impulses which spread towards the CNS leading to 'epileptic' convulsions. This postulate is in agreement with KRUSHINSKY'S[2] hypothesis which suggests that the development of the convulsive crisis is primarily dependent on low brain stem structures. The distribution of ChAc and AChE seems genetically independent.

Résumé. Le système cholinergique est étudié au niveau de la cochlée chez des souris se distinguant par leur sensibilité à l'épilepsie acoustique. Une activité accrue de l'enzyme de synthèse de l'acétylcholine a pu être mise en évidence dans la souche génétiquement sensible à la crise audiogène.

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Extinction of the Vasodilator Component of the Defence Reaction in the Cat

The somato-autonomic relationship is perhaps expressed in the most dramatic manner in emergency situations such as aggression or flight. In these situations the somato-autonomic link is obviously vital for survival, since autonomic adjustments play an important role in enabling the performance of the behavioural task. The cardiovascular system has an essential role to play in these adjustments and its ability to respond selectively and adequately to signals from the environment is particularly important in flight or aggression.

It has been established that a typical feline, threatening posture with vocalization can be elicited by electrical stimulation in well-defined areas of the brainstem and amygdala of cats, and that this behavioural reaction is accompanied by a pattern of cardiovascular adjustment whereby the cardiac output is increased, the blood flow from the skin and mesenteric vascular beds being redirected to those of skeletal muscle[1,2]. In this cardiovascular response active vasodilatation in the skeletal muscles plays an important role and it has been shown that in cats it is brought about largely by activation of the cholinergic sympathetic nerve fibres, since injections of atropine greatly reduce it[1,3]. Moreover, a very similar cardiovascular reaction including cholinergic muscle vasodilatation occurs in animals when they are startled by a sudden and new stimulus[3]. With repetition of the stimulus the response subsides. The cholinergic muscle vasodilatation appears also as a response to noxious stimulation, and this vascular reaction has been easily conditioned when an auditory stimulus preceded the noxious stimulation[3–5]. The startle reaction to novelty ('unknown stimulus') and the reaction to noxious stimuli appear to be closely related to flight or aggression[3,6,7]. Various species respond with 'alarm', flight or display of a threatening posture to definite visual or auditory stimuli. Some cats when confronted by a dog often display a threatening posture similar to that elicited by stimulation in specific areas in the brainstem or amygdala. We have chosen the 'naturally' elicited threatening reaction in cats confronted by a dog to investigate the extent and evolution of cardiovascular involvement.

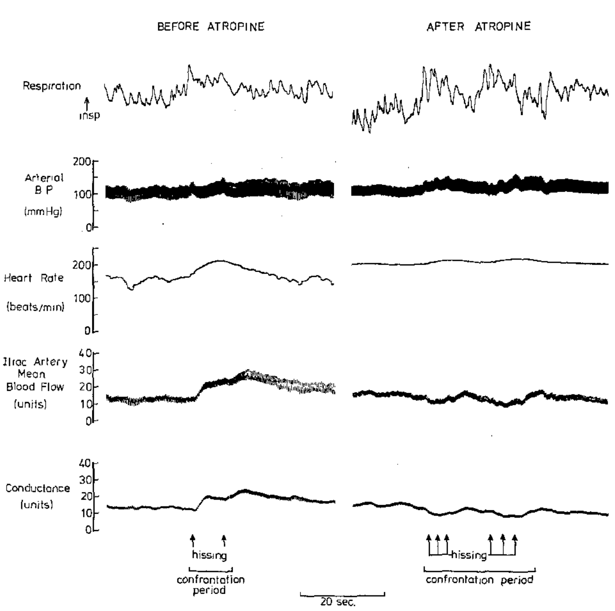

Fig. 1. Cardiovascular concomitants of the threatening response of a cat when confronted with a dog. The vasodilator component is blocked by an intra-arterial injection of atropine sulphate (0.2 mg/kg).

ADAMS et al.[8] have shown that cats responding to threatened aggression by another cat may do so without the cardiovascular changes so characteristic of the defence reaction elicited by brainstem stimulation. In particular, they found that the cholinergic muscle vasodilatation may be absent. It is possible that in these experiments the vasodilator response had already been extinguished before the measurements were made; this question has, therefore, been specifically examined.

In our experiments on cats, observations of behaviour were correlated with measurements of arterial blood pressure, external iliac blood flow, heart rate, electromyographic activity in the hindlimb, and respiratory rate. The external iliac blood flow was monitored using an MBI flow meter with an electromagnetic probe (Micron) implanted above the deep femoral branch which was ligated. The blood pressure was monitored via transparent vinyl cannula inserted into the aorta through the common carotid artery. The conductance (flow/pressure) in the external iliac artery was recorded continuously, with the use of an electronic divider (Analog 4 Quadrant Multiplier AD 426 in the feedback of an AD 401 operational amplifier). The cat was in a box facing a chicken wire or glass

