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Achilles Tendon Rupture

Aetiology and Pathogenesis of Subcutaneous Rupture Assessed on the Basis of the Literature and Rupture Experiments on Rats

by

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Previous Papers on the Same Subject:

I Barfred, T.: Histology of the rat Achilles tendon before and after tendon rupture. Acta path.microbiol.scand. 1971 a. A-79: 287–292.

II Barfred, T.: Kinesiological comments on subcutaneous ruptures of the Achilles tendon. Acta orthop.scand. 1971 b, 42: 397–405.

III Barfred, T.: Experimental rupture of the Achilles tendon. Comparison of experimental ruptures in rats of different ages and living under different conditions. Acta orthop.scand. 1971 c, 42: 406–428.

IV Barfred, T.: Experimental rupture of the Achilles tendon. Comparison of various types of experimental rupture in rats. Acta orthop.scand. 1971 d, 42: 528–543.

V Barfred, T. & H. Langgård: Water and electrolyte content in muscles and tendons of wild and domesticated rats at different body weights. Arch. phys. Med. 1973, 54: 348–353.

PREFACE

My interest in Achilles tendon rupture was aroused by Rasmus Movin, M.D., Frederiksberg Hospital.

The experimental work was carried out in the Laboratory for Theory of Gymnastics, University of Copenhagen, where I enjoyed the very best facilities in an extremely stimulating environment. I am deeply grateful to Professor Erling Asmussen, Ph. D., for affording me this possibility. Professor Asmussen as well as his staff have been a great help, in practical as well as theoretical problems. In particular, I want to thank Mr. Christian Persson, chief mechanic, for his skillful work in reconstructing the testing machine placed at my disposal by Mr. P. Gert Sørensen, Chief Mechanical Division, National Institute for Testing Materials.

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CHAPTER 1

INTRODUCTION

Age and state of training have been emphasized by many authors as important features in the aetiology of subcutaneous rupture of the Achilles tendon. The rising incidence of such ruptures during the past decades has been ascribed to increasing demands on athletic achievements and more widespread athletic activity. It is not known whether training induces factors – normal or pathological – which make tendon rupture possible, or whether athletes simply place themselves in dangerous situations which would not have happened to others. Only a few animal experiments have been performed with a view to approaching these problems, i.e. paying regard to the animal's age and state of training at the beginning of the experiment. In previous experiments, moreover, little regard has been paid to the special circumstances which frequently are present at the rupture episode.

The object of the present study was to attack the matter on the basis of animal experiments, with due regard to these very factors. Such animal experiments may elucidate important aspects of the problem, but it is so many-sided that an overall description required a broad review of the relevant literature. Other muscle and tendon ruptures will be mentioned only sporadically, although they do indeed pose the same problems as rupture of the Achilles tendon.

The primary question was whether the healthy tendon may rupture when exposed to indirect trauma. The next endeavour was to demonstrate under which circumstances the tendon – degenerated or not – is most exposed to rupture.

Describing the aetiology and pathogenesis of Achilles tendon rupture is not merely of academic interest, but a presupposition for possibly arriving at prophylactic measures and may perhaps influence the treatment. Finally, it is perhaps possible to create a better basis for the attitude of the insurance companies as to whether ruptures of the Achilles tendon are to be interpreted as accidents or as a consequence of disease.

CHAPTER 2

SUBCUTANEOUS RUPTURE OF THE HUMAN ACHILLES TENDON

Definition

Subcutaneous Achilles tendon rupture (A.R.) is defined as a rupture of the tendon substance arising without any preceding discontinuity in the skin or subcutaneous tissue. The rupture may be partial or total. According to the mode of origin a distinction is made between direct and indirect A.R.

Incidence

Christensen (1954) found 57 A.R. among 70,000 patients treated in an orthopaedic department during the period 1936 – 1954. Schönbauer (1960) found 151 cases of A.R. among 97,000 patients admitted to the Unfallkrankenhaus, Vienna, from 1925 to 1959. The reported incidence has been increasing during the past decades (Mayr 1957, Schönbauer 1960, Viernstein 1963, Frings 1969, Freilinger et al. 1970, Philadelphy et al. 1971). An absolute figure for the incidence or its increase cannot be stated, as the above authors have not mentioned the size of the population served by the departments concerned.

Age

The mean in the major series has been stated to range from 34 to 44 years, the earliest mean age being reported by Frings (1969) whose material of 317 patients was from Krankenhaus für Sportverletze and thus must be considered a selected one. Most A.R.s occur between 30 and 50 years of age (Fig. 1), but in Frings' material many patients were between 20 and 30. Pillet & Albaret (1972) found the age distribution curve to have two peaks, at 30–35 and at 45–50 years. The youngest patient on record was 13 years (Benassy 1969). Only a few of the patients have been older than 60.

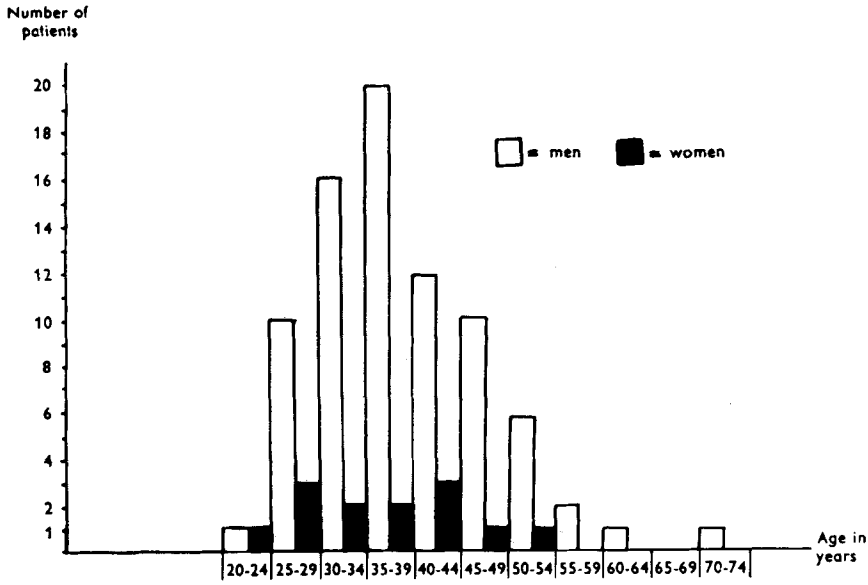

Fig. 1. Age distribution and sex ratio of 92 patients with subcutaneous rupture of the Achilles tendon (from Arner & Lindholm 1959 a).

Sex

In all materials there has been a marked male preponderance. In a collected analysis by Riede (1966 a) 648 were males and 170 females. The mean age was 41.9 for the males and 32.9 years for the females.

Training

Out of the 839 cases which comprised Riede's collected material, 606 had sustained their A.R. during athletic activity. According to Arner & Lindholm (1959a) 96% of their patients went in for sport to an extent considered beyond that of the normal population. 79 out of 89 patients stated that they were out of training, and 23 stated that the A.R. had occurred when they resumed their sport activities after a long interval.

Schauwecker et al. (1967) reported that 39 out of 48 cases of A.R. arose in connection with athletic activity, but that only 8% of the patients were athletes in training. Among 57 cases, Christensen (1954) found 26 to have occurred during athletic activity. In half these cases the A.R. was sustained when the subjects resumed their training. Freilinger et al. (1970) claimed that young patients, in particular, sustained their A.R. while at the top of their training. In Ljungqvist's (1968) material of partial ruptures too the younger group (19–30 years) were at the top of training.

Heredity

Nothing has been reported concerning a familial predisposition except that Toygar (1947) reported on a brother and sister who sustained A.R. at one year's interval.

Previous Diseases

In the early literature, in particular, there has been mention of diseases supposed to predispose to A.R. (syphilis, gonorrhoea, tuberculosis, typhoid fever, rheumatoid arthritis, and systemic infections), but none of these conditions has played any notable role in the large series which have accumulated up till now. On the other hand, a history of pain in the Achilles tendon, described as peritendinitis, seems to be fairly common in some materials. Judet (1964) reported that two-thirds of 166 patients with A.R. had had such complaints prior to the rupture. Riede (1966 a) stated that 11 out of 34 patients had been suffering from pain for varying periods before the rupture. Neither author mentioned whether these could have been partial ruptures which according to the description of Ljungqvist (1968) causes the very symptoms which have been interpreted as peritendinitis. Auquier & Siaud (1971) found that 3 out of 20 patients sustained their total A.R. after "tendinite nodulaire", a condition which they believed represented partial rupture. Others have not found anamnestic signs of diseases in the Achilles tendon prior to its rupture (Arner & Lindholm 1959a). In several large series no mention has been made of this aspect. Local or systemic steroid therapy as a cause of A.R. has been reported only as individual cases except for Ljungqvist's material (1968) in which half the patients had received local injection of steroid. However, the causal relationship was not quite evident.

Little has been stated concerning the patients' condition as regards fatigue prior to the episode of rupture. Hunter (1835) described how he sustained A.R. after having danced all night, and Debrunner (1950) reported a case in which A.R. arose after a long, fierce tennis tournament. Westlin (1969) found a relationship between fatigue and skiing injuries, but among 797 injuries only 2 were A.R.

Profession

Grouping by profession shows that people with sedentary work are most apt to sustain A.R. (Arner & Lindholm 1959a, Hooker 1963). In the former material only 2 out of 79 had heavy work.

Activity at A.R.

