total left ventricle. This may be carried out by calculating the weighed mean

$$IS_{\%} = \frac{\sum (IS_i \cdot W_i)}{\sum W_i} \quad (2)$$

W_i = weight of piece i.

In the table, infarct sizes calculated by the aid of equations (1) and (2) are compared to those determined morphometrically. The figure shows that there is a good correlation between both methods. The coefficient of correlation is 0.94 with a high level of significance ($p > 99.9\%$).

In literature various radioactive methods have been described for detection of ischemic areas and determination of IS[8-10], but until recently[11] no serious effort was made to correlate those with IS determined morphometrically. The SD of the ^{86}Rb values from the regression line (figure) was found to be 6.5%; that is far better than the SD which one may expect by calculating IS from serial serum creatine phosphokinase (CPK) determinations after infarction (20–30%)[12]. If we only take into account the SD of 'normal' myocardial CPK activity, which is important in case of determining IS from myocardial depletion of CPK, a SD of about 16 or 14% may be expected[12,13]. The SD of 6.5% in this work is the consequence of using an internal standard (right ventricle). The limits of sensitivity of our method are determined by methodical errors (5%), which are composed of the errors of weight and cpm determination (including that of cpm correction). If the determination of IS is carried out later than 48 h after infarction, an erroneous result may be obtained, because connective tissue cells begin to grow into the area of infarction. These cells would take up ^{86}Rb, and thereby the calculated IS would be reduced. A systematic error of this method is caused by neglecting the amount of ^{86}Rb which is taken up from infarcted tissue. This fact finds its expression in reducing the coefficient of regression below unity (figure).

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Motor nerve terminal defect following tenotomy[1]

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Summary. Post-tetanic potentiation and the underlying post-tetanic repetition in cat soleus muscle require normal motor nerve terminals. These indices of nerve terminal viability are depressed 10 days and absent 15 days after tenotomy of the soleus muscle.

A muscle detached from its insertion by section of the tendon (tenotomy) is unable to develop significant tension and exhibits signs of chronic disuse. Tenotomy results in alterations in the muscle with concomitant changes in contractile properties[3], its innervation and associated spinal reflexes. The muscle atrophies, discharge from muscle spindles[4-8] and other receptors[9,10] is enhanced and monosynaptic ventral root reflexes and early potentials in the dorsal spinocerebellar tract are augmented[3,11-15].

The relative inactivity of muscles resulting from pinning of the limb causes slight alterations in presynaptic function at the neuromuscular junction[16,17]. However, nerve terminal function following marked muscle inactivity such as that resulting from tenotomy has not been determined using tests which can specifically reveal alterations in motor nerve endings[18-20].

High frequency tetanization of cat soleus nerves conditions the nerve terminals such that subsequent stimuli during the post-tetanic period elicit a burst of repetitive action potentials (post-tetanic repetition, PTR) in the vicinity of the nerve endings[21]. The PTR, which can be recorded in ventral root filaments, causes brief, asynchronous tetanization of the muscle, resulting in potentiation of muscle contractile strength (post-tetanic potentiation, PTP)[22]. PTP is dependent on the frequency of tetanic stimulation; optimum PTP occurs in cat soleus muscle in response to tetanization at 400 Hz. PTP and its underlying PTR are lost prior to transmission failure at the neuromuscular junction when the nerve terminals are subjected to physical[18] or chemical trauma[19,20]. Hence these events serve as sensitive indicators of nerve terminal viability.

Methods. 6 adult cats under pentobarbital anesthesia were tenotomized by disconnecting the Achilles tendon from the calcaneus. The tendon was folded back on itself to prevent reunion by connective tissue. 3 cats were investigated at each of 10 and 15 days following tenotomy. 5 normal cats provided control data. On test days cats were given α-chloralose (80 mg/kg i.v.), an in situ soleus nerve-muscle preparation prepared and the spinal cord exposed by dorsal laminectomy[23]. The soleus tendons were attached to a strain gauge for isometric recording of contractile tension at one-half maximum physiological extension (determined in situ prior to cutting the calcaneus). The soleus nerve was stimulated with supramaximal pulses (0.1 msec duration) at 0.4 Hz except for 10-sec tetanic trains at 25–400 Hz. Ventral roots were subdivided to obtain filaments containing func-

tionally single soleus α-motor axons. Action potentials were recorded by conventional techniques.

