Gallic acid exhibited fungistatic action against Alternaria humicola, A. solani, Cephalosporium sacchari, Curvularia lunata, C. pallescens, Cladosporium herbarum, Chaetomium indicum, Fusarium nivale, F. oxysporum, Helminthosporium oryzae, H. sativum, Memnoniella echinata, Nigrospora sphaerica, Paecilomyces fusisporus, Pythium aphanidermatum and Rhizopus nigricans at 3% concentration while against Aspergillus variecolor, A. terreus, A. nidulans and Leptospherulina trifolii at 3.5% concentration. However, Aspergillus niger, A. flavus, Chaetosphaeronema herbarum, Penicillium funiculosum and P. oxalicum remained unaffected (table).

The role of Phenols as fungitoxic agents is well established. Phenolic acids, viz., benzoic acid, salicylic acid and protocatechuic acid, are well known antifungal substances[5]. Benzoic acid and salicylic acids have also been recorded as antifungal factor of Populus tremuloides[6]. However, the isolation of gallic acid as an antifungal factor from Rosa chinensis in present study has been done for the first time.

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Differential effects of disuse preceding denervation on the onset and development of fibrillation in fast and slow muscles[1]

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Summary. Section of the sciatic nerve, performed after a week of muscular disuse, is followed by fibrillation earlier in the soleus (S) than in the anterior tibialis (AT) muscle of the rat. The subsequent development of fibrillation, which is different in the control denervated S as compared with the control denervated AT, tends to become similar in the disused-denervated muscles.

In a previous paper[2], it was reported that the onset of fibrillation in the denervated soleus-gastrocnemius muscles of the rat is greatly accelerated if the muscles are put into disuse for some days before denervation.

In the present work, it was investigated if disuse affects equally the fast and slow muscle fibres (both of which are present in the tested muscular group[3,4]), or preferentially affects one type of fibres. Under the same experimental conditions of disuse and denervation, the onset of fibrillation was selectively investigated in soleus (S, slow) and anterior tibialis (AT, fast) muscles. The subsequent development of fibrillation, both in control and experimental muscles, was also investigated.

Methods. Spinal cord section, or plaster cast immobilization of the limbs were performed in adult albino rats, 250–300 g in weight, as described[2]. The distal tendinous insertions of S and AT were cut on one side. In a number of cases, the whole tendo calcaneus was cut. Unilateral (cordotomized, immobilized, or control animals) or bilateral (tenotomized animals) section of the sciatic nerve was performed 6–7 days later, near the trochanter, at 3.5–4.0 cm from the point of nerve insertion into the muscles, the nerve stump to AT being 2–3 mm longer than the stump to S.

EMGraphic records were taken, under ether anaesthesia, via a pair of needle electrodes, insulated except for the tips, with an interelectrode distance of 2 mm, from the middle portions of both S and AT. Repeated insertions were performed transcutaneously in the same animal, using the fibula as a reference point for S. The records were from the superficial layers of AT, where succinic dehydrogenase activity is low[5], at a depth not greater than 2 mm, and approximately from the central layers of S. In some cases, fibrillation was acutely recorded from the exposed S, at a depth of 1–2 mm, the animal being sacrificed afterwards.

The development of fibrillation was estimated by measuring the integrated electrical activity of the muscles through a Beckman-Offner EMG integrator, at 12–24-h-intervals, over a period of a week. Fibrillation activity was also monitored on a CRO.

Results. In the previously tenotomized S, the onset of fibrillation was, on an average, as precocious as reported for the soleus-gastrocnemius group[2], occurring 25.63 ± 1.14 h after denervation (mean of 23 cases, ± S.E.); the control time in the contralateral denervated muscles was 54.31 ± 1.09 h. In the tenotomized AT, fibrillation arose consistently later: 39.86 ± 2.22 h after denervation (mean of 21 cases; control time 55.87 ± 2.13 h).

The difference between S and AT was still greater in immobilized limbs. Fibrillation began respectively 26.26 ± 2.29 h and 48.52 ± 4.32 h after denervation (mean of 10 cases).

Less markedly different were the results in the cordotomized animals. Fibrillation began 22.41 ± 1.18 h and 30.42 ± 3.52 h after denervation, respectively in S and AT (mean of 15 cases).

The development of fibrillation was first of all investigated in the simply denervated, control muscles. In S (see figure) fibrillation increased rather quickly, reaching a peak 48 h after its onset, but afterwards it fell off markedly. In AT, fibrillation developed more gradually during the first 2 days, and then leveled off, so that after 168 h the electrical activity was much about the same in AT and S.

