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# Adrenergic control of tendon jerk reflexes in man

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**Summary**

1. Tendon jerk responses and H reflexes were recorded from conscious human volunteers before and after intravenous injection of methylamphetamine, thymoxamine and propranolol, and during intravenous infusion of noradrenaline.

2. Methylamphetamine produced a significant increase in the amplitude of the tendon jerk, whereas noradrenaline had no effect in doses which caused a greater pressor response than methylamphetamine.

3. Thymoxamine produced a dose-related reduction in the tendon jerk.

4. Propranolol had no significant effect on the jerk.

5. None of these drugs significantly affected the H reflex.

6. It is suggested that central adrenoceptors, possibly $\alpha$ in type, exist in man, and that stimulation of these receptors facilitates tendon jerk reflexes by an action on the fusimotor system.

## Introduction

Studies of the distribution of catecholamines in the cat spinal cord by histochemical fluorescence techniques have demonstrated that noradrenaline-containing nerve terminals form close contact with the cell bodies and dendrites of spinal motoneurones (Dahlström & Fuxe, 1965) and that these terminals disappear following lesions of the descending tracts (Andén, Haggendal, Magnusson & Rosengren, 1964). These lesions also lead to changes in the fluorescence of cell bodies in the medulla oblongata and mesencephalon (Dahlström & Fuxe, 1965), suggesting that the cells give rise to a descending noradrenergic pathway controlling motor activity. This concept is supported by the pharmacological studies of Ellaway & Pascoe (1966, 1968) in which drugs enhancing adrenergic transmission, such as methylamphetamine and desmethylimipramine, increased the fusimotor discharge evoked by stimulation of descending tracts in the spinal rabbit, while adrenoceptor blocking drugs, such as chlorpromazine and phenoxybenzamine, produced the opposite effect.

The results of experiments with sympathomimetic drugs on spinal reflexes in the whole animal have been conflicting. Schweitzer & Wright (1937) found a transient facilitation of the knee jerk in cats during the pressor response to large intravenous doses of adrenaline, but this soon gave way to a long lasting depression. However, Sigg, Ochs & Gerard (1955) found that with lighter planes of anaesthesia the depressant effect was replaced by facilitation. Unanaesthetized animals have given similar responses (Wilson, 1956; Kissel & Domino, 1959).

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The purpose of the work described in this paper was (a) to investigate the effects of intravenous methylamphetamine and noradrenaline on monosynaptic reflexes in conscious human subjects, (b) to determine whether these effects were produced by stimulation of $\alpha$- or $\beta$-adrenoceptors, and (c) to localize the site of action of the drug to the $\alpha$- or $\gamma$-motoneurone system. (For a preliminary account of this work see Phillips, Richens & Shand, 1970).

**Methods**

An apparatus was designed which allowed recordings to be made of the contraction of the gastrocnemius-soleus muscle resulting from a standard tap to the Achilles tendon, and the electromyographic response (E.M.G.) of the muscle following an electrical stimulus to the medial popliteal nerve (H reflex). The subject was seated in a specially-designed chair with a reclining back, and his foot strapped to a footplate so that the knee and ankle joints were approximately at right angles. The footplate was hinged about the axis of the ankle joint, so that the contraction of the calf muscle could be recorded isometrically or isotonically. In practice, isometric recording was found to be more satisfactory so this was used throughout. The force of contraction was measured with a Grass FT 10C force-displacement transducer with a maximum range of 500 mg–10 kg. This was mounted in a heavy wooden block and was connected to the footplate by a rigid rod. The wooden block also served as a stop to prevent the knee from lifting during a contraction of the calf muscles. Taps were applied to the Achilles tendon with a hinged hammer which was allowed to fall through a 60° arc under the force of gravity before striking the tendon. Although this provided a mechanical stimulus which was quite constant in force, the state of relaxation of the subject's muscles determined to some extent the resulting stretch of the muscle spindles, and hence the amplitude of the reflex contraction. Subjects were instructed to relax during the recordings, but some found this difficult and produced responses which varied greatly in amplitude. Others, however, gave responses which varied by no more than 10%. In practice, it was found reliable to take the mean of 10 responses elicited randomly at a mean interval of 3–4 seconds. The hammer was provided with a trip switch which triggered a Tectronix 561B oscilloscope on which the recordings were made. Photographic negatives were taken with a Shackman AC 2/25 camera and subsequently projected for accurate measurement. The reflex E.M.G. response to the tendon tap was also recorded with superficial electrodes, but as there was not a strict correlation between the amplitudes of the E.M.G. response and mechanical contraction, possibly from rotation of firing in motor units or changes in synchrony, the contraction response was used as a measure of reflex activity.