Results. In normal cats, 10-sec trains of tetanic stimuli evoked PTP which was proportional to the tetanic frequency (figure 1). Maximum PTP was elicited by tetanization at 400 Hz. PTP was significantly less than controls at all tetanic frequencies 10 days after tenotomy and totally absent 15 days after section of the Achilles tendon.

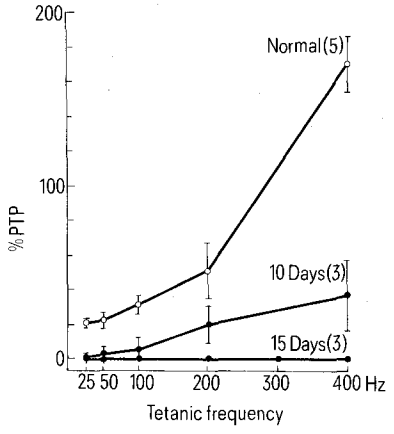

Fig. 1. Post-tetanic potentiation (PTP) of contractile tension as percent increase over pre-tetanic contractile strength in normal cat soleus muscle and 10 and 15 days after tenotomy. Numbers in brackets indicate the number of animals investigated.

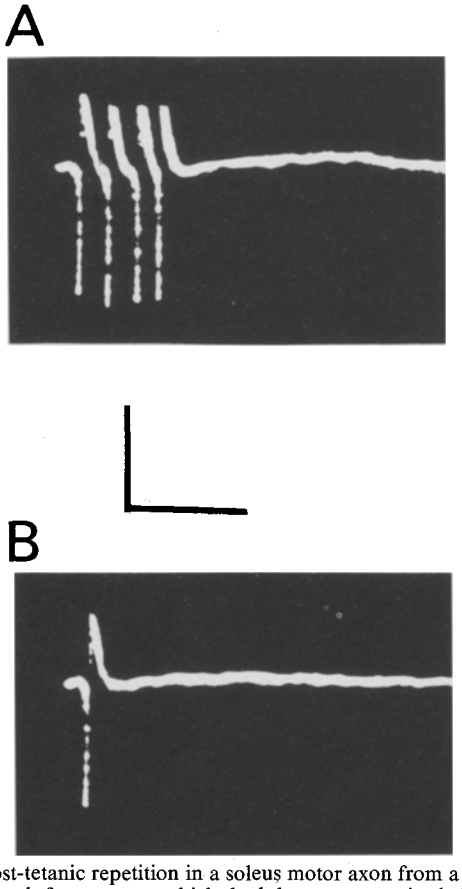

Fig. 2. Post-tetanic repetition in a soleus motor axon from a normal (A) cat and from a cat which had been tenotomized 15 days previously (B). Scale: 10 msec and 0.1 V.

Post-tetanic repetition (PTR) in the normal cat consisted of an antidromically conducted action potential elicited by the stimulus, followed by 2–5 repetitive action potentials (figure 2, A). No PTR was observed in soleus nerves of any of the tenotomized animals, but the action potential evoked by peripheral stimulation remained (figure 2, B).

No deficit was observed in the ability of single α-motor axons to faithfully conduct impulses at frequencies up to 400 Hz. The muscle contractile tension developed in response to indirect single and tetanic stimulation averaged about 30% of normal both 10 and 15 days after tenotomy.

Discussion. These data indicate the presence of a functional defect in motor nerve terminals following tenotomy. The defect is similar to that observed in the early stages of nerve terminal degeneration resulting from physical or chemical trauma[18-20]. In chemically-induced neuropathies[19,20] nerve terminal dysfunction results from a toxic action of the agent on the axon, with subsequent degenerative changes. While this does not apply in the case of terminal defect after tenotomy, other possible mechanisms are suggested.

Muscle atrophy caused by tenotomy results in shrinkage of muscle fibres. This could alter the critical geometry of the neuromuscular junction and be reflected in function. However, atrophy does not have a marked effect on other parameters of neuromuscular function[16,17]. Further, PTR and PTP are unaffected in cats with experimental vincristine neuropathy, in which considerable atrophy frequently occurs[24].

The enhanced receptor discharge reported to occur in tenotomized muscles[4-10] may be causally related to the nerve ending defect. It has been suggested that this increased sensory discharge may actually hasten muscle atrophy[15]. Perhaps a trophic influence (factor or activity) is continually provided to motor nerve perikarya by afferent fibres. Alteration of this influence by tenotomy would then have a deleterious effect on maintenance of motor nerve viability which is signalled by a nerve terminal alteration.

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