwood & Lader (1975) have recently reported large increases in platelet monoamine oxidase (MAO) activity following the subcutaneous injection of adrenaline and suggested that "stress" might produce rapid changes in the activity of the enzyme. In view of the relevance of this finding to the conflicting reports of a reduction of platelet MAO activity in schizophrenia (Murphy & Wyatt, 1972; Meltzer & Stahl, 1974; Shaskan & Becker, 1975; Bailey, Crow, Johnstone & Owen, 1975) we have attempted to verify the observations of Gentil and his colleagues and to improve the experimental design by (a) more frequent blood samples using an in-dwelling

Table 1 The effect of adrenaline and placebo on platelet MAO activity

Benzylamine concentration	*Sample*	*Injection received*		
		Placebo (n=6)	*Adrenaline (n=6)*	* t
1 mM	Pre-injection	27.8 ± 2.7	33.2 ± 6.3	—
1 mM	+20 min	27.7 ± 3.9	37.3 ± 5.9	4.61†
1 mM	+40 min	31.0 ± 4.8	33.5 ± 5.6	0.32
1 mM	+60 min	29.4 ± 4.3	33.7 ± 6.4	0.21
1 mM	+80 min	29.8 ± 4.3	35.1 ± 6.6	0.11
2.1×10^{-5} M	Pre-injection	1.70 ± 0.30	1.82 ± 0.44	—
2.1×10^{-5} M	+20 min	1.82 ± 0.26	2.08 ± 0.37	1.93
	Age (yrs)	28.2 ± 5.6	27.5 ± 3.0	0.25

* *'t'* values refer to a comparison between pre- and post-injection samples of subjects receiving adrenaline only. † $P < 0.05$.
Results, mean ± s.d. expressed as nmoles product formed per mg protein 30 min^{-1}.