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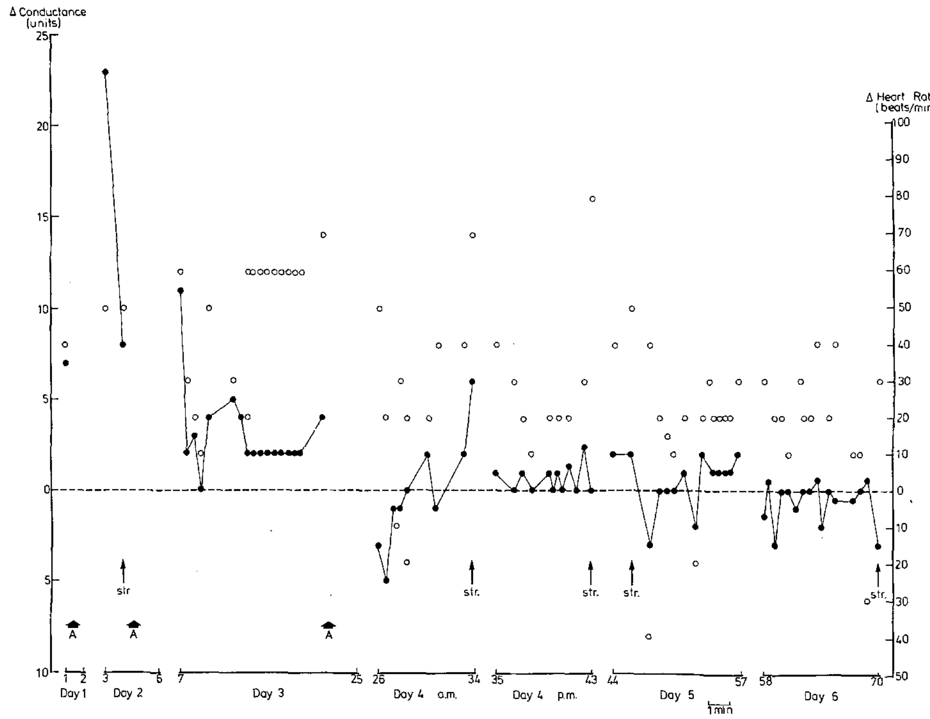

Fig. 2. Changes from resting level of conductance (flow/pressure) and heart rate during each bout of threatening posture in a single cat when confronted with a dog on 6 consecutive days. The conductance changes are represented by black dots, the heart rate changes by open circles, str. indicates striking with a paw, A = injection of atropine (the data after atropine injections are not included), and the figures at the bottom indicate the sequential number of each bout of rage.

partition through which a dog could be seen when it was brought into the room. Three cats have been involved in these experiments and the observations on all of them have been consistent. The cats displayed a threatening posture almost as soon as they sighted a dog: in most cases they remained seated, the pupils dilated and the ears flattened; there was piloerection, the lips were retracted, and the animal growled and hissed and, sometimes, with claws unsheathed, the cat would strike with one forepaw in the direction of the dog. The main features of the cardiovascular response during these bouts of rage were an increase in conductance to flow in the external iliac artery and a cardiac acceleration. There was also a slight increase in the mean arterial pressure and a rise in the pulse pressure. The increase in conductance in the external iliac artery was abolished by atropine indicating that it was probably due to cholinergic sympathetic vasodilatation in the hindlimb (Figure 1). The atropine test was repeated several times in all three cats and on each occasion the increase in conductance was abolished. However, with repetition of the confrontation experiments, day after day, the cardiovascular changes diminished. The conductance increase became smaller although the cat still displayed its full threatening posture with vocalisation. In Figure 2 the changes are shown in external iliac artery conductance (black circles) and heart rate (open circles) during each bout of rage, on confrontation with a dog, in 1 cat. On the 3rd day the conductance increase had already diminished and in one 'rage' reaction there was no change of conductance at all. On the 4th day the picture was changed: vasoconstriction appeared instead of vasodilation in the hindlimb, and bradycardia occurred for the first time. After 6 days of repeated confrontations, during which the cat did not experience any noxious stimulation, the vasodilation in the hindlimb was extinguished in all 3 animals and was frequently replaced by vasoconstriction. The cardiac acceleration was also diminished and instead, on some occasions, bradycardia was observed. The threatening posture, with piloerection, pupillary dilation, vocalization and striking with the paw, continued with undiminished intensity throughout the whole series of confrontations.

The course of extinction of the vasodilator response, as shown in Figure 2, resembled that of chronic extinction of any conditioned response. In the confrontation experiments we presented the cats with the conditioned stimulus – the sight of a dog – but since no noxious stimulation followed, the cat's defence reaction began to be extinguished. It was the muscle vasodilatation, hitherto held to be a characteristic component of the defence reaction, which was the easiest to extinguish. We have also observed such extinction in experiments on dogs[5]. The behavioural component of the conditioned reaction (e.g. threatening posture) was the last to disappear.

Résumé. Le chat en face d'un chien a une réaction charactéristique posturale de menace accompagnée d'une réaction cardiovasculaire, caractéristique avec une vasodilatation dans les muscles squelettiques qui est sensible à l'atropine. La répétition des confrontations décrites produit une extinction des composantes cardiovasculaires, surtout de vasodilatation.

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