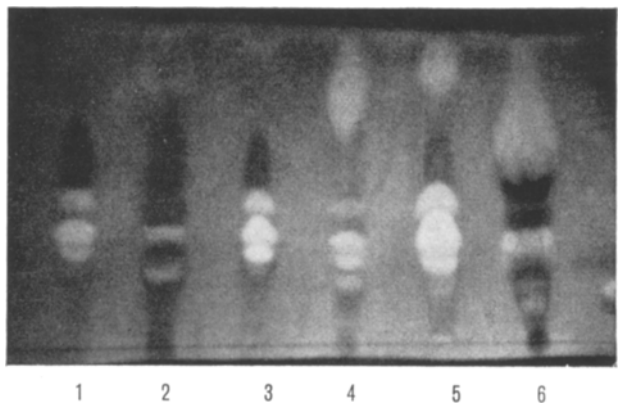
chromatography or by thin layer chromatography and identified, using a standard as reference. Lipids were stained with Rodamine 6G, and photographed under UV-light. Furthermore, lecithin and lysolecithin were identified with the test for choline[11]. Reagents used were products of Carlo Erba RS for chromatography; standards for lipids were supplied by Sigma.

Results. In the experiments reported it is shown that, during aseptic autolysis, phospholipids are destroyed in the same way (qualitatively) in heart and liver, while kidney seems to behave differently. The partial disappearance of lecithins and phosphatidilethanolamine and cardiolipin (diphosphatidilglyceride) and the contemporary appearance of lysolecithin and other lyso compounds can be seen: this appearance is much better demonstrated by the specific test not reported here for the amino groups and for choline. The controls, made by incubating phospholipid from normal tissue and phospholipid standard in the same condition of tissue in autolysis, show a partial destruction of cardiolipin and phosphatidilethanolamine (but not as clearly as in tissues in autolysis), and do not show destruction of lecithin, as we have, obviously, with lecithinase in the same way as during autolysis. From non-published data we found that there is a remarkable liberation of fatty acids and the quantity of these recovered is stoichiometrically superior to disappeared lecithin, and may partly come from hydrolysis of phosphatidilethanolamine and cardiolipin, or partly from hydrolysis of triglycerides of tissues. In the liver, the amounts of lysolecithin formed is equal to the lecithin that disappears, and the activity is higher than in kidney and heart.



Paper chromatograms according to MARINETTI to illustrate the differences in phospholipid composition of normal and autolytic rabbit tissues. The chromatogram was stained with Rodamine 6 G and photographed under UV-light. Spots 1 and 2 belong to heart phospholipid, 3 and 4 to kidney, and 5 and 6 to liver. Spots 1, 3, and 5 are phospholipid of normal tissue: the more polar component, the dark, spread-out spot at the top is cardiolipin; below cardiolipin the two light spots are from phosphatidilethanolamine and lecithin; the lower spots are in the region of sphingomyelin and phosphatidilinositol, but for these components there is much disagreement in the literature. Spots 2, 4, and 6, from the bottom upwards, are lysolecithin, lysophosphatidilethanolamine (in the area of phosphatidilinositol and sphingomyelin), lecithin, a non-identified spot, and cardiolipin. On the solvent front are migrated neutral lipid.

We can therefore say that during autolysis there is a phospholipase A activity in all three organs compared with the endogenous lecithin; we cannot yet say, however, that there is a hydrolytic activity against phosphatidilethanolamine and cardiolipin, because of their breakdown, as already mentioned, in the medium used for autolysis.

This is only a preliminary note, but we can anticipate that aseptic autolysis could be a good method for studying the pathways of endogenous lipid catabolism[12].

Riassunto. Gli autori hanno studiato il comportamento dei fosfolipidi durante l'autolisi aseptica di cuore, rene e fegato di coniglio. Dai risultati ottenuti concludono che si ha attività fosfolipasica in tutti e tre gli organi considerati verso le lecitine endogene del tessuto, attività difficilmente evidenziabile in vivo con altre tecniche.

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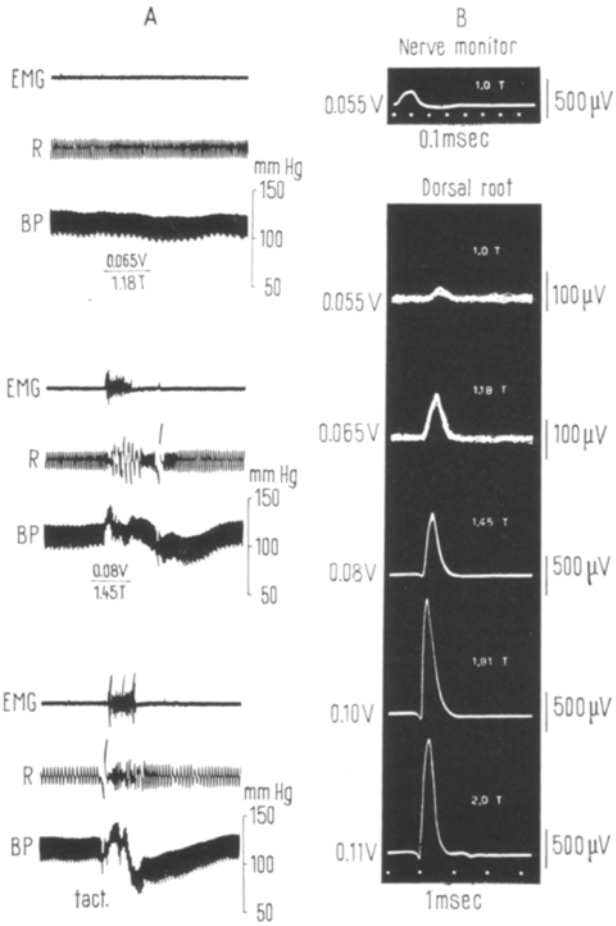
Excitation of Sham Rage Behaviour by Controlled Electrical Stimulation of Group I Muscle Afferents[1]