H reflexes were elicited by an electrical stimulus to the medial popliteal nerve through silver button stick-on electrodes of the type used for electroencephalographic recording. One electrode was applied over the patella while the second (the cathode) was placed over the popliteal fossa overlying the medial popliteal nerve. A pad of cotton wool strapped over the electrode served to approximate the electrode and nerve more closely when the subject adopted a sitting posture. The impedance of the electrode was checked on a Mingograf recorder and adjusted to match the high impedance output (5 K$\Omega$) of a Disa type 14 E11 stimulator. Square wave stimuli of 1 ms duration were used. Similar electrodes were applied to the medial side of the belly of the gastrocnemius-soleus muscle for recording the surface E.M.G. Stimuli

of gradually increasing intensity were applied to the nerve to elicit the low-threshold, long-latency reflex response (H reflex) resulting from stimulation of Ia afferent fibres, and the higher-threshold, short-latency response (M response) resulting from stimulation of the $\alpha$-motor fibres (see Matthews, 1970, for references to this technique). Three responses were elicited over 10 s at each stimulus intensity and the mean amplitude of the potentials plotted against the stimulus intensity.

The maximum amplitude of the H reflex before and after drug administration was taken from the plot and was used as a measure of $\alpha$-motoneurone excitability (see **Discussion**). Although the amplitude of the M response was dependent on neuromuscular transmission, the isometric tension (contraction) developed by the muscle at a stimulus intensity which occluded the H reflex was found to be a more convenient measure of neuromuscular integrity. The stimulus to the nerve to produce an M contraction was not supramaximal on some occasions, but the responses were found to vary little during the course of the experiments.

The study was performed as a series of experiments each of which involved six normal volunteers in a double-blind, balanced, randomized design of two or three procedures, each separated by an interval of one week. The following drugs were given by intravenous injection over two minutes: methylamphetamine hydrochloride, thymoxamine hydrochloride (Opilon, William Warner), (±)-propranolol hydrochloride (Inderal, ICI), levodopa (Larodopa, Roche) and phentolamine mesylate (Rogitine, Ciba). Normal saline (0·9% w/v NaCl solution) was used as a placebo injection. (−)-Noradrenaline tartrate (Levophed, Winthrop) was administered by intravenous infusion, diluted to 4 $\mu$g/ml in 5% dextrose solution, and given via an Avon A23 oxytocin infusion set. The infusion rate was controlled by a Watson-Marlow roller pump, and was increased in 1 ml steps from 1 to 4 ml/minute. Five per cent dextrose was used as placebo infusion.

A set of control records was taken before the injection, 5 min afterwards, and sometimes serially up to 30 minutes. When infusions of noradrenaline were being given records were taken after a steady blood pressure had been reached at each infusion rate. This was usually 4–5 min after changing the rate. The amplitude of the tendon jerk responses after administering the drug was expressed as a percentage of the control amplitude. The responses of the six subjects in each experiment were averaged and the significance of the difference between the means after active drug and placebo treatments was calculated with Student's *t* test, with correction for unequal variance where necessary. The maximum H reflex response, M contraction, mean blood pressure and heart rate were analysed in the same way. Mean blood pressure was calculated by adding one-third of the pulse pressure to the diastolic value.

## Results

### *Tendon jerks*

The apparatus used in these experiments measured the tension produced by downward pressure of the ball of the foot on the footplate. It was not possible to define the relationship between this measure and the actual tension occurring in the gastrocnemius-soleus muscle, because it depended upon a variety of factors, including leverage around the ankle joint and the visco-elastic properties of the tissues making up the joints of the foot. The peak tension recorded by the transducer during the jerk responses varied widely between subjects, ranging from 0·5 to

6·8 kg for the control jerks. The mean for all the subjects who participated in these experiments was 3·6 kg. Because there was such a wide variation between subjects, and because it was not possible to standardize exactly the recording conditions from day to day in the same subject, it was decided to convert all the responses to a percentage of the control amplitude for each subject.