Practically all athletic activities have been mentioned. As stated by Kristensen & Thestrup Andersen (1970) the distribution differs geographically, depending on the favourite sports. In Denmark there is mainly a

question of badminton, tennis, football, and gymnastics. Solheim (1960) found a similar distribution in Norway. Among 79 A.R. in Solheim's material there were no skiing injuries, but at that time the slalom binding had not yet come into general use. In Germany, mainly football players are exposed (Frings 1969), and in addition short-distance runners, gymnasts, and handball players. In Switzerland and in some series from France, the majority of A.R. have been sustained by falling forward while skiing, a factor which acquired particular importance in the middle of the 50's, after the introduction of slalom bindings which firmly lock the feet to the skis (Mayr 1957). Philadelphy et al. (1971), in Austria, found 87 out of 119 cases of A.R. to have been sustained by falling on skis. Apart from athletic performances, walking, running, stumbling, and jumping down have been mentioned as activities at A.R.

Course of Accident

Direct A.R. occurs when the tendon is hit by a blow while it is tightly tensed by the triceps surae muscle. These cases make up between 1% (Arner & Lindholm 1959a) and 25% (Westermann 1962). The reported number of direct traumas may easily become too high. The fact is that many patients report having felt as if they were hit by something in the region of the heel, although this is denied by onlookers.

Where the indirect A.R.'s are concerned, the episode of the rupture has been described in all degrees of severity, from ordinary walking during which the patient merely has a feeling that the tendon gives way, to severe falls while skiing in which the skis are suddenly arrested, from great speed, by a non-sliding surface with the result that the body continues forward because of the inertia, whereas the foot is firmly locked to the skis.

Arner & Lindholm (1959a) found 3 main types in their material:

(1) Push-off with simultaneous extension of the knee in which they believed that the extension of the knee had occurred at a time while the foot was plantar flexed, so that only the forefoot was supporting weight. This type made up 49 out of 92 cases. For a discussion of the push-off cf. p. 87.

(2) Sudden, unexpected dorsiflexion in the ankle accompanied by strong contraction of the triceps surae. This may occur on falling forward with fixed foot or by stumbling in a hole (16 out of 92 cases).

(3) Jumping down on the plantar flexed foot accompanied by strong contraction of the triceps (9 out of 92 cases). In one case the trauma was direct, and in 17 it could not be classified.

Several authors (Roux 1964, Wellmitz 1965) have emphasized the role of the footwear as to whether the foot is fixed at the moment of the trauma (football boots with knobs, spiked shoes, tennis shoes). Quénu &

Stoianovitch (1929) stressed that often the trauma hit the tendon obliquely. "Le muscle et son tendon raidi se trouvent surpris dans une position anormale par rapport a l'axe habituel de leurs mouvements".

As a rule, only one trauma is reported, but "rupture en deux temps" has been reported by int. al. Quénu & Stoianovitch 1929, Davidsson 1956, Gaudin & Baud 1961, Hooker 1963, Dupont & Hayez 1970, and Ljungqvist 1968. The interval between the primary and secondary trauma has been reported to be from a couple of hours up to several months.

Symptoms and Signs

In connection with the episode of the rupture a noise – like the crack of a whip – has often been described, not only by the patient, but also by other persons present (Erskine 1971). The rupture often causes initial, severe pain, but painless cases of A.R. do occur. No attempt has been made to relate the whip crack to signs of degeneration, peritendinitis, or the degree of histological changes in biopsies from the ruptured tendon. On the other hand, a relationship between absent or negligible pain and signs of degeneration has been reported by Kolb & Salem (1953) as well as Dupont & Hayez (1970). Arner & Lindholm (1959a), however, found no relationship between the severity of the pain and the degree of histological changes. Other workers have not commented on this aspect.

Site

Schönbauer (1960) found 53 % of the A.R.'s to be localized 3–4 cm from the calcaneus, with decreasing frequency proximally and distally. This distribution corresponds to the findings of others.

According to Arner & Lindholm (1959a) and Schönbauer (1960), among others, the A.R. is always complete. However, Ljungqvist (1968) has given a convincing description of partial A.R. By site in the tendon Persson & Ljungqvist (1971) distinguished between lateral (14), medial (12), and central (2) ruptures and mixed types (12) in a material of 40 cases. Heim (1970), among 48 A.R.'s arising during skiing, found 3 partial ruptures, and Mounier-Kuhn (1971) found 9 partial ruptures among 98 operated cases. Denis (1971) had operated upon 3 partial A.R.'s. In Denmark partial A.R. was first described by Abrahamsen in 1923. Riede (1966 a), in his collected analysis, found that the left Achilles tendon was more often involved than the left (left 57 %, right 43 %). This he related to an analysis of the dominant push-off leg which was the left one in 58 % out of 532 school pupils. Not infrequently A.R. on one leg is followed, some time later, by A.R. on the other leg, according to Viernstein (1963) in 6% within one year. Only a few cases of A.R. simultaneously on both legs are on record, the first one being reported by Petit in 1722. According to Frings (1969) a total of 22 such cases are on record.

The reason why the cases in which the Achilles tendon tears off a fragment of the calcaneus are not included in the present paper is int. al. that the distribution of such cases by age and sex is entirely different from that stated above. The literature contains a number of case reports stating age and sex (Tuffier & Desfosses 1899, Helbing 1901, Wendt 1901, Fuchsig 1902, Rauenbusch 1906, Beesly & Price 1914, Mülleder 1922, Milch 1931, Wagner 1932, Fermaud 1935, Lasserre & Saft 1935, Struppler 1937, Papin 1938, Lang 1939, Rothberg 1939, Struppler 1939, Korn 1942, Sala de Paplo 1943, Cherry 1947, Schönbauer 1952, Arneth 1953, Arner & Lindholm 1959 b, Axt 1961, Decoulx et al. 1962, Legré & Trifaud 1965, Bierwag 1969, Lowy 1969, Protheroe 1969, Stampfel & Tcherne 1969). These authors have reported a total of 44 cases, including 26 females averaging 64 years in age and 18 males averaging 50 years. As a general tendency they found the trauma to be more severe in the male than in the female cases. Moreover, slipped calcaneal epiphysis has been described in a 13-year-old girl (Mooney 1935) and in an 11-year-old boy (Plettner 1971).

Treatment and Prognosis

The treatment consists in tendon suture, either simple suturing or suturing supplemented by some form of plasty. Only a few workers advocate conservative treatment (Lea & Smith 1968, Gillies & Chalmers 1970). The results of operative treatment appear to be fairly independent of the method used. In the various materials there have been only a few poor results, the remainder being classified as good or excellent. However, Trillat & Mounier-Kuhn (1971) found 12% poor results, the majority because of infection or re-rupture. Most authors have been able to report on top athletes who have returned to their sport after the A.R. and who have obtained results equal to or even better than their previous achievements (Lawrence et al. 1955, Riede 1965, Ljungqvist 1968, Frings 1969). Of Judet's (1964) 166 patients 92% went back to their sport.

Summary

A.R. seems to be occurring with increasing frequency, mainly in men aged 30–50 years. As a rule, it is unpreceded by local or systemic diseases, but often by athletic activity greater than in the normal population. A.R. often occurs at a time when the patient is out of practice, but as a rule in connection with athletic performances, sometimes associated with a trauma which seems inadequate. The left leg is more often involved than the right. The injury may be partial, but is usually total, localized 3–4 cm from the calcaneus. After suturing, the tendon shows such a marked tendency to healing that it can subsequently tolerate maximum stress. Avulsion fractures of the calcaneus tend to occur in an entirely different, and older group of patients than does A.R.

CHAPTER 3

PATHOLOGY

At operation for A.R. the tendon ends are seen to be oedematous, split up brush-like, separated by a haematoma whose appearance depends upon the time which has passed since the rupture. At times cysts are found at the tendon ends (Christensen 1954). There have been no descriptions of "smooth" ruptures of the tendon of the nature which may be seen in dead material due to material fatigue.

As a rule the peritendineum is intact (Christensen 1954, Arner & Lindholm 1959a, Pillet & Albaret 1972).

Data concerning the histological findings in tendon tissue are derived from three sources: biopsies removed at operation for tendon ruptures, autopsy material, and investigations of tendon tissue removed after experimental procedures.

Biopsy Materials

Histologically studied small series of A.R. were not published until the early 50's. Before that time, there had been merely case reports to which no major importance can be attached. Kolb & Salem (1953) studied 7 biopsies, 6 removed within 24 hours of the rupture. In small specimens they found patches with pyknosis or loss of the nuclei. The fibrils were homogeneous and acidophilic in these areas. Only mild changes were found in the vessel walls.

Lawrence et al. (1955) found 4 normal tendons in a series of 5 biopsies. Davidsson (1956) described 6 biopsies after A.R. Five showed degeneration, implicitly pretraumatic. In 3 of these 5 tendons there was granulation tissue (1, 2, and 7 days post-rupture), in one there was new-formed fibrillar structure (2 days post-rupture), and one exhibited neither formation of granulation tissue nor fibrils (2 days post-rupture). In all 5 specimens there were scattered fibrils of irregular course and in places varying degrees of necrosis. In the sixth specimen (2 months after partial rupture at the muscle-tendon junction) granulation tissue and new-formed fibrils were present, but no necrosis. In a subsequent study (Davidsson &

Salo 1969) two biopsies were examined 6–8 hours after A.R. in athletes who were out of training. In some areas there was normal tendon tissue, in other areas changes, especially in the form of thrombosed vessels with necrotic walls. These changes were definitely believed to have been present prior to the A.R.

Among 10 biopsies Mayr (1957) found 4 normal ones. The other 6 exhibited various degrees of mucoid or hyaline changes.

Orell (1958), and later Arner, Lindholm & Orell (1958/1959) described a total of 74 biopsies. Fifteen had been examined within 24 hours of the rupture. None contained granulation tissue. The early biopsies always showed oedematous splitting, fragmentation, and irregular position of the collagen fibres between areas of normal tendon tissue. Frequently, there would be areas of faintly staining, almost amorphous tissue, with few or no visible nuclei. In cases where the changes were very scanty the biopsy was small and could not be considered representative. At the end of a month or two most of the degenerative changes had disappeared, and granulation tissue had been largely replaced by poorly organized fibrous tissue. The histological changes were applicable for dating the ruptures. Two pathologists could independently carry out this dating with fair accuracy. The authors received the impression that the peritendineal arteries were affected by medial hypertrophy. They stressed that in their opinion there was not a question of acute tendon necrosis, but of necrobiotic changes, presumably due to reduced vascular supply.