In the animals with spinal cord transection, fibrillation development in S and AT was rather similar, increasing very slowly and very gradually throughout the whole experimental period (168 h).

In the tenotomized and in the immobilized animals, the results were less consistent. In some animals, the devel-

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opment of fibrillation modified as after cord section, whereas in other animals there were no significant differences with the control muscles.

Discussion. The importance of motor activity, possibly associated to a 'trophic' neural factor[6–9], in the control of muscle membrane properties has been stressed by many authors in recent years[10–13]. It was suggested also by our experiments on the soleus-gastrocnemius muscles[2], showing that disuse strongly favours the onset of fibrillation following denervation.

From the present work, it results that experimental disuse is in general less effective in fast muscle than in the slow one, in accelerating the onset of fibrillation. This is in keeping with the observations that disuse affects fast muscle to a lesser degree than the slow one, as far as trophism[14,15], histochemical characteristics[16,17] and dynamic properties[17–20] are concerned. It seems possible to relate such results mainly to the actual reduction in muscle use caused by the experimental procedures rather than to a different importance of muscle activity in maintaining muscle properties. Tenotomy in the rabbit[21], and limb fixation in the rat[22], have been shown in fact to have little, if any, effect on EMGraphic activity of fast muscles, while strongly reducing that of the slow ones; on the other hand, fast muscle is physiologically much less used than slow muscle[22,23]. So it seems reasonable to assume that in our experiments too, the reduction in motor activity after tenotomy, limb casting or cordotomy was relatively less marked in AT than in S, with a correspondingly less marked effect on fibrillation activity. A higher degree of disuse caused in the muscles could similarly be the reason why cordotomy was more effective than tenotomy or casting in favouring fibrillation also in AT: notwithstanding the resulting dramatic changes in physiological conditions, cordotomy is probably[7,24] a better way of eliminating muscle use than tenotomy, which

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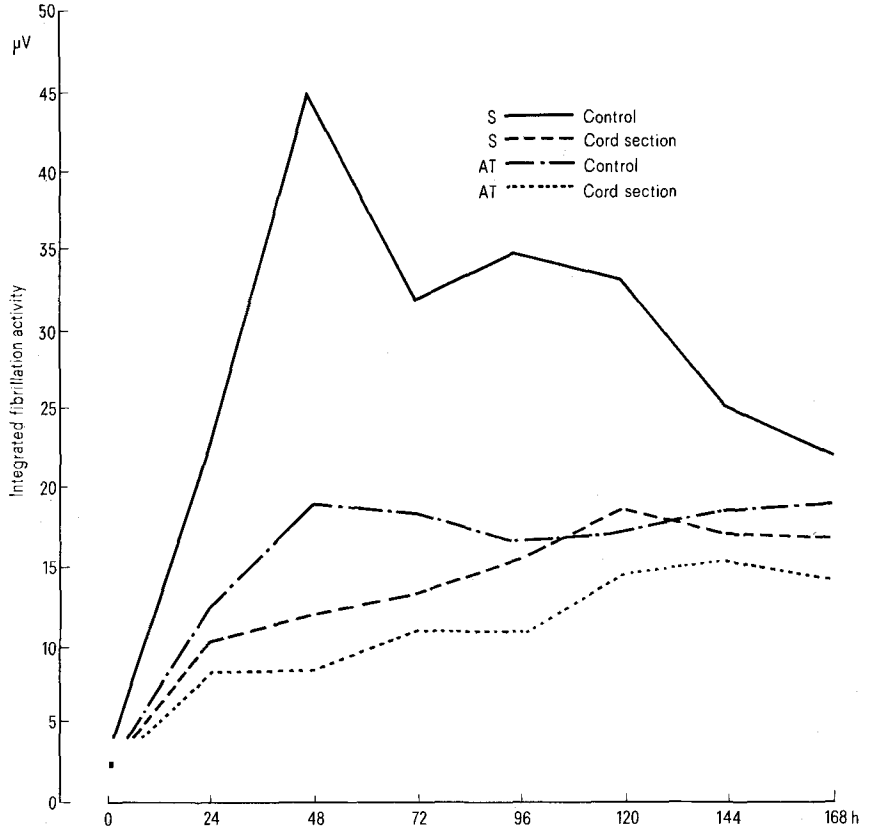

Development of fibrillation in S and AT, both in the control and cordotomized animals. Zero time is at the onset of fibrillation. The numerical data below the graph are the mean values (± S. E.) of the integrated electrical activity in the different experimental conditions, at the times indicated on the abscissa. Numbers in parentheses refer to the number of experiments.

