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SOME OBSERVATIONS ON PECKING IN PIGEONS

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An attempt was made to analyse the pecking behaviour in pigeons induced by apomorphine. The pecking was completely suppressed by tranquillizers, barbiturates and cortisone. It was intensified by histamine, nicotine, lobeline, testosterone, progesterone and sodium taurocholate. None of the drugs tested could induce in pigeons pecking typical of apomorphine. Apomorphine induced pecking in other birds too. It was concluded that the pecking phenomenon after apomorphine is similar to the natural feeding movements performed by pigeons while eating grains and possibly it is the function of a specialized area in the limbic system of the brain which is stimulated by apomorphine.

Pecking behaviour in pigeons induced by apomorphine is described by Koster (1957) and by Dhawan & Saxena (1960a). The same phenomenon was observed by one of the authors during the study of the antiemetic properties of phenothiazine derivatives. This action of apomorphine in pigeons was thought to be an excitatory one, as intravenous administration of chlorpromazine could stop the pecking with the onset of tranquillization. Recently Dhawan & Saxena (1960a & b) reported experiments performed with a heterogenous group of drugs to elucidate the mechanism of pecking in pigeons. Since this behaviour of pigeons following apomorphine injections is not yet well understood, we are reporting some additional observations in this paper.

METHODS

Pigeons of either sex weighing from 250 to 300 g were used. There was no restriction on feeding or drinking except at the time of experiment, when both the grains and water were withdrawn. For observation of pecking each bird was kept in a separate wire mesh cage, 10 in. × 6 in. × 6 in. in size.

In the first series of experiments various drugs were tested for their effect on apomorphine pecking. For this the drugs were given half an hour before the standard dose of apomorphine, and birds were then observed for 30 min. Pecking in this period for a minimum of 10 times was taken as a positive response. In the second series various drugs were injected in pigeons to find out whether they could induce pecking activity like that seen after apomorphine. Lastly, the effect of different types of physical stress on the pecking behaviour induced by apomorphine was observed in quails, sparrows, parrots, hens, rabbits, rats, guinea-pigs, mice and dogs.

All the drugs were given intraperitoneally, and each animal was used for the next experiment only after an interval of 4 days. During each type of experiment on pigeons, 6 birds were always kept as controls.

RESULTS

It was observed that 2.0 mg/kg of apomorphine induced marked pecking activity in all the pigeons. Two pigeons out of the series showed only mild pecking activity, and these were excluded from the study. Pecking with this dose of apomorphine started in 2 to 4 min and continued throughout the 30 min period of observation. It was always preceded by some degree of excitement.

Three groups of drugs, namely, the phenothiazine derivatives, barbiturates, cortisone and nicotine, completely inhibited pecking (Table 1). In the phenothiazine group of drugs, trifluprómazine was found to be the most powerful drug in suppress-

TABLE 1

ANTIPECKING EFFECT OF DRUGS ON THE APOMORPHINE-INDUCED PECKING IN PIGEONS

Apomorphine 2 mg/kg was given half an hour after the injection of test drug

No. of pigeons	Drug	Dose mg/kg	% Antipecking activity	% vomiting
5	Trifluprómazine	2·0	20	—
5		4·0	40	—
5		6·0	80	—
5		8·0	100	—
6	Chlorpromazine	6·0	—	—
6		8·0	83	—
6		10·0	100	—
6	Prochlorperazine	16·0	—	—
6		24·0	50	—
6		28·0	83	—
6		32·0	100	—
5	Promethazine	6·0	20	20
5		10·0	40	40
5		20·0	60	60
5		40·0	60	100
5		60·0	100	100
4	Pentobarbitone	8·0	25	—
4		16·0	100	—
4	Amylobarbitone	8·0	25	—
4		16·0	100	—
4	Cortisone	4·0	25	—
4		8·0	75	—
4		16·0	100	—
2	Nicotine	4·0	100	100

ing apomorphine pecking and was also the most potent tranquillizing agent. Surprisingly, promethazine was found to be a persistent emetic in pigeons. Emesis started within 5 min after the injection of promethazine and usually lasted for 15 to 25 min. Emetic activity and its severity increased with the dose, and the dose which was 100% emetic was also 100% antipecking. Table 1 shows the emetic and antipecking activity of promethazine in different doses.

The other drugs which blocked the pecking activity were pentobarbitone, amylobarbitone, cortisone and nicotine (Table 1). The first three drugs suppressed pecking completely in doses of 16 mg/kg, while nicotine was effective as an antipecking agent only in a dose of 4 mg/kg, a dose which produced vomiting in all the pigeons. In lower doses nicotine apparently intensified the apomorphine pecking in pigeons (Table 2).

Like nicotine, some of the drugs also intensified the apomorphine pecking. Although it was not possible to express this quantitatively it was repeatedly confirmed by the different observers. Drugs like nicotine, lobeline, testosterone, and sodium taurocholate made the apomorphine pecking so compulsive that the birds pecked incessantly and no intervals between the bouts of pecking were seen, as was noticed in control pigeons.