Frings (1961), reviewing 88 A.R., found 3 biopsies with normal tendon tissue, but did not state how many biopsies had been taken.

Gaudin & Baud (1961) described 10 biopsies 3 of which had been removed during the first hours after A.R. They observed polynuclear cells and "lympho-plasmocytic" cells, but dared not establish whether the changes had been present prior to the rupture. Mucoid and fibrinoid changes were observed in only one case, but in this instance the history indicated that the rupture had occurred in two stages.

Lanz (1964) had occasion to study 3 biopsies within 2 hours of the rupture. He found no degenerative changes.

Legré & Trifaud (1965) reported on a case of calcaneal avulsion in which the calcaneus had been X-rayed before the rupture. At the site of insertion skeletal structure was sparse. Two months later the avulsion occurred. Biopsy showed in the bone decalcification (thinning of bony trabeculae?), nuclear pyknosis, hylainization, and increased formation of cartilage. Changes of this nature have not been described by others. On the other hand, Axt (1961) found in a biopsy of the tendon following calcaneal avulsion, degenerative changes in the form of lipid deposits, fibrinoid swelling, and wavy shrinkage of the adjacent tendon fibres.

Hooker (1963), among 5 biopsies from 46 patients with A.R., found

one showing signs of pretraumatic degeneration. Stavrache & Georgescu (1965), in a non-specified number of biopsies within 24 hours of A.R., found myxomatous degeneration, oedema, chronic cellulitis, sclerosing, and hyalinization, and in addition vasculitis of the obliterative type. Similar changes were found in other biopsies 1–3 months after the rupture.

In de Jong's (1966) opinion pre-existing degeneration must be a presupposition for A.R. Among 18 biopsies from 137 patients he found only 3 with mild changes.

Picaud et al. (1966) studied 62 biopsies from 130 cases of A.R. 12 had slight lesions of hyaline or sclerotic type. 25 biopsies taken within 4 hours of the A.R. were normal.

Dahmen (1966) described 6 biopsies taken more than 6 weeks after A.R. The changes were characterized as degenerative.

Lang & Viernstein (1966) divided biopsies from 30 A.R. into three groups. From 0–4 days after the rupture they found both normal and abnormal fibres, irregularly intermixed. The latter were undulating and split, staining a pale blue with alcian blue and pink with PAS. The capillary basement membrane was thickened, often surrounded by a PAS-positive halo. A few capillaries were completely occluded. Normal fibrocytes were not present, but nuclei were found in the most normal parts of the biopsies. In half the cases there were changes also 5 cm from the rupture. At the end of 4 days incipient granulation tissue was visible, and at this juncture the description corresponded to that rendered by Arner et al. (1958/1959). In the opinion of Lang & Viernstein the capillary changes were primary, followed by those of acid and neutral mucopolysaccharides.

Könn & Everth (1967) studied 64 biopsies from subcutaneous tendon ruptures, 27 of which were Achilles tendons. None had been studied before 24 hours had elapsed after the rupture. In 15 cases from 1 to 4 days had passed. In these specimens there was haemorrhage, splitting of collagen fibres, and occasionally vascular changes in the form of hyaline thickening of the arteriolar walls. In addition, they found – but did not further comment on – oedema, fragmentation, homogenization, and disappearance of nuclei. 12 biopsies were studied 5–14 days after the rupture. The appearances were characterized by granulation tissue, and fibrillar structure was disappearing. Later, necrosis as well as granulation tissue disappeared, being replaced by new-formed tendon tissue. In a few of the 15 early biopsies Könn & Everth found regenerative changes indicating pre-traumatic lesions.

In Schauwecker et al.'s (1967) material of 48 cases, 35% showed signs of degeneration. They did not mention the interval passing from the A.R. to the operation or whether biopsies had been taken from all 48 cases.

Ljungqvist (1968) described 24 biopsies removed at operation for partial rupture. In all the specimens there was more or less devitalized tendon tissue with an altered or reduced stainability, split fibres, and areas which took no stain at all, not even in the vessels. In all cases of more than 5 days' standing there was granulation tissue and new-formed tendon tissue. Within this material, comprising mainly active athletes, Ljungqvist found no vascular changes.

Studying 14 biopsies among 37 cases Kristensen & Thestrup Andersen (1970) found normal appearances in 9 and varying degrees of degenerative changes in 5.

Ralston & Schmidt (1971) felt that the changes found in 5 biopsies taken within 4 days of A.R. had to be considered posttraumatic.

Autopsy Materials

Achilles tendons in autopsy materials have been studied by Chaletzkaja (1934) who found, in 48 persons aged 3–82 years, increasing extracellular deposition of lipids, often with cholesterol crystals and more diffusely a protein-like substance. The changes increased parallel to arteriosclerotic changes in the vessels.

Schneider & Grilli (1955) examined 60 Achilles tendons (from 30 individuals?) taken at autopsy. All the persons were over 37 years of age. The site of the changes appears to have been close to the insertion, but is not specified. They found lipid deposits in 50 % of the tendons, but did not mention the frequency of other changes, viz. islets of mesenchymal cells, necrosis, decomposition of nuclei, small cysts, and fragmentation. In their opinion, pressure by shoes was responsible for some of the changes. According to Schneider (1959) lipid deposits followed upon vascular changes (arterio-sclerosis), but he did not state whether the degenerative changes were related to the lipid deposits. Incidentally, he emphasized that the degenerative changes had their site of predilection close to the insertion of the tendon, i.e. not where ruptures are most apt to occur.

In a monograph from 1966 Dahmen tried to distinguish between changes of ageing and degenerative changes. *Changes of ageing* consisted of desiccation, decrease of interfibrillar substance, lipid deposits, increasing thickness of the fibrils, longer striation periods in the fibrils and a larger number of striations in each period. Lastly, the content of acid mucopolysaccharides decreased. The *degenerative* changes, said to be irreversible, consisted macroscopically of a yellowish staining of the tissue which was non-glistening and soft. On microscopic inspection the structure was looser, the tendon poor in nuclei, the fibres split up, and double refraction decreased, indicating a poorer orientation of the fibres. There were numerous thin fibrils. The striation periods were shorter and the number of striations smaller. The amount of acid mucopolysac-

charides increased. The normal material was derived from 7 persons (18 months – 66 years of age), partly from amputated limbs and partly from tenoplastic operations. In the normal material Dahmen found no lipid deposits in an extracellular situation and no signs of necrosis or splitting. The degenerated material consisted of tissue removed at operation for tendon rupture, slipped disc, or meniscus injuries. The technique was very thorough: examination in polarized light, electron microscopy, X-ray diffraction, and finally PAS staining and staining with alcian blue for demonstrating acid mucopolysaccharides.

Lang & Viernstein (1966) found no abnormality in the investigation of 10 grossly normal Achilles tendons. Incidentally, they stated that one tendon "ready for rupture" could be expected in each half-million.

Könn & Everth (1967), in connection with the histological examination of biopsies from ruptured Achilles tendons, studied 123 "normal" tendons from persons aged 0 – 71 years. Apart from the conventional staining methods, they used also alcian blue and PAS. In normal tendons they found in places mild hyaline changes. Two cases with arteriosclerosis exhibited more severe vascular changes accompanied by oedematous splitting and thickening of the fibres around a necrotic area.

Experimental Investigations

The first major experimental investigation for elucidating the pathology of the tendon was performed by Palla in 1909. He crushed the Achilles tendon in rabbits with a Kocher forceps. After a varying period the rabbits were killed and the tendons studied histologically. At *3 hours* he found tendon bundles swollen, in places lumpy, the nuclei of the tendon cells swollen and polymorphous. At *6 hours* the nuclei were scanty, serrated, polymorphous. The tendon bundles were lying pell-mell. Polymorphonuclear leukocytes were found in the surroundings. At *15–24 hours* there was invasion by leukocytes. The nuclei were barely visible, and fibrillar structure had practically disappeared. At *48 hours* there was further leukocytic infiltration and young connective-tissue cells. At *3 days* there was total absence of nuclei, and fibrillar structure was discernible only in places. At *6 days* regenerative processes began to predominate. At *5 weeks* there was in one case bony as well as cartilaginous tissue at the site where the tendon had been crushed.

Wrete (1951), training guinea-pigs in running 2 x half an hour daily for 2 months, found no significant changes in metachromasia, viz. no sign of an altered acid mucopolysaccharide content. Rollhäuser (1954 a) also found no microscopic changes after having trained guinea-pigs in running.

Borsay et al. (1952 a, b) severely over-stressed the dog Achilles tendon by direct stimulation of the triceps surae for 5–10 minutes daily. At the end of 5 days they found splitting and small transverse fissures as well as

protein precipitation. At 19 days there were similar, more marked changes, and in a few places no nuclear staining. In addition, incipient lipid deposits. At 37 days the changes were even more marked, showing incipient granulation tissue in the fissures which extended almost right across the tendon. Borsay concluded that the lipid deposition occurred only after previous injury to the tendon and that by severe over-strain it was possible to induce changes of the type seen in human tendon ruptures.

In the experimental part of his study, Davidsson (1956) ligated the Achilles tendons of rabbits in 2 sites 2 cm apart. At the end of 1 week he found the tendon to be necrotic, the fibrils fused, homogeneous, and only remnants of nuclei left. In between, however, there was well-preserved tendon tissue. At 3 weeks there was unmistakable ingrowth of granulation tissue. At 5 weeks the necroses had disappeared. In between vessels and cellular tissue there were numerous, thin collagen fibrils. Even at 5 1/2 months the tendon had not assumed its normal glistening appearance, and on microscopic inspection the tendon tissue was extremely irregular and cellular.