It is generally assumed that group I muscle afferents exclusively project onto segmental levels of the spinal cord and the cerebellum[2], with the possible exception of group I afferents from the forelimb which seem to project to the motor cortex as well[3]. In support of this opinion, high rate stimulation of group I fibres of the hamstring nerve could not induce electrocorticographic or behavioural arousal of intact unrestrained cats, nor could it discharge reticular units[4]. Since it is known that different effects may be obtained by working on preparations with different excitability backgrounds, we have tested muscle afferent stimulation in the acute decorticate animal.

Results. In 20 acute decorticate cats, 0.1 msec, 100–600 c/sec rectangular pulses were delivered to the central stump of a cut hamstring nerve through a bipolar collar-type electrode carrier, the proximal electrode being always the cathode. The stimulus threshold for the most excitable group I fibres was periodically checked by CRO monitoring from the sciatic trunk above the entrance of the hamstring nerve, and found to remain constant throughout the whole experiment. Stimulating voltages were always indicated as multiples of this threshold voltage (T meaning times threshold).

In 12 out of 20 decorticate animals, sham rage outbursts could be consistently elicited by stimulating the hamstring nerve at intensities lower than 1.70 T. In 10 animals intensities below 1.50 T, and in 2 animals as low as 1.36 T were effective. In 5 decorticate animals in which at the end of the experiment the dorsal root electrogram was recorded, as well as in 5 intact animals under barbiturate anaesthesia in which this control was performed, group II threshold ranged between 1.70 and 2.12 T, confirming previous observations by other authors[4,5]. The Figure summarizes an experiment in which the muscle afferents evoking sham rage were clearly identified by dorsal root recording. It was shown in this animal that stimulation of the left hamstring nerve at 300 c/sec at a strength of 0.08 V (i.e. 1.45 times the threshold activation of the nerve, 0.055 V: B, upper tracing) constantly elicited a violent outburst of sham rage (A, middle portion), identical to that induced by tactile stimuli (A, lower portion). The animal was then anaesthetized with pentobarbital and immobilized with gallamine. By leading from dorsal root L7 (B, lower tracings), it was shown that group I threshold was still 0.055 V and that stimuli of 0.08 V (1.45 T) produced a pure activation of group I fibres, the most excitable group II fibres being activated only at a much higher voltage (2.0 T).



Sham rage outbursts upon 300 c/sec stimulation of group I afferents in the left hamstring nerve. In A, the polygraph recording of sham rage behaviour: EMG = electromyogram of the left triceps muscle; R = respiration; BP = femoral arterial pressure. In B, the nerve monitor represents 15 superimposed sweeps of electrical response to threshold activation of the hamstring nerve, led from the sciatic nerve at a short distance from the stimulating cathode. Large deflection on the left is shock artefact, followed by a barely detectable neural potential. In B, tracings indicated under dorsal root are monophasic records (15 superimposed sweeps) from cut dorsal root L7, upon stimulation of the left hamstring nerve at the amplitudes indicated. Stimulation recording distance is 11.2 cm. Note that the time base is different for nerve monitor and dorsal root tracings.

To rule out the possibility that rage mechanisms were not directly excited by group I volleys, but rather by impulses in skin or high threshold muscle afferents generated by a reflex muscle contraction, a few decorticate animals were immobilized with gallamine soon after the threshold voltage for a group I volley eliciting sham rage was found: the same group I volleys that were previously capable of eliciting rage outbursts could still induce a marked mydriasis and a definite increase in blood pressure. Both phenomena disappeared after decerebration. Finally, the hypothesis that group I impulses might evoke sham rage by excitation of the cerebellar mechanisms regulating rage behaviour[6], has been disproved by the possibility of inducing rage outbursts by stimuli limited to group I afferents after acute ablation of the whole cerebellum.

Upward conduction of group I afferent impulses to higher brain stem levels, though unexpected on the evidence of previous authors, is likely to have been shown in our experiments because of the high excitability of the decorticate cat. Alternatively, descending influences from cerebral cortex and subcortical structures might depress transmission of group I volleys to the spino-reticular, or other ascending pathways in the intact and decerebrate animal, and emphasize it in the decorticate state.

Riassunto. La stimolazione elettrica ad alta cadenza (100–600 c/sec) del puro contingente di fibre muscolari appartenenti al gruppo I è in grado di scatenare accessi di falsa rabbia nel gatto decorticato, anche dopo ablazione del cervelletto e somministrazione di curarizzanti.

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