A search was made for a drug which would precipitate pecking like apomorphine. The drugs from different groups, for example, emetine, ouabain and copper sulphate (emetics), dexamphetamine, atropine methylphenidate, nicotine and lobeline (central nervous system stimulants), acetylcholine, neostigmine and physostigmine (parasympathomimetics), adrenaline (sympathomimetic) and insulin (hypoglycaemic), were

TABLE 2

DRUGS WHICH APPARENTLY INTENSIFIED THE APOMORPHINE PECKING IN PIGEONS

Apomorphine (2 mg/kg) given half an hour after the test drug. 1 = Excessive. 2 = Moderate. Potentiation of pecking was recorded as either 1 or 2 separately by three observers, and 1 was taken to be reliable when there was an agreement in the observations of all the observers

No. of pigeons	Drug	Dose mg/kg	Increase in pecking
4	Nicotine	0·2	2
5		0·3	1
4		0·4	2
4	Histamine	0·8	?
4		1·6	2
5	Testosterone	10·0	1
4		20·0	1
4	Oestradiol	10·0	2
5		20·0	2
4	Progesterone	20·0	1
4	Sodium taurocholate	40·0	1
4		80·0	2

given intraperitoneally in a wide range of doses. None of these induced pecking typical of apomorphine. However, nicotine, lobeline, methylphenidate and acetylcholine produced a behaviour pattern very akin to that seen after apomorphine. Some pigeons did show pecking activity after the injections of some of these drugs, but the pecking score never exceeded 10 in 30 min.

The relation of feeding to pecking was seen by starving the pigeons, by insulin injection and by giving glucose before and after the onset of pecking. Feeding sensations were artificially created by keeping the grains under a transparent glass floor over which the pigeons were kept standing. All these methods did not influence the pecking behaviour of pigeons.

The effect of certain other agents on pecking in pigeons was also observed. Anxiety and fear were created by keeping dogs or hens near the cages, by loud noise and by sudden exposure to bright light. All these, including the simple movements of an observer towards the cage from a distance, resulted in a prompt cessation of pecking activity. However, pecking started again after the removal of a disturbing factor.

Apomorphine induced pecking in other birds like quails, sparrows and parrots in the same dose, that is, 2 mg/kg. In the case of quails, pecking was positive only when the birds were grouped in one cage, while parrots exhibited only slight pecking activity. In hens, a dose of 3.0 mg/kg was required to induce pecking. In rats, mice, rabbits and guinea-pigs, apomorphine produced characteristic chewing movements. In dogs the emetic action was noted.

DISCUSSION

It is interesting to note that apomorphine, which consistently produces an emetic action in man, dogs, cats and some other animals, fails to do so in pigeons. This finding confirms that of Dhawan & Saxena (1960a) and Madjarek & Stern (1956). Instead of emesis, apomorphine consistently induces pecking behaviour in pigeons. The observations described give no satisfactory explanation for the phenomenon of pecking. However, it has been possible to analyse pecking behaviour to some extent.

Pecking does not appear to be related to the act of vomiting, as none of the emetic drugs (emetine, ouabain, copper sulphate, atropine, nicotine, promethazine) induced pecking in non-emetic doses or in emetic doses before, during or after vomiting. In fact, it is possible to dissociate emetic and pecking effects, from the observations with promethazine and nicotine, which, in emetic doses, show antipecking activity.

One possibility is that pecking is the expression of general stimulation of the nervous system, since pigeons always show some degree of excitement before they start pecking after the injection of apomorphine. This hypothesis is not tenable, since a number of known stimulant drugs failed to induce pecking.

The pecking phenomenon is also not due to a decreased excitability of the brain as drugs like acetazoleamide (Millichap, Woodbury & Goodman, 1955), azacyclonol (Dhawan & Saxena, 1960a), barbiturates and some phenothiazine derivatives do not induce pecking. The last two groups, on the contrary, exhibit strong antipecking activity.

The feeding centre too does not show any relation to pecking, as can be deduced from our observations and those of Dhawan & Saxena (1960a). Starvation or hypoglycaemia produced by excessive doses of insulin is ineffective. Pecking also remains unaffected even when glucose is given before or during the pecking activity induced by apomorphine.

It is, moreover, difficult to accept the view that pecking is a feeding " hallucination," as suggested by Koster (1957). Firstly, birds behave quite normally when they are interrupted during pecking, and pecking continues only when the pigeons are kept undisturbed. Secondly, grains were put under a glass floor in an attempt to simulate artificial " hallucination," but this procedure did not induce pecking in normal pigeons. In addition, various hallucinogenic agents (Dhawan & Saxena, 1960a) are ineffective in producing pecking. Pecking is not a sign of anxiety or fear complex, as the physical methods used to produce these do not make the pigeons peck.

All the observations described above could be explained on the assumption that there is a specialized part of the nervous system, like the chemoreceptor trigger zone of emesis (Wang & Borison, 1950), which is specifically stimulated by apomorphine. This area normally appears to control the voluntary movements of the birds, stimulating pecking, which they make while picking up the grains. When apomorphine stimulates this area there appears a compulsive voluntary feeding-like movement—a pecking phenomenon. Additional evidence is obtained from our observations to show that pecking is identical to feeding movements. Apomorphine is able to induce pecking in birds like quails, sparrows, parrots and hens which have the same type of feeding movements as pigeons while picking up the grains. Secondly, apomorphine produces chewing movements in rats (Du Toit & Christensen, 1948), mice, rabbits and guinea-pigs. Chewing movements of these animals are comparable to the pecking or feeding movements of pigeons.

No drug in this investigation and that of Dhawan & Saxena (1960a, b) could induce typical apomorphine pecking. Therefore, it appears that, for a drug to be an effective pecking agent, a selective action like apomorphine on the pecking zone with the least general effects on the central nervous system is necessary.

Lastly, it is suggested that, as pecking seems to be a behaviour pattern, an area controlling this activity is more likely to be situated in the limbic system of the brain.

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