Dahmen (1966) carried out experiments similar to those of Borsay et al. and Davidsson. In the stress experiments he found less marked microscopic changes than Borsay did, in particular very scanty lipidosis. The fibrils were thinner than normal, and there was mild, irregular metachromasia. In the ligation experiments similar changes were found in the tendons examined 6–10 weeks after the ligation. The considerable regeneration described by Davidsson was hardly seen, and it was as if the thin fibrils were not new-formed, but degradation products of the original fibres. Dahmen also investigated the effect of injecting hyaluronic acid into and around the Achilles tendon. The changes found were very mild, but of the same nature as those mentioned above. He emphasized the monotonous reaction of the connective tissue to widely different actions. In his opinion, this uniform reaction was due to primary changes in the mucopolysaccharides.

Lang & Viernstein (1966) cut the Achilles tendons of rabbits without inflicting other injuries upon them. This study was performed in order to assess which changes may be expected in the two tendon ends following rupture. After a varying number of days (1–14) they still found normal tendon ends. In contradistinction, others have found that after being cut the rabbit Achilles tendon reacts by splitting of the fibrils even within 24 hours (Bertolini et al. 1970) and within 8 days by swelling and cellular pyknosis (Skoog & Persson 1954).

After storing tendon tissue in saline solution for 24 hours, Viidik & Lewin (1966) found the fibres to have become oedematous, less coherent, and moreover the fibrocytes had disappeared or had become pyknotic.

Comments

A statistical analysis of the above-mentioned results of studying tendon biopsies in A.R. cannot form a basis for estimating whether or not the tendon was healthy at the time of trauma. The reported results are so conflicting that the differences, as stated also by Guillet et al. (1966), must be due to the pathologist's personal evaluation of the rate at which the changes may arise and of the nature of these changes. Changes in the histological appearances appear to be fairly stereotyped following widely different actions (Dahmen 1966). According to Palla's and Bertolini's *in vivo* and Viidik's *in vitro* experiments, the rate at which the changes appear seems to be considerably faster than generally believed.

CHAPTER 4

PREVIOUS RUPTURE EXPERIMENTS

In 1880 Segond described the first rupture experiments on the intact bone-muscle-tendon-bone unit. In an autopsy series he exposed the distal phalanges of fingers to sudden, strong flexion and thereby induced avulsion of extensor tendons with a small chip of bone from the distal phalanx. In elderly persons the bony fragment was larger. He found that the injury was easier to produce in women than in men. It is not apparent from his publication whether some of the breaches were tendon ruptures.

Busch (1881) was unable to reproduce these experiments, either with respect to tendon ruptures or bone avulsions.

Schoening, in 1887, performed similar experiments resulting in the avulsion of a bony chip, as found by Segond, and in youngish individuals in a central rupture of the tendon close to its insertion into the distal phalanx. Rupture could be induced even after cutting the extensor tendon on a level with the metacarpophalangeal joint, viz. proximally to the attachment of the lumbrical and interosseous muscles. It was a *sine qua non,* however, that the interphalangeal joint was extended. Haegler (1896) confirmed Schoening's results. In two-thirds of his cases he found avulsion of bone and in one-third tendon rupture. Moreover, in an attempt to induce rupture in the flexor profundus tendon, he obtained in one case tendon rupture close to the insertion. Neither of the two last-mentioned authors mentioned pretraumatic degeneration as a presupposition of rupture.

The first animal experiments were carried out by Sallefranque in 1887. A dog was anaesthetized with ether, and during simultaneous tetanic stimulation of the muscle the biceps brachii, quadriceps femoris, and triceps surae were investigated. The dog succumbed already after the first experiment. In a total of 7 experiments he found 2 avulsions of bone, one breach at the muscle-tendon junction, and 2 tendon ruptures in the left and right biceps tendon. In addition, Sallefranque tried to pull the biceps brachii apart in 3 cadavers. Each time, the result was muscle rupture. The experimental conditions are cursorily described and permit no conclusion.

Blix (1895) reported that the tetanized frog muscle could tear itself apart, but he did not give any details of the experimental set-up.

Loos (1901), like Sallefranque, performed rupture experiments on cadavers and obtained muscle rupture of the biceps brachii. Rupture experiments on frog muscles resulted in ruptures at the muscle-tendon junction, whether or not the muscle was tetanized. In these experiments, the tetanized muscle could not tear itself apart.

Wendt (1901–02) performed a few rupture experiments on human calcaneus-Achilles tendon preparations. At a load of 100 kp tendon rupture occurred in one case, avulsion of small fragments from the calcaneus in one, but in the remaining cases no rupture. The load was strikingly light and details concerning the experiments are lacking.

The rupture experiments which are most often cited were conducted by McMaster in 1933. Immediately after killing young rabbits he carried out rupture experiments on a gastrocnemius-Achilles tendon preparation suspended by the femur and by the calcaneus. The latter was separated from the adjacent bones. It is not apparent whether the soleus muscle was included in the set-up. The loading was performed in part slowly and in part rapidly – the latter by letting a heavy weight fall. Stimulation of the muscle was not used. With this experimental set-up the long axis of the calcaneus was in continuation of the tendon axis, i.e. corresponding to more than extreme plantar flexion. In 9 experiments without previous injury to the tendon he found 2 ruptures at the point of fixation, 5 avulsions of bone, and 2 muscle ruptures. After one-quarter severance of the tendon or after crushing the tendon by a Kocher forceps, the result was also a rupture in muscle or bone. The tendon itself did not rupture until it was half-severed. Injury to the tendon by crushing 2–5 weeks before the rupture experiment did not result in tendon rupture. A series of experiments in which the tendon was ligated in two sites, 1 cm apart, resulted in 10 days, 2 weeks, and 3 weeks in rupture in muscle or bone. Not until 5 weeks had passed did a tendon rupture occur at the ligated site. McMaster's conclusion was, without any reserve, that a healthy tendon never ruptures.

Lindblom (1939) tried to induce rupture in the human supraspinatus tendon. The muscle was fixed with numerous sutures. On loading the tendon between the humerus and these sutures muscle ruptures occurred when the humerus was adducted at the time of loading, but tendon ruptures when it was abducted. Lindblom emphasized that in this position the inner part of the supraspinatus is tense, whereas its outer part is relaxed. He did not state that internal or external rotation would be able to cause a similar uneven loading.

Without being aware of Lindblom's publication from 1939, and without paying any regard to the direction of the loading, Wilson & Duff (1943) carried out a similar experimental series without inducing tendon rupture.

Fink & Wyss (1942) studied a femur-gastrocnemius-Achilles tendon-calcaneus preparation from frogs, both with and without stimulation of the muscle. The experiments were carried out with dynamic as well as with static loading. Under all circumstances muscle rupture occurred, either in the muscle belly or at the muscle-tendon junction.

Stucke (1949) fastened human Achilles tendon-calcaneus units in a material testing machine. The experimental set-up was not described in any detail, also not the magnitude of the load, the patient's age, or cause of death. The tendon, with small fragments of bone, was torn from the calcaneus. Stucke found no avulsion of a triangular fragment as seen in human pathology. In subsequent experiments on a bone-muscle-tendon-bone unit on dogs, Stucke (1951) did not use external loading, only stimulation of the muscle. This showed that four-fifths of the tendon had to be severed before the tetanized muscle could cause rupture of the remainder.

Borsay et al. (1952 a, b) did not carry out actual rupture experiments, but studied the effect upon the Achilles tendon of direct tetanic stimulation of the gastrocnemius for 5+10 minutes daily through 5–37 days. Their experimental animals were hares and dogs. After 37 days' stimulation incomplete rupture occurred in one of the dogs. The histological changes have already been described above (p. 22). The experimental procedure seems extremely unphysiological, partly because the stimulation was very prolonged – for a tetanic stimulation – and partly because it was administered as direct stimulation of the muscle instead of indirect stimulation of the nerve.

Davidsson (1956) used mature anaesthetized experimental animals, cats and rabbits. He did not state whether the cats were wild or domesticated. In the majority of the experiments the gastrocnemius + soleus were stimulated indirectly via the sciatic nerve, and simultaneously the loading was performed by a 30 kg falling weight. The fastening was done through burr holes in the femur and calcaneus. No mention is made of the direction of traction. The result was in about half the experiments avulsion of the tendon with a chip of bone or avulsion at the attachment of the muscle. In the remaining experiments rupture occurred at the insertion. These ruptures are not described in detail. In particular, it is not apparent whether any tendon remnant was left behind on the calcaneus. In further experiments the Achilles tendon was first severed 1/4, 1/2, or 3/4. The latter two resulted in ruptures at the incision, whereas at 1/4 severance the rupture occurred elsewhere. On rabbits the tendon was ligated in 2 sites. Rupture experiments 10 days to 4 weeks later resulted in tendon rupture between the ligatures, 6 weeks later in rupture elsewhere, which is the very opposite of McMaster's results (1933). Davidsson was unable to advance any explanation.

Viidik (1968 a and 1969 b) performed rupture experiments on a calcaneus-Achilles tendon-gastrocnemius-femur preparation after killing the animal. The experimental animals were rabbits, partly untrained and partly rabbits trained in running for 40 weeks from the age of 3 months. Although the training was performed at a rate as fast as possible, the rabbits only ran about 1/2 km daily. For comparison, it may be mentioned that domesticated rats in revolving cages have done 3–8 km in the 24 hours (Slonaker 1912). In the preparation prior to the rupture experiment the soleus was detached from the lower leg for some reason not stated. It is beyond doubt that the distribution of the forces has thereby been altered, but it is impossible to say whether this procedure has affected the results. The calcaneus was placed in a holder of metal which could secure a position corresponding to 90° flexion in the ankle. Tear-off fractures were the most common result. In the exercised group as well as in the control group a few ruptures occurred in other sites, but no tendon ruptures.