The accumulated results of several series of experiments are shown in Figure 1.

*Saline*

The tendon jerks 5 min after an injection of saline (Fig. 1) were usually slightly smaller than the control responses in most subjects, although considerable variation was seen. Two subjects received saline on four occasions, and 5 min after the injection the mean jerk amplitude in one subject varied from 80 to 112% of the control level and in the other subject from 80 to 103% of the control. The mean change calculated from 24 observations in 16 subjects was to $92 \pm 5{\cdot}4\%$ of the control level.

*Methylamphetamine*

Five minutes after i.v. injection of 0·2 mg/kg the tendon jerk responses had increased by 64% over the pre-drug value when compared with the responses after an injection of saline (Fig. 1). This increase was significant ($P<0{\cdot}025$). The mean blood pressure had increased by $19 \pm 3{\cdot}6$ mmHg to 103 mmHg. The effect on the heart rate was variable, one subject showing a marked bradycardia (−17 beats/min) and another a marked tachycardia (+18 beats/min).

*Noradrenaline*

Intravenous infusion of up to 16 $\mu$g/min of noradrenaline produced no significant change in the amplitude of the tendon jerk despite a pressor response which exceeded that of methylamphetamine. The mean blood pressure rose by $28 \pm 3{\cdot}2$ mmHg to 112 mmHg during the infusion of 16 $\mu$g/minute. The heart rate slowed progressively as the infusion rate was increased, reaching 53 beats/min at 16 $\mu$g/min compared with a control rate of 69 beats/minute.

*Thymoxamine*

Placebo-controlled studies were performed at two dose levels, 0·05 and 0·1 mg/kg. The smaller dose produced a fall to $55 \pm 10{\cdot}8\%$ of the pre-drug jerk amplitude, but this was not significant ($P<0{\cdot}1$). The larger dose produced a fall to $38 \pm 5{\cdot}0\%$, which was highly significant ($P<0{\cdot}005$). A further 12 observations (Figs. 1 and 3) with a dose of 0·2 mg/kg (without saline control) produced a fall to $29 \pm 6{\cdot}5\%$ of the pre-drug level. Although no control was used, comparison of these results with 24 saline controls from other experiments (mean fall to $92 \pm 5{\cdot}4\%$) gave a difference which was highly significant ($P<0{\cdot}001$). A total of 30 observations were made after injections of thymoxamine in this series of experiments, and only on one occasion (with the low dose) was a fall in amplitude of the jerk not seen. Figure 2 shows the time-course of the depression of jerk responses after injection of 0·1 mg/kg of thymoxamine compared with saline. The subjective symptoms (nasal stuffiness, conjunctival injection and a feeling of fullness in the head) had usually disappeared 1 h after the injection. No significant change in blood pressure was