———	2.3 ± 0.5 (11)	22.5 ± 1.9 (32)	44.6 ± 4.6 (28)	31.5 ± 2.6 (30)	34.9 ± 2.8 (13)	32.9 ± 2.6 (20)	24.9 ± 3.3 (11)	21.8 ± 3.2 (10)
—·—·	2.6 ± 0.9 (11)	12.5 ± 2.5 (32)	19.1 ± 4.7 (28)	18.4 ± 3.2 (30)	16.7 ± 3.2 (13)	17.2 ± 4.3 (20)	18.5 ± 4.8 (11)	18.7 ± 3.6 (10)
- - - -	2.1 ± 0.4 (14)	10.3 ± 1.1 (29)	12.1 ± 1.6 (17)	13.3 ± 2.6 (18)	15.5 ± 2.4 (12)	18.7 ± 2.7 (16)	17.0 ± 2.7 (13)	16.4 ± 2.3 (7)
.....	2.2 ± 0.4 (14)	8.4 ± 1.2 (29)	8.7 ± 1.9 (17)	10.9 ± 2.2 (18)	10.8 ± 2.1 (12)	14.2 ± 1.5 (16)	15.1 ± 1.7 (13)	13.9 ± 1.9 (7)

is a very complex condition[7,14,25] and casting, which does not ensure a true immobilization[7,26,27].

The observations on fibrillation development in the control muscles are reminiscent of those of Salafsky et al. on the same muscles[5]. Spinal cord section, and in some cases tenotomy or limb immobilization, caused fibrillation in S to develop more slowly and gradually than in the control muscles, thus approaching the pattern observed in the faster AT. According to many authors[17-20,22], prolonged (2–4 weeks) muscular disuse causes the slow muscle to become faster. In a few collateral experiments, we could not appreciate in vitro any change in contractile properties of S after a week of disuse (unpublished observations), but it might be suggested that the 'fast' pattern of fibrillation development represents a precocious, though indirect, evidence of the change in muscle properties. The effects of disuse on the slow components of AT[5,28] could similarly explain the slight change in fibrillation development which was observed also in this muscle.

According to the theory that a neurotrophic factor is cooperating in the control of muscle membrane properties, it would be possible also to speculate that in the disuse pretreated muscle, fibrillation, due to its earlier onset, develops when the peripheral nerve stump is still releasing the trophic substance[29], although at a progressively reduced rate[30], with a resulting slower change in membrane properties. However, this explanation is made doubtful by the fact that tenotomy and casting were practically as effective as cordotomy in accelerating the onset of fibrillation, while they did not consistently affect the subsequent development.

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Thiamine transport by human intestine in vitro[1]

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Summary. Surgical specimens of human gastrointestinal mucosa and muscle were incubated in vitro with thiamine-thiazole-2-^{14}C. Labelled thiamine uptake was uphill in mucosal tissues and downhill in muscle. Small intestinal mucosa accumulated labelled thiamine in a phosphorylated form, while gastric and colonic mucosa, as well as muscular layers of all gastrointestinal segments studied, did not.

Small intestine of several animal species is able to absorb in vivo[2-8] or to transport in vitro[9-15] small amounts of thiamine by an active mechanism[16]. In man, intestinal absorption of thiamine was studied exclusively in vivo[17-20]. The results, recently confirmed by Thomson[21,22] and Levy[23], suggest that, in human intestine, thiamine absorption involves a process saturable by low concentration of vitamin. However, a direct demonstration of an active transport mechanism could be better achieved by using an in vitro technique. This prompted us to choose, in the present research, an in vitro tissue uptake procedure for studying thiamine transport by human gastrointestinal tissue incubated with labelled thiamine. The aim of this investigation was to study: the ability of human intestinal tissue to accumulate labelled thiamine; the contribution to accumulation both of mucosal and muscular layers of gastrointestinal wall; the topographic distribution of the accumulation system along the alimentary canal, particularly the small intestine; the chemical form of thiamine which is accumulated and the functional meaning of accumulation as related to different intestinal segments.

Materials and methods. Specimens of sound gastric and intestinal (duodenum, jejunum, ileum and transverse colon) tissue were obtained from patients at the time of surgical resection for gastric and intestinal ulcer and tumors. Upon removal, the tissues were placed in cold (3–5 °C) Krebs-Henseleit (K-H) bicarbonate buffer, pH 7.4, equilibrated with 5% CO_2 in O_2[24]. Mucosal and muscular layers, thoroughly separated by dissection, were cut into pieces of approximately 200–300 mg and distributed to incubation flasks containing gassed K-H bicarbonate buffer. The time interval from surgical removal of tissues to the start of incubation never exceeded 30 min. Histological tests, performed by light microscope before and after incubation, showed that all tissues were

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