Tittel & Otto (1970) performed rupture experiments on rat Achilles tendons after various forms of training. After sacrifice the calcaneus and the muscle-tendon junction were fastened in a material testing machine, i.e. not the entire bone-tendon-muscle-bone chain. The rupture was found to occur in the distal third of the tendon. It is not stated whether the site of the rupture was dependent on the state of training.

Welsh et al. (1971) studied rabbits, but did not report their age or state of training. By loading the bone-tendon-muscle-bone chain they found exclusively muscle ruptures (gastrocnemius). The experiments were carried out after sacrifice without stimulation of the muscle.

Summary

The injury which McMaster (1933) and Davidsson (1956) could inflict on the tendon in animal experiments without this area becoming the weakest link in the bone-muscle-tendon-bone chain speaks for the view that the healthy tendon never ruptures.

However, Schoening (1887), Haegler (1896), and Lindblom (1939) did succeed in inducing tendon ruptures. It is common to their experiments that they were done on human cadavers and that the tendons were unevenly loaded by a change in the position of the bone into which they inserted. Wendt (1901–1902) induced rupture of the human Achilles tendon, but the circumstances of his experiment are far from clear.

Tittel & Otto (1970) have induced Achilles tendon ruptures in rats, but only on part of the bone-tendon-muscle-bone chain.

None of the animal experiments has fulfilled the demands which should be made. In order to correspond approximately to clinical conditions (cf. p. 17) the experimental animals have to be mature and in a reasonable

state of training. The latter cannot be obtained in caged animals. Only Davidsson (1965) and Viidik (1968 a, b) have studied adult animals and only Viidik has tried to procure a well-trained experimental animal.

No tendon ruptures occurred in Viidik's experiments. It may be objected to his experimental method, as mentioned already, that the soleus had been detached from the lower leg, whereby the distribution of force may have been altered and that the training programme for the animals does not appear to have been particularly severe. Moreover, the muscle was not stimulated during the rupture experiment, and – as emphasized by Viidik himself – the anatomy of the rabbit Achilles tendon at its insertion differs from that in man in being almost punctate in an apparently weak calcaneus.

Thus, the results are not as consistent as interpreted by those who have tried to evaluate the aetiology and pathogenesis of Achilles tendon rupture, and they do not afford sufficient evidence for the oft-repeated claim that tendon ruptures cannot be induced experimentally in a healthy tendon.

CHAPTER 5

PREVIOUS THEORIES

Two main views prevail concerning the origin of A.R. One is that the tendon never ruptures without being affected with pathological changes. The other view is that the normal tendon may rupture under unfavourable circumstances – especially mechanical. Several authors have expressed doubt, e.g. Adler (1971) who wrote: "Es ist aber zu bedenken, dass eine gesunde Sehne eigentlich nie rupturiert".

Degeneration Theory

In the opinion of some authors the pathological changes may have been caused by tendinitis or peritendinitis due to overstrain (Grassheim 1922, Schönbauer 1960, Viernstein 1963, Judet 1964). However, clinical signs of such aseptic inflammation have been found in only a minority of patients with A.R. Others believe that overstrain, either on a single or on repeated occasions, may cause minor ruptures which add up, if the power of regeneration does not keep pace (Borsay et al. 1952 a and b, Kolb & Salem 1953).

According to most authors the changes in the tendon are due to reduced vascularization, caused either by changes in the vessel wall (hypertrophy of the media) (Stavrache 1965, Riede 1966 a), by vascular thrombi (Davidsson & Salo 1969), or by a reduced number of capillaries per tissue volume and thereby an increased distance for the oxygen to diffuse (Delarue & Denoix 1946, Arner & Lindholm 1959a, Schneider 1959, Freilinger et al. 1970, Könn & Everth 1967).

Möseneder & Klatnek (1969) thought that a reduced venous pump function, e.g. in motorists, may cause reduced supply of oxygen and thereby degeneration.

A main objection to vascular changes in explanation of insufficiency of the tendon is the good power of regeneration after A.R. (Gaudin & Baud 1961). This argument would not seem weighty, as healing of the tendon occurs by ingrowth of vessels and granulation tissue from the surroundings, not from the tendon proper which is a priori poorly vascularized

(Stavrache & Georgescu 1965). A more serious objection to the vascular theory is that the published series do not include groups of patients with generalized vascular diseases, such as arteriosclerosis or diabetes. However, of Schubert's (1971) 5 patients with rupture of the quadriceps tendon just proximal to the patella 4 were diabetics and one arteriosclerotic.

The main arguments for the degeneration theory have been the results of the previously mentioned histological studies and of the rupture experiments, but none of these results can be considered definitive. Lastly, the feeling that the trauma has not been adequate is an essential reason for searching the explanation of an A.R. in a pathological condition of the tendon. Schönbauer (1960) reported the causal relationship as follows: Slight trauma – severe degeneration, severe trauma – mild degeneration. However, he did not think that even the most severe trauma could induce rupture in a healthy tendon.

Others feel that some of the reported traumas are so severe, especially skiing accidents, that the ruptures must be considered purely traumatic (Stucke 1961, Ricklin 1962).

Mechanical Theory

Adherents of the mechanical theory (int. al. Christensen 1954, Solheim 1960, Roux 1964, Picaud et al. 1966, Frings 1969, Dupont & Hayez 1970, Schmidtsdorff 1971) agree that the trauma consists of a sudden lengthening of the tendon-muscle unit with simultaneous strong contraction of the muscle. Only the earliest authors believed that contraction of the triceps surae alone was sufficient (Richerand 1821).

Silfverskiöld (1941) emphasized that the muscle-tendon groups in which tendon rupture occurs are pluriarticular, whereby the straining of the tendon may take place very quickly. Quénu & Stoianovitch (1929) and Lindblom (1939) believed that an oblique traction on the tendon rendered it more vulnerable. Contracture of the muscle, e.g. due to the use of high-heeled shoes, may perhaps increase the vulnerability of the tendon (Sjöström 1759, Picaud et al. 1966). Albrecht (1925) thought that pressure by the edge of a boot was a factor in the occurrence of A.R. In the anatomical structure of the tendon (cf. p. 37) Christensen (1954) believed there was a possibility that one part of the tendon (gastrocnemius) can damage the other (soleus) by a saw-like effect. Maydl (1883) believed int. al. that a spiral-like rotation of the tendon during loading might be unfavourable. The stiffness which affects tendon tissue with advancing age is considered by many as contributory to A.R. (Gilcreest 1933, Davidsson 1956, Viernstein 1963). These authors emphasize that changes of ageing are not interpretable as abnormal.

According to Pirker (1934) the sudden loading of the tendon gives rise to an "affect-like" defence reaction. Brugisser (1956) believed that long,

proprioceptive paths were blocked by short reflex arcs, so that the defence reactions of the organism against damage to the tendon do not have time to manifest themselves. Riede (1965) and Grafe (1969) reported that the sudden stretching causes a tendon stretching reflex which was supposed to induce a stronger contraction than a voluntary muscle contraction because of synchronization of motor units.

Some authors have fastened upon the condition of the muscle, either that it is cold (Picaud et al. 1966, Schauwecker et al. 1967), or that it is tired (Hunter 1835, Debrunner 1952). A disproportion between the tendon and muscle due to training may also be a contributory cause of A.R. (Bate 1951). Apart from this, the properties of the muscle seem to have been fairly unheeded. In particular, none of the above-mentioned authors has studied muscle physiology to elucidate the magnitude of the muscle force. Only Grafe took an interest in this aspect, finding that the Achilles tendons are exposed to a total load of 1070 kp in the landing and subsequent push-off in a double somersault.

Thus, the only author who has dealt with the kinesiological aspect of the problem found that the load on the Achilles tendon may easily approach or exceed the 400 kp (Stucke 1950) which most authors report as the rupture limit of this tendon.

Insurance

In insurance cases A.R. is generally considered a disease. Hagen (1969) wrote that A.R. was accepted by the Danish Directorate of Employment Injuries Insurance only if there had been a "really extraordinary event". From the examples adduced it is apparent that A.R. after a strong push-off to move a sewer tube is not accepted, whereas in connection with a handball tournament it is, since "there may easily arise situations which expose an Achilles tendon to severe trauma without the injured person being able to describe in any detail a special event as the presumed cause". Similar views are held by insurance companies in West Germany, Austria, and Switzerland, a few major traumas being accepted, although it is still believed that mild degeneration has been present before the A.R. (Probst 1967, Reichenbach 1967, Tillmann 1968, Treptow 1970, Philadelphy et al 1971). Hohorst (1967) emphasized that in each individual case one must assess the patient's weight, the acceleration during the traumatizing movement, and the histological condition of the tendon.

Comments

These theories are based to a great extent upon clinical and histopathological experience as well as the results of some of the rupture experiments quoted above. Less attention has been given to the structure and function of the tissues which make up the bone-muscle-tendon-bone chain. These

three types of tissue will be described in more detail in the following chapters, with a special view to factors which may be considered relevant in the aetiology and pathogenesis of Achilles tendon rupture.

CHAPTER 6

THE TENDON

Anatomy of the Tendon

The Achilles tendon is the organ which transmits forces from the gastrocnemius and soleus muscles to the calcaneus. The former muscle arises from the posterior surface of the femoral condyles and the latter from the upper end of the tibial and fibular posterior surfaces and the interposed tendinous arch.