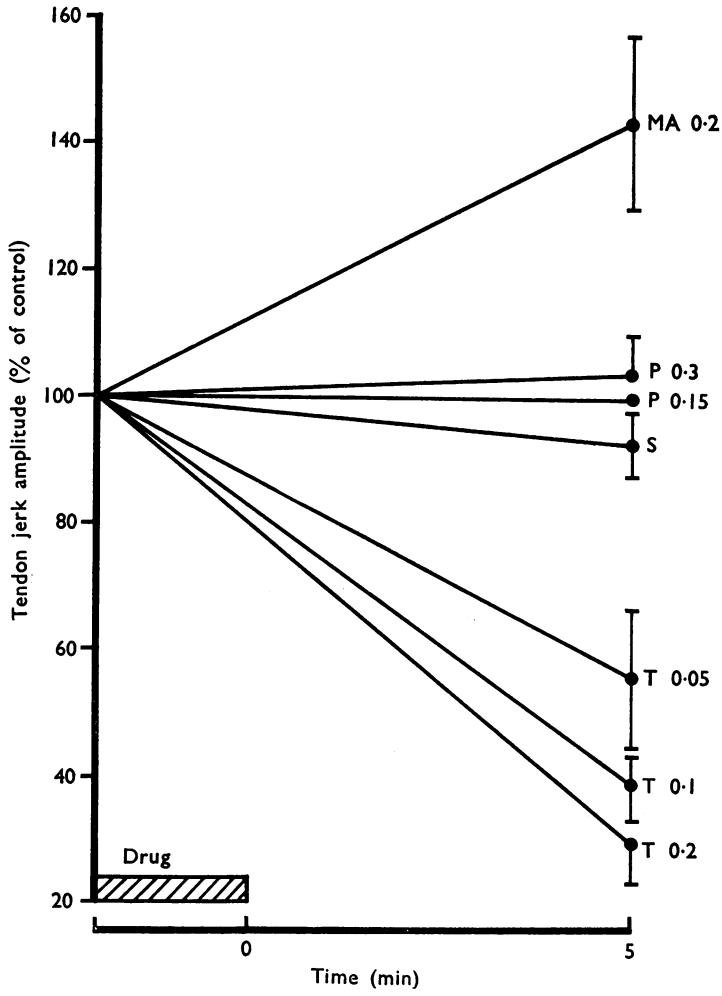


FIG. 1. Accumulated data on tendon jerk responses. Drugs and doses in mg/kg are signified by letters and numbers (MA=methylamphetamine, P=propranolol, S=saline, T=thymoxamine). The points 5 min after drug administration were calculated as a percentage of the pre-drug responses. The values after the high doses of propranolol and thymoxamine were based on observations in 12 subjects, and the value after saline was based on 24 observations in 16 subjects. All other points are mean observations in 6 subjects. The bars represent one S.E.

produced by the two lower doses ($-2{\cdot}4 \pm 2{\cdot}6$ mmHg with 0·05 mg/kg and $+2{\cdot}7 \pm 2{\cdot}7$ mmHg with 0·1 mg/kg), but a slight increase was usual in the heart rate. With 0·2 mg/kg a precipitous fall in blood pressure, accompanied by symptoms of hypotension, was seen in two subjects. Bradycardia occurred during these episodes, suggesting a ' vaso-vagal ' origin for them.

*Propranolol*

Injections of 0·15 mg/kg and 0·3 mg/kg of propranolol produced no significant change in the amplitude of the tendon jerk. No significant change in blood pressure was seen with either dose, but the heart rate invariably fell ($-9{\cdot}0 \pm 2{\cdot}0$ beats/min with 0·15 mg/kg, $P<0{\cdot}01$, and $-11{\cdot}0 \pm 1{\cdot}8$ beats/min with 0·3 mg/kg, $P<0{\cdot}001$).

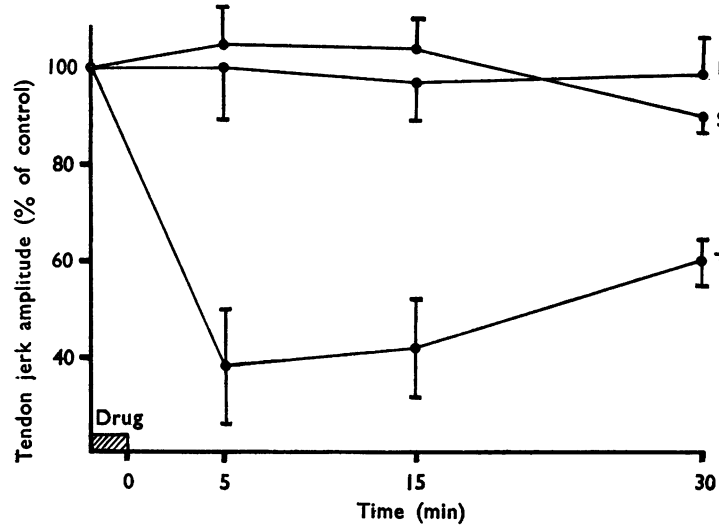


FIG. 2. Time course of the effect of thymoxamine on the tendon jerk. The effect of 0·1 mg/kg of thymoxamine (T) in six subjects was compared with the effect of a control injection of saline (S). The effect of propranolol (P) was not significantly different from saline. The bars represent one S.E.

