connected a muscle's tibial tendon to the recording-lever. The variations in added compliance that occur with some common linkages have been emphasized recently by Jewell and Wilkie[9]. They note that the total series compliance of isometrically mounted muscles has varied widely in different investigations from 10 per cent muscle length/tetanus tension to 2 per cent muscle length/tetanus tension. From Hill's[10] experiments in which springs of known compliance were added in series with an isometrically mounted muscle one can infer that $(P/P_0)_i$ should vary inversely with added series compliance.

Several investigators[11,12] have reported the related observation that the degree of apparent potentiation can be increased by deliberately adding extra compliance in series with a muscle. We simply wish to emphasize that unintentional variations in added compliance can largely determine the magnitude of potentiation and suggest that the use of muscles with similar values of $(P/P_0)_i$ will help minimize the variability of potentiation data.

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PHARMACOLOGY

Pharmacologically Active Extracts from Oak Gall

Feldberg *et al.*[1,2] have shown that guinea pigs are protected to some degree against a lethal histamine aerosol by injection of extracts of Hungarian oak gall. As well as confirming these results, we have shown that the extracts exert some protection against other agonists. However, the work reported raises the question of the mode of action of these extracts.

Dried alcoholic extracts of Hungarian oak galls (*Andricus quercus-tozae*, Bose) were obtained by the method of Feldberg and Kovacs[1] and solutions were prepared in 0·9 per cent saline for injection. Guinea pigs were randomly divided into two equal groups. One group, normally six animals, received saline intraperitoneally and the other group received 40–50 mg powdered oak gall extract in 2 ml. saline by the same route. After 1 h, the pigs were exposed to an aerosol of the agonist and the time of exposure before convulsions and/or collapse was recorded. Table 1 gives typical results obtained.

A further investigation was made using the technique of Konzett and Rössler[3] as modified by Holgate and Warner[4] to measure the pulmonary resistance in guinea pigs exposed to intravenous injections of histamine, acetylcholine or 5-hydroxytryptamine before and after intraperitoneal or intravenous injection of extract. Intraperitoneal injection of doses of extract which protected animals exposed to histamine aerosol failed to change significantly the responses to any of the foregoing bronchoconstrictors.

Table 1. Protection of Guinea Pigs against Aerosols of Various Agonists

Aerosol	Percentage concentration of agonist (w/v in water)	Mean time to convulsions and/or collapse (sec) Treated	Controls	Statistical significance
Histamine acid phosphate	0·8	450	125	$0{\cdot}01 > P > 0{\cdot}005$
Acetylcholine bromide	3·0	173	107	Not significant
5-hydroxytryptamine creatinine sulphate	0·6	266	58	$0{\cdot}01 > P > 0{\cdot}005$

The maximum tolerated dose of extract by the intravenous route was ~ 10 mg/kg. In one experiment with this dose of extract, the weight of histamine had to be increased four times to maintain its response. No evidence of antagonism to histamine was obtained with an intravenous dose of 5 mg/kg extract. Neither acetylcholine nor 5-hydroxytryptamine could be antagonized.

The results confirm Feldberg and Kovacs's findings that the extracts confer some degree of protection against a histamine aerosol[1]. Furthermore, they show that a similar protection is afforded against 5-hydroxytryptamine; the less obvious effect against acetylcholine is not statistically significant.

Standard antagonists lead to a reduction in the degree of bronchial resistance as measured by the Konzett–Rössler technique in addition to protecting against an aerosol. In contrast, while affording protection against aerosols, the oak gall extract shows only a slight effect on the degree of bronchial resistance. Although limitations are imposed by the crude nature of the extracts used, it appears possible that the protection afforded may not be achieved by blockade at the drug receptors in the bronchioles, but by some other mechanism.

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Effects of Thiols on Oxytocin and Vasopressin Receptors

It was shown by van Dyke *et al.*[1] that thioglycollate inactivates oxytocin and vasopressin presumably by reducing the S–S bond in these polypeptides. This effect has been widely used to detect the presence of posterior pituitary hormones in tissue extracts by incubating them with thioglycollate and testing whether their pharmacological activity is afterwards abolished. We have confirmed that thioglycollate inactivates oxytocin and vasopressin, but find that this is a slow process and that the lack of activity of S–S polypeptides in the presence of thioglycollate is largely due to a pharmacological antagonism at the receptor level. Another thiol, α-thioglycerol, also exhibits this antagonism, although its ability to destroy S–S polypeptides is negligible.