The tendon aponeuroses from the 3 muscle bellies join to form the common tendon 10–15 cm above the calcaneus. The muscle fibres from the gastrocnemius extend to 11–26 cm and those of the soleus to 3–11 cm above the calcaneus (Cummins et al. 1946). Thus, the length of the tendon is not well-defined. 12–15 cm from the tendon insertion a pronation of 30°–150° begins (White 1943, Cummins et al. 1946, Biro & Tarsoly 1967). Cummins et al. (1946) as well as Keller (1951) found that the individual fractions of the tendon were almost impossible to separate, when the twisting of the tendon was slight, whereas it was easy in cases with pronounced rotation. This difference was due to marked interdigitation of the tendon fibre bundles in cases with little twisting. Interdigitation appears to be the alternative to twisting.

The twisting involves crossing of the tendon fibres 2–5 cm from the calcaneus (Biro & Tarsoly 1967). At this level the tendon is almost circular, having a diameter of 1–1 1/2 cm. Above as well as below it fans out, and at the insertion it has a transverse diameter of 1 1/2–2 1/2 cm.

The fan-shaped spread of the tendon close to its insertion has been described in detail by Mollier (1937), Schneider (1956), and Altmann (1963). In addition to the fanning out of the fibre bundles there is further splitting and interdigitation towards the insertion. The most peripheral parts of the tendon continue into the periosteum, the remaining fibres into the bone (Vis 1957, Cooper & Misols 1970). This spread of the tendon is not believed to involve an increase of the fibre mass (Mollier 1937, Cooper & Misols 1970), but this has never been studied quantita-

tively. The cross-sectional area itself is probably increased, as there are areas of loose connective tissue in between the tendon fibres and of slender fibrocytes converted into chondrocyte-like cells arranged in columns as in cartilage. Frequently, there is a saline track marking the junction to that part of the cartilage which is calcified (Cooper & Misols 1970). Hence, the junction to the bone is not sharp (Becker 1971).

Edwards (1946) stated that the vascular supply of the tendon was derived from the muscle- and bone junctions, not via these tissues, but via periosteal and peritendineal vessels. Moreover, on the anterior aspect of the tendon there is an arcade (Schnorrenberg 1962, Minne et al. 1967) whence the vessels, in a kind of mesotendon, enter the tendon. These vessels must be considered the most important ones to the tendon (Schnorrenberg 1962, Gambier et al. 1962, Minne et al. 1967, Schatzker & Brånemark 1969). The vessels are longitudinally situated between the primary bundles, connected by arcades. The density of the vessels in man is said to be least in the middle third of the tendon (Lagergren & Lindholm 1958/59, Schnorrenberg 1962, Minne et al. 1967), assessed by microangiography as well as by other injection techniques. Using stereomicroangiography on the rabbit Achilles tendon, Bergljung (1968) found a uniform vascular supply throughout the tendon. Bergljung's studies demonstrated, at the same time, that only injection of contrast medium *in vivo* is able to visualize all the vessels.

The nerve supply of the tendon has been described by int. al. Ralston et al. (1960). They found free nerve endings from nerves $< 3\ \mu$, presumably pain receptors, and un-encapsulated and encapsulated nerve endings derived from $5-12\ \mu$ thick nerve endings, presumably serving a proprioceptive function.

Marchand et al. (1971) found tendon spindles and muscle spindles in close relation to each other. The tendon spindles found at the muscle-tendon junction are believed to record the tension in the various parts of the muscle, whereas tendon spindles localized more distally in the tendon are believed to record changes in total tension. According to Houck et al. (1971) tendon spindles react only to an increase in tension arising because of active contraction, whereas the increase in tension occurring with passive movements within the physiological range does not exceed the threshold value of the tendon spindle.

Lang (1960) described Vater-Paccini corpuscles in the peritendineum around human Achilles tendons, presumably operative in the posture sense.

None of the authors who have been concerned with the nerve supply of the tendon has described abnormal conditions which might represent impaired function.

The Achilles tendon is situated in the posterior lower-leg fascia

together with the rudimentary plantaris tendon. The layer of fascia is strongest distally where it is difficult to separate from the posterior part of the tendon (Lang 1960, Schnorrenberg 1962). In the opinion of Loetzke (1956) and Schnorrenberg (1962) this fascia might contribute to the slight antecurvature of the Achilles tendon in this area. More proximally the fascial splitting exhibits the nature of a tendon sheath, but without any synovial lining. As already mentioned, the "tendon sheath" has been found intact in operations for A.R. (p. 18).

The calcaneal insertion takes place in the middle third of the calcaneal posterior surface. Stucke (1949) reported that in man the tendon insertion moved more proximally with advancing age. Between the tendon and the upper third of the calcaneus there is partly a thick-walled bursa and partly fatty tissue (Weston 1970). Corresponding bursae are found between the posterior aspect of the femoral condyles and the upper part of the gastrocnemius muscles.

The Achilles tendon passes two joints without being in any contact with either. The first joint is the talo-crural articulation whose axis is approximately at right angles to the Achilles tendon in the frontal plane. The shape of the talus makes a slight change in the position of the axis in dorsal and plantar flexion (Hicks 1953). This causes slight rotation at the time of the flexion-extension movement.

The axis of the subtalar joint is directed from the posterior lateral corner of the calcaneus 42° upwards and 16° medially (Manter 1941). Thereby, the triceps surae also acts as a supinator on the calcaneus. The mobility in the subtalar joints was measured by Ahlberg (1940) as 20–30° supination and 10–15° pronation. Ahlberg found no change in mobility with advancing age, whereas Hübscher (1901) observed decreasing mobility in perimetric measurements. Widén (1954) too found a slightly reduced mobility with age.

The lever arm on which the Achilles tendon acts in relation to the ankle axis is presumably subject to marked individual variations, but the position of the ankle joint is also decisive to the length of the lever arm, i.e. the perpendicular distance from the Achilles tendon to the axis of the ankle. From a slight plantar flexion in the ankle, plantar flexion as well as dorsal flexion will reduce this distance (Athabegian 1903). This distance has been reported to be from 3 to 6 cm (Reys 1915, Elftman 1939 a, Gersten 1956, Smith 1957, and Grafe 1969). For the use of computerization of the tension in the Achilles tendon during skiing, Quigley & Chaffin (1971) established a formula for this distance. They found that the distance decreased from less than 5 cm in a neutral position of the ankle to 3 cm at 35° dorsal flexion. The other relevant lever – from the ankle axis to the metatarsal heads – has been reported to be 2.7 times greater (Haxton 1944, Gersten 1956). The leverage system around the axis of the

subtalar joint may also be operative in stresses on the Achilles tendon as well as in forces which the Achilles tendon can transmit from the m. triceps surae to the os calcis in supination.

The knee joint also has a direct influence upon the Achilles tendon, partly by its great mobility in extension/flexion and partly due to the possibility of rotation in the knee joint which amounts to about 25° passively in 160° flexion (Lindahl & Hallén 1965). This rotation may entail torsion of that part of the Achilles tendon which is derived from the gastrocnemius muscles. Perhaps this torsion may increase the saw action which Christensen (1954) believed was a contributory cause of A.R. (cf. also p. 31). In addition, rotation of the lower leg in relation to the femur will be reduced in extension. Thereby forced rotation of the foot may occur (supination on lateral rotation of the lower leg) (Jones 1945).

The lever arm for the gastrocnemius muscles in relation to the knee-joint axis is 3 cm with extended knee and about 2 cm with flexed knee (Bugnion 1892).

The maximum difference in length which the movements in the knee and ankle joints can induce in the triceps surae + Achilles tendon has been reported by Riede (1965) to be about 10 cm corresponding to a change in length from about 35–45 cm.

Role of the Direction of Traction

To transmit muscle force at various directions of traction is a function of the tendon which has been discussed by various authors, especially by Mollier (1937), Schneider (1956), and Altmann (1963). Particular attention has been given to the kinking which may arise at the transition from a fairly yielding material (the tendon) to practically unyielding material (the bone). The structure of the tendon seems to be in many respects well-suited for avoiding or reducing this kinking.

The direction of traction at the insertion may be maintained by the tendon "utilizing" the bone as a bar or by the tendon being kept in position by tendon retinacula. Despite great mobility in the knee joint the direction of traction at the origin of the gastrocnemius muscles will be maintained fairly constant, because the muscle rests on the condyles. The quadriceps tendon – the inferior patellar ligament – is also maintained in the same position in relation to the tibial tuberosity. The great pressure loading of a tendon which may result from this is often accompanied by cartilage or bone formation as demonstrated by Ploetz (1938) who showed, by transposition of rabbit tendons, that cartilage formation might appear and disappear depending upon the pressure forces. The direction of traction of the Achilles tendon in dorso-plantar flexion is maintained as follows: In maximum dorsiflexion the tendon rests against the bar constituted by the upper part of the calcaneus and the interposed bursa,

and in plantar flexion the direction is maintained by the posterior crural fascia described above. Since, at the same time, the tendon is flat antero-posteriorly at its insertion, the difference in loading of the individual tendon fibres will not be particularly great in the movement of flexion/extension.

In supination-pronation movement of the calcaneus there is nothing to maintain the direction of traction except for the cartilaginous structure in the juxta-skeletal part of the tendon. This cartilage-like structure is believed to increase its rigidity. Thereby, the kinking is distributed over a longer part of the tendon. The result has been compared to the effect of the stiff cuff placed around an electric flex where it enters e.g. an electric iron (Petersen 1930, Mollier 1937, Schneider 1956). Mollier (1937) described the fan- or funnel-shaped spread of the tendon at its insertion. He reported that always there would be tendon fibres whose direction, due to the embedding in the bone, corresponded to any direction of muscle traction. According to Mollier, marked sliding of the tendon fibres would entail that the highly angular fibres at the other side of the tendon would also be loaded, but this internal displacement was prevented by the tendon structure, viz. by the considerable interdigitation present in the tendon. This interdigitation was also supposed to entail that the entire muscle force could be transmitted to the bone – not to the entire insertion, but to a small part of it.

Elliott (1965 a), quoting Mollier (1937), wrote that "the tension is transmitted to all parts of the insertion in all positions of the joint". As is apparent from what has been described above, Mollier did not postulate such an ideal state. He also did not establish, on theoretical or on experimental grounds, what weakened the tendon more: simultaneous angulation of parallel tendon fibres or angulation of a funnel-shaped insertion where major or minor parts of the tendon are not participating in weight-bearing.

As already mentioned, the degree of rotation of the Achilles tendon appears to be an alternative to the degree of interdigitation. It is an experience from cable industry that twisted structures are particularly resistant to bending stress, whereas plaited structures are particularly resistant to rotation stress (Knudsen 1972). One of the presuppositions that the plaiting or twisting is able to strengthen the material against the named stresses is that the fibres can slide in relation to one another, i.e. that a kind of lubricant is present (Meier 1961). In the tendon it is believed that the mucopolysaccharides serve this function, among others, but the extent of or variations in this function are unknown.

The way a metal cable is constructed is extremely important to its durability and its properties, especially in withstanding bending and pressure. According to Meier (1961) these properties are so difficult to

calculate on the basis of construction and material that these cables are characterized merely by their strength in the longitudinal direction (tensile strength). The way in which the tendon is constructed is considerably more complicated and permits even less a calculation of its properties against the combination of traction, bending and pressure.

In conclusion it may be said that the Achilles tendon seems amply compensated for flexion-extension movements, but that its construction can only up to a certain, not known extent reduce the bending stress in supination-pronation movements of the calcaneus.

Composition and Ultrastructure of the Tendon

The most striking feature of tendons on ordinary light microscopy is their well-oriented construction of practically homogeneous, slightly undulating tendon fibres, separated by interspaces which contain fibrocytes, in places loose, vascular connective tissue.

The fibrocytes – by some authors called fibroblasts – have elongated nuclei with a high chromatin content and a stellate cytoplasm. The fibrocytes are not, or at least only to a very slight extent, capable of fibrillogenesis. On the other hand, this cell is considered to be the source of int. al. mucopolysaccharides (Schwartz 1961, Dorfman 1962, Schiller 1966). In the course of tendon regeneration the fibrillogenesis occurs in and around fibroblasts of the granulation tissue that has invaded from the peritendineum (e.g. Buck 1953, Davidsson 1956, Schmitt et al. 1970 a). When regeneration has been completed, the fibroblasts become fibrocytes. Ingelmark (1945) demonstrated that the cell count per cubic mm in man is rapidly decreasing until growth is completed.

In the light microscope the tendon fibre is the basic element. It is characteristic of the tendon fibre that it is separated from the other tendon fibres by fibrocytes (Elliott 1965 a). The fibre is composed of numerous fibrils which may be said to be the electron microscopic basic element, as it is not possible to visualize a smaller unit by preparation of the fibres (Borysko 1963). The fibril is composed of subfibrils (tropocollagen) demonstrable particularly during fibrillogenesis, i.e. before the subfibrils join to make fibrils. Tropocollagen is the collagen molecule which is made up of 3 left-coiled, not entirely identical polypeptide chains. These chains are wound into a right-coiled unit (Brown & Menkart 1963). The tropocollagen has a diameter of 14 Å and a length of 2900–3000 Å (Hall 1961, Grassmann 1965). Its molecular weight is 300,000–360,000 (Wynston & Nagamani 1965). Despite attempts, it has not yet proved possible to set up an accurate model for the sequence of the amino acids in the polypeptide chains (Hanning & Nordvig 1965, Herring 1968). Yet, the content of hydroxyproline is so constant that multiplying the hydroxyproline concentration by a factor 7.46 gives a

reliable expression of the collagen content (int. al. Neumann & Logan 1950). Tropocollagen has no striation (Rhodin 1967), but the sequence of the amino acids is believed to condition the striation in the fibril (Wassermann 1954). The striation in the fibril is repeated at a periodicity of 6–700 Å (Schwartz 1961, Herring 1968). The explanation of the periodicity is that the tropocollagen is arranged in layers with a parallel shift in the range 1/5 or 1/7 (Hodge et al. 1965). Cox & Grant (1969) have suggested a model of the construction of the collagen fibril. According to this model, the tropocollagen is made up of 5 bonding zones and 4 bonding-free zones, each bonding zone opposing a bonding zone in the adjoining molecule. The subfibrils are arranged side by side with an accidental parallel shifting of the molecules. In the model the molecules are not arranged end-to-end, so that the intermolecular cross-links must be assumed to be of the utmost importance to mechanical as well as chemical stability (cf. p. 43).

The formation of the polypeptide chains, perhaps also of the tropocollagen, takes place in the fibroblast, whereas the fusion to fibrils takes place outside the cell (Wassermann 1954, Jackson 1957, Schwartz 1961). According to Rudall (1965) and Arendt (1966) the fibrils are thicker the farther from the cell they are situated. The diameter is reported to be from 100–1300 Å, usually between 200 and 600 Å. Wood & Keech (1960) and Wood (1960), in their *in vitro* experiments, found the thickness and number of fibrils to depend upon the temperature, pH, and the presence of various mucopolysaccharides. In their opinion this applied also *in vivo*. As the mechanical properties may change with fibril thickness (Chvapil & Hruza 1962, Mitton & Morgan 1960), it must be expected that to some extent the mechanical properties have been determined already at the formation of the fibril. In a comprehensive survey on the formation of collagen, Chvapil & Hurych (1968) reported that int. al. the supply of iron and ascorbic acid to the organism affects the collagen quantitatively as well as qualitatively.

Gross (1964) and Rudall (1965) believed that the decomposition of connective tissue takes place in the way that mucopolysaccharide is dissolved before collagenolytic enzymes can exert their action. According to Gerber et al. (1960) and Harkness (1961) the turnover for collagen in rat tendons was about 100 days, whereas Neuberger (1955) reported the half-life to be 500 days and Strehler (1969) 1000 days. This very slow turnover corresponds to the view that the tendon cells are incapable of fibrillogenesis, at least after growth has been completed.

Intra- as well as inter-molecularly the collagen is reinforced by cross-links, partly hydrogen bonds and saline bonds, and partly a number of different co-valent bonds (Herring 1968, Cooper & Russel 1969, Jackson & Steven 1969). Furthermore, there are cross-links between

collagen and other substances, e.g. mucopolysaccharides. The solubility depends upon these bonds, the co-valent ones making the collagen insoluble (Piez 1969 a). These firm bindings occur gradually and may be taken to some extent as indicating the age of the collagen, but already a few days after collagen formation there will be insoluble collagen present (Gross 1964). The firmness of the bindings also manifests itself in shrinking and relaxation induced by changes in temperature and by chemical agents. Chvapil (1967) stressed that changes in these physical-chemical properties were not tantamount to changes in the mechanical properties.

So far, mainly the formed elements, viz. the cell and the fibril, have been described. In addition, there is the ground substance which previously was considered just a kind of lubricant. Apart from water and electrolytes, the ground substance contains protein-polysaccharide complexes and glycoproteins. The quantity of mucopolysaccharide is about 0.5% of the tendon weight (Partington & Wood 1963). Balian & Bowes (1967) reported that a large portion of it was situated in the peritendineum. The mucopolysaccharides consist chiefly of chondroitin sulphate B and C, mostly the latter (Hall 1961). Meyer & Rapport (1951) stated that hyaluronic acid was not present, whereas Partington & Wood (1963) and Dahmen (1966) found small quantities in tendon tissue. Meyer et al. (1956) and Mathews (1967) reported that the tendon contained mostly chondroitin sulphate B. According to Cessi & Bernardi (1965) the protein-mucopolysaccharide complex formed a beehive-like structure with central cores of protein. Partridge et al. (1965), studying the protein part in more detail, were able to isolate it partially. Mathews (1967) believed that the protein part contained a 3700 Å long chain on which about 60 chondroitin molecules were attached as side chains. Thus, the side chains were supposed to face the collagen. The bindings between these columns of protein-mucopolysaccharide and collagen appear to be very firm, judging by the consumption of energy on liberation of the mucoproteins (Chvapil & Zahradnik 1959, Schubert 1966). There is no definite model of the topographic distribution of collagen and protein mucopolysaccharide. The site of the glycoproteins too remains to be elucidated.

Fry et al. (1964) thought that the collagen was the structural background of the tendon strength, whereas others believe that the protein-mucopolysaccharide complexes are decisive (Jackson 1953, Harkness & Harkness 1959, Chvapil & Hruza 1962, Partington & Wood 1963, Mathews 1967). In experimental lathyrism, Levene (1967) found that the collagen was in most respect as it is normally, but its mechanical strength was slight. He felt that this had to be attributed to the cross-links, not primarily to the mucopolysaccharides. Plenk & Zweymueller (1971) observed that the mechanical strength in the epiphysis is also reduced in

lathyrism. In the opinion of Robert & Robert (1970) the glycoproteins are more important than the mucopolysaccharides in the composition and stability of the collagen.

The turnover of the mucopolysaccharides is about 10 days (Boström 1969), while that of hyaluronic acid is about one day (Hall 1961). Thus, any disturbance in the formation of these substances ought to be rapidly reflected in the tendon. The water- and electrolyte-binding ability of the tendon is conditioned by its content of organic substances, partly collagen, partly protein-mucopolysaccharide and glyco-protein complexes (Laurent et al. 1969). Changes in the structure and concentration of these substances affects the properties of the tendon. The associated changes in water and electrolytes presumably do not *per se* affect the properties of the tendon, but reflect changes in the organic substances.

The water and electrolyte content will be described in more detail in Chapter 9.

According to what has been stated above, it must be assumed that both the environment (ground substance) in which the collagen forms and the environment in which it currently exists determine its properties. Isolated quantitative determination of collagen will not necessarily be correlated to the mechanical strength of the tendon.

Mechanical Properties of the Tendon

The mechanical properties of the tendon have been reviewed in comprehensive monographs, int. al. by Harkness (1961), Elliott (1965a) and Viidik (1966, 1968 a, and 1969 b).

The most striking properties are its great tensile strength, its relative inextensibility, its elasticity, plasticity, and viscosity. Lastly, the flexibility is an extremely important character which has been discussed further on p. 37.

The length-tension diagram of the tendon (Fig. 2) is s-shaped, with a flat toe part, convex towards the length axis. This flat part – corresponding to a 2–3% lengthening (Rollhäuser 1951) – is due to straigtening of the wavy structure as seen on simultaneous microscopy (Rigby et al. 1959, Viidik & Ekholm 1968). The next phase, marked on Fig. 2, is due according to Rollhäuser (1951) to expression of water from the collagen fibrils. Then follows a steep, almost linear segment of the curve. Viidik & Ekholm (1968), measuring by electron microscopy the periodicity in collagen fibrils strained at the time of fixation, found a significant increase in the length of the periods on a straining corresponding to the steep part of the curve. It must be assumed that the deformation during this phase occurs essentially in the molecular collagen structure.

The linear part is followed by a bending of the curve, possibly a

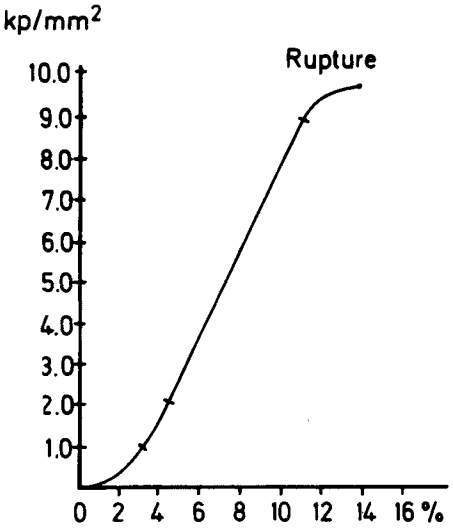

Fig. 2. Length-tension curve for a tendon from a 44-year-old man. Elongation given in per cent. The sub-division of the curve into 4 phases is described on p. 42 (after Rollhäuser 1951).

decrease in tension. The transition is described by Rigby et al. (1959) as a safe limit, repeated loads beyond this limit entailing an essentially lower tensile strength and a greater elongation, i.e. a flatter curve. This is caused by irreversible, structural changes in the tendon, presumably rupture of the cross-links between the collagen molecules; rupture of the peptide bindings of the collagen molecule would have required an approx. 30 times greater load (Highberger 1947).

Elliott (1965a) found the tensile strength of the tendon to be reported from 0.6 to 21 kp/mm^2, generally between 5 and 10 kp/mm^2. The tensile strength per mm^2 wet tendon was found by Cronkite (1935/36) to be 6–12 kp and by Gratz & Blackberg (1935) to be 7 kp. Measurements of the cross-sectional area are inaccurate. Instead, several authors have used dry (Elliott & Crawford 1965) or fresh (Viidik 1967) tendon weight per length unit, which gives more reproducible results. Most authors quote Stucke (1950) who found the tensile strength of the human Achilles tendon to be about 400 kp, corresponding to 4.7 kp/mm^2. As early as 1846 Valenthin and in 1847 Wertheim reported similar values, and according to Karasev (1969) the Achilles tendon could withstand loads of 470 kp, corresponding to a tensile strength of 7.8 kp/mm^2. On the basis of rupture experiments on parts of human Achilles tendons, Yamada (1970) calculated that the total tensile strength was only 200 kp. Offhand, these values seem very high, but they must be compared to the force of the appurtenant muscle (cf. p. 57).

Just as important as the tensile strength is the ultimate strain limit. The elongation has been stated to range from 7 to 15% of the resting length (Stucke 1950, Rollhäuser 1951, Viidik 1968 a, Yamada 1970). In the case of the Achilles tendon it is 1–2 cm, but as the muscle increases much

more in length than the tendon upon the same loading, the strain (elongation per unit resting length) of the total muscle-tendon unit will be considerably greater (cf. p. 56).

The tendon is not truly elastic, as it is unable to resume its original shape completely and instantaneously. In the toe part of the length-tension diagram there occurs at the first load a minor, permanent elongation which is not, at least not *in vitro,* abolished by removing the load (Rigby et al. 1959, Abrahams 1967). Rigby et al. (1959) call this permanent lengthening a "conditioning". It is of plastic nature, being independent of time, unlike the viscosity. The viscosity manifests itself in after-stretching and after-shrinkage, or in other words by a rapid loading causing less instantaneous elongation than slow loading. If the loading is maintained unchanged, this difference in lengthening will approach zero in an asymptotic manner (Sonnerup 1969). When disregarding the initial plastic changes, the tendon will, on loading to the safe limit, act as a purely visco-elastic material. Not until thereafter do the plastic properties start predominating. There have been no experiments to show whether these plastic deformations, irreversible *in vitro,* are also irreversible *in vivo.*

A model on the basis of elastic, plastic, and viscous properties has been suggested by Viidik (1968 c) and by Frisen et al. (1969 a and b), but as yet there have been no results concerning differences in the various fractions between tendons from animals at different ages or from different parts of the body.

Owing to the above-mentioned properties it is evident that the elongation is less marked on rapid than on slow loading, i.e. the length-tension curve is steeper. On the other hand, it is not evident whether the rupture limit – the end-point of the curve – follows the same rule. If the rupture always occurs at the same stress level, this limit may be reached at a less marked elongation when the loading is rapid. Reversely, a fixed elongation at rupture may require a greater load when the loading is rapid. Stucke (1950) whose experiments on human Achilles tendons were of static nature, believed that a dynamic loading had to be one-quarter greater than a static one. Chvapil & Hruza (1962) found the tensile strength in rat tail tendons to increase gradually with increasing rate of loading – measured in g/min. – in order to decrease a bit at the highest rates. A similar increase was found for the elongation at the rupture limit. According to Tipton et al. (1967 b) the tensile strength in the medial ligaments of the rat knee was independent of the straining rate within the range studied.

Welsh et al. (1971) observed a lower tensile strength in rabbit Achilles tendons on slow traction (1.3 cm/min) than on rapid traction (130 cm/min), but the area below the length-tension curve was significantly larger at slow traction, corresponding to the fact that greater work

performance was required to induce rupture at slow traction. Moreover, the same authors stated that the effort needed to attain the rupture limit was greater, but the tensile strength lower, the longer the tendon was.

General changes of the pH within the physiological range do not have any influence upon the shrinkage temperature which is somewhat over 60°C and which is a melting phenomenon. However, major local pH changes towards the acid side may be imagined to reduce the temperature limit for melting to the physiological temperature range (Nordschow 1966). There have been no studies to show whether this may influence the stability of the Achilles tendon.

The influence of temperature within the physiological range upon tendon tissue has not been finally clarified, although it would appear as if an increase in temperature within this range may cause increased elongation and a lowered tensile strength at rupture limit. According to Gersten (1955) heating of tendon tissue in Ringer's solution and by ultrasonic treatment exerted the same effect. With increasing temperature there was a decreasing inclination of the length-tension curve, simultaneously with a fall of the elastic limit. The elastic limit was used by Gersten to describe the point at which the length-tension curve deflects and thus corresponds to the safe limit (p. 43). At 40°C the tension at the safe limit had fallen to half the value at 30°C. It must be emphasized that the investigation was carried out on cold-blooded animals (frogs). Ellis (1970) investigated how temperatures from 22–55°C influenced the length-tension diagram of rabbit extensor tendons. Not until a temperature exceeding 44°C did changes occur. The ultimate strain was not investigated. In rat tail tendons, Rigby et al. (1959) found, at a temperature exceeding 37°C, that the strain at repeated loadings increased as the safe limit simultaneously became lower. Rigby (1964) observed that very long-lasting loading could reduce the shrinkage temperature, but not down to the physiological range. Rigby too did not study the ultimate strain.

Lehmann et al. (1970) investigated the influence of temperature upon the extensibility of rat tail tendons. They report that at the same loading the elongation was greater at 45°C than at 25°C and that the difference in elongation increased with increasing loading. The same team (Warren et al. 1971) demonstrated at 25°C that the ultimate strain was 15.5 %. At 39°C the tendons were loaded until a 2.6 % strain by about one-quarter of the loading which caused rupture at 25°C. This procedure lasted for about 10 minutes. After a 10-minute "rest" the tendons were loaded until rupture. Now, the tensile strength had dropped to about one-third of that at 25°C, whereas the elongation had fallen to one-fifth. It is amazing that a long-lasting, not particularly violent loading has such a marked effect at temperatures within the physiological range. The experiments have not

been confirmed on other tendons or other animals, and as emphasized by Piez (1969 b) it must be borne in mind that rat tail tendons differ essentially from other tendons in not being loaded in the same way. In addition, the normal temperature of rat tails is only about 25°C. On this background it is not possible to draw any parallel to other animal tendons, let alone human tendons.

The changes which may occur in connection with ageing, training, and inactivity will be described in Chapter 9.

CHAPTER 7

THE BONE

The bone is just as important a link as the other two members of the bone-tendon-muscle-bone chain. The outer shape of the bone determines the mobility of the joints and the length of the lever arm, its inner structure the strength to withstand pressure, traction, and bending forces.

Its outer shape is in the main genetically conditioned, but severe muscle imbalance may alter this shape, as may be seen e.g. in patients with myelomeningocele. The change in function which occurs in the calcaneus when rats are transformed into bipedal animals may also affect the shape of the bone. In such animals the calcaneus increases in length (Goff & Landmesser 1957).

At the tendon insertion or muscle attachment the bone is often rough, possibly with spicules, presumed to lend the bone