*Other drugs*

In order to test the effect of another agonist drug which is known to cross the blood-brain barrier, levodopa was injected i.v. on two occasions into one subject in doses of 0·3 and 1·2 mg/kg. Little change in the amplitude of the jerk was seen with either dose, and as nausea was produced, especially with the higher dose, no further experiments were performed with this drug. The $\alpha$-adrenoceptor blocking drug, phentolamine, was given i.v. to one subject (as two separate injections each of 0·1 mg/kg) but only agonist properties of the drug were seen, namely a slight rise in mean blood pressure (7 mmHg) and an increase in heart rate (20 beats/min). The tendon jerks increased following each injection, the increase being to 128% of the control response after the second injection. No further experiments were performed with this drug.

*Blocking experiments*

As methylamphetamine and thymoxamine produced changes in the ankle jerk of opposite sign, it was of interest to determine whether $\alpha$-adrenoceptor blockade with thymoxamine would prevent the stimulant effect of methylamphetamine. The results of one series of experiments in six subjects are illustrated in Figure 3. The tendon jerks were measured 5 min after an injection of 0·2 mg/kg of thymoxamine, and again 5 min after an injection of either 0·2 mg/kg of methylamphetamine or a similar volume of saline. Although the increase produced by methylamphetamine never approached the control level, there was a significant difference ($P<0·05$) between these responses and those after saline. Thymoxamine, however, had failed to block the pressor response to methylamphetamine (Fig. 3).

It was considered that this failure might have been the result of allowing too long an interval between the injections, which might have led to a waning of the

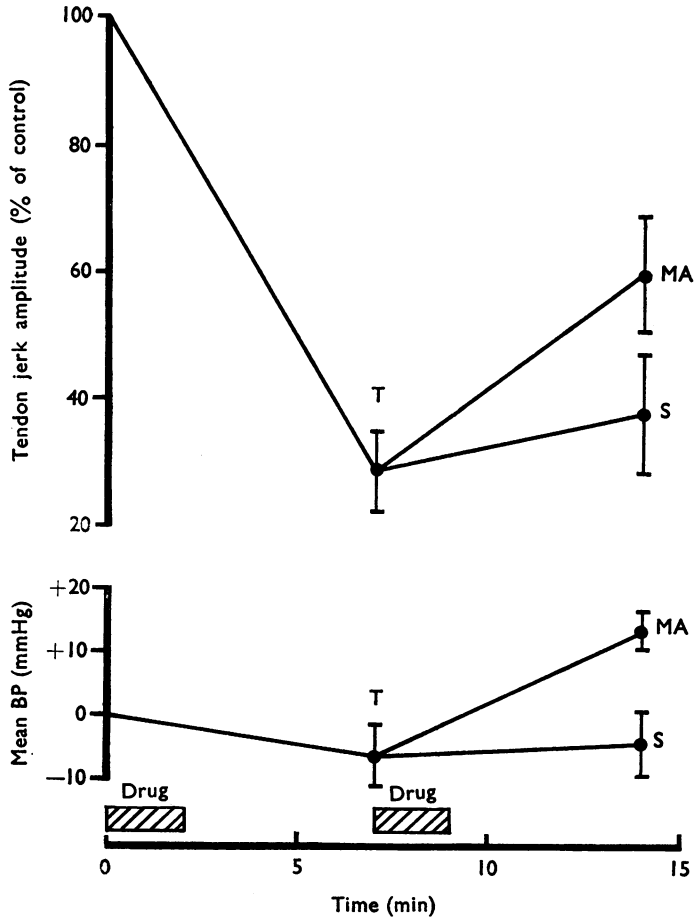


FIG. 3. Effect of thymoxamine on the responses to methylamphetamine. Tendon jerks and blood pressure were measured after an injection of 0·2 mg/kg of thymoxamine (T), and again after a second injection of either saline (S) or 0·2 mg/kg of methylamphetamine (MA). The points represent the mean ± S.E. of the observations in six subjects.

brief $\alpha$-adrenoceptor blockade produced by thymoxamine. Another procedure was therefore adopted in which a 2 min injection of 0·2 mg/kg of thymoxamine was immediately followed by a 2 min injection of 0·2 mg/kg of methylamphetamine (or saline). Methylamphetamine reduced the magnitude of the fall in jerk amplitude by 18% (Fig. 4), but this was significantly different ($P<0·02$) from the fall after saline. However, the pressor response had again not been abolished by this procedure. As it seemed that thymoxamine was not a satisfactory antagonist to the $\alpha$-agonist properties of the dose of methylamphetamine used in these experiments no further attempts were made. In any case, a convincing demonstration of $\alpha$-blockade would have been shown only if *abolition* of the agonist response occurred, because the blocking drug itself produced a marked change in the baseline, making degrees of antagonism difficult to recognize. Phentolamine also appeared to be unsatisfactory, and it was considered unjustifiable to inject phenoxybenzamine into volunteers.

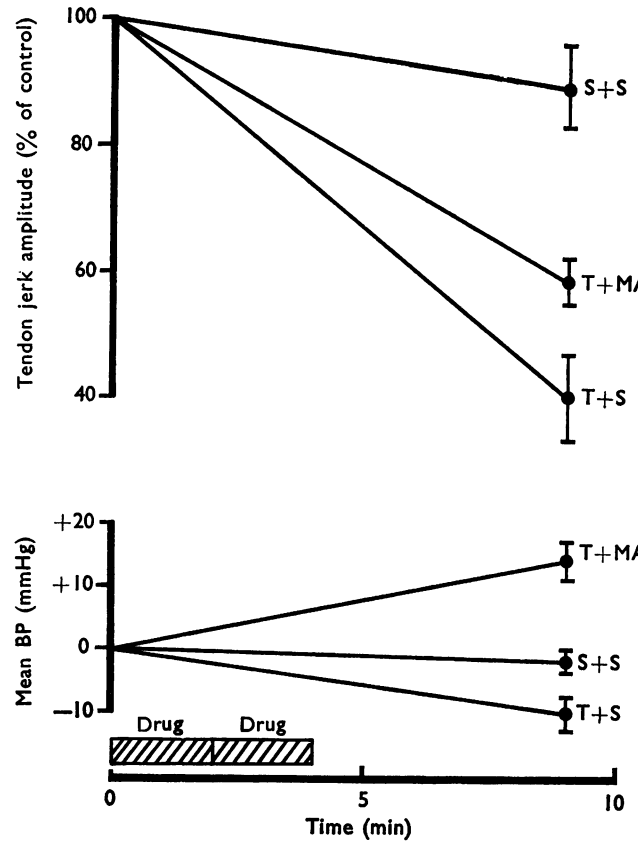


FIG. 4. Effect of thymoxamine on the responses to methylamphetamine. Six subjects were injected with saline+saline (S+S), 0·2 mg/kg of thymoxamine+saline (T+S) or 0·2 mg/kg of thymoxamine+0·2 mg/kg of methylamphetamine (T+MA) on three separate occasions. The points given are the mean ± S.E.

### *H reflex*

When the amplitude of the H reflex was plotted against stimulus intensity the maximum H reflex was found to be consistent for the duration of these experiments. The mean change 5 min after injection of saline was to 98 ± 1·6% of the control value (24 observations). The H reflex was most variable when close to threshold, but became progressively more stable with increasing stimulus intensity. The minimum interval of 2–3 s between stimuli gave responses of constant size when the peak amplitude of the response was reached, but between threshold and peak the second and third responses of a group of three were usually a little smaller than the first.

When the effects of methylamphetamine, thymoxamine and propranolol on the H reflex were examined no significant difference was found between the effects of any drug and those of saline. There was no evidence to suggest a selective effect on threshold responses and no change in the latency of the reflex was seen at any time.

### *M contraction*

The tension response of the muscle to stimulation of its motor nerve supply was recorded before and after administration of methylamphetamine, thymoxamine and propranolol. No significant change with any of these drugs was detected.

## Discussion

A change in amplitude of the tendon jerk produced by a drug can result from an action at one of several sites: (a) the synapse between Ia sensory fibres and the homonymous $\alpha$-motoneurones; (b) the muscle spindle receptors, changing the output of sensory information from the spindle; (c) the fusimotor ($\gamma$) neurone, changing spindle bias; (d) any of the descending tracts to $\alpha$- or $\gamma$-motoneurones; and (e) the neuromuscular junction. Some of these possibilities can be examined by recording the H reflex and M contraction at the same time as the tendon jerk. The M contraction, which results from direct stimulation of $\alpha$-motor fibres while the reflex volley from Ia afferent stimulation is blocked, tests neuromuscular transmission. The absence of a significant change in this response after administration of methylamphetamine or thymoxamine rules out a neuromuscular cause for the marked change in amplitude produced by these drugs.

There is considerable evidence that the H reflex results from activation of Ia afferent fibres from the annulospiral endings of the muscle spindle (Diamantopoulos & Gassel, 1965), and that its size is a measure of the excitability of the $\alpha$-motoneurone pool (Matthews, 1970). A change in its amplitude signifies a change in the excitability of the pool or an alteration in transmission across the single synapse between the Ia fibres and $\alpha$-motoneurones. It follows that a change in the amplitude of the tendon jerk without accompanying change in the H reflex suggests an alteration in spindle sensitivity. The marked increase in tendon jerks produced by methylamphetamine and decrease by thymoxamine occurred without significant change in the H reflex, and thus it is likely that a change in spindle sensitivity had occurred. This could have resulted from either a peripheral effect on the muscle spindle receptors, or an effect centrally on the fusimotor neurones, causing an alteration of spindle bias. But the failure of noradrenaline, which does not penetrate into the central nervous system, to alter the jerk amplitude makes a peripheral action unlikely, for this drug was administered in doses which produced a much greater stimulation of $\alpha$-adrenoceptors, as judged by its pressor effect, than methylamphetamine.

It seems likely, then, that methylamphetamine and thymoxamine were acting centrally by altering the discharge rate of fusimotor neurones. This concept is supported by work on the rat (Steg, 1964), rabbit (Ellaway & Pascoe, 1968) and cat (Bergmans & Grillner, 1968; Maxwell & Sumpter, 1972). These animal experiments have provided evidence that adrenoceptors are present in the spinal cord, and regulate fusimotor activity. These receptors may be facilitated by a descending noradrenergic tract (Ellaway & Pascoe, 1968; Bergmans & Grillner, 1968). Further evidence comes from the histochemical fluorescence studies of Dahlström & Fuxe (1965), in which the terminals of descending noradrenergic fibres were found in close contact with small nerve cells in the ventral horn of the cat. Although our results could be explained by the existence of a similar pathway in the spinal cord of man, they could equally well be produced by an action of these drugs at a higher level,

leading indirectly to a change in fusimotor discharge via a non-adrenergic tract. It is not possible to distinguish between these sites of action.

Apart from the observation of Matthews (1965, 1966) that the phenothiazine, chlorproethazine, reduced tendon jerks without affecting the H reflex, no other evidence is available on the effects of sympathomimetic antagonists on spinal reflexes in man. Phentolamine was found to have a stimulant effect on the tendon jerk in our experiments, but this can be explained by the marked agonist properties which are shown by this drug before $\alpha$-adrenoceptor blockade develops (Das & Parratt, 1971). The blocking drug, thymoxamine, is considered to be specific for $\alpha$-adrenoceptors (Birmingham, Akubue & Szolcsanyi, 1967) and this raises the possibility that the central receptors which this drug was influencing were $\alpha$-adrenoceptors. This conclusion was reached for receptors in the hypothalamic region of the chick (Marley & Stephenson, 1969) and of man (Besser, Butler, Ratcliffe, Rees & Young, 1970). The unsatisfactory results of our experiments with blocking drugs make a definite conclusion about the nature of these receptors premature, but it should be noted that while thymoxamine had a marked and consistent effect on jerk responses, propranolol produced no change. Morales-Aguilera & Vaughan Williams (1965) found propranolol to be inactive on the knee jerk of the rabbit and guinea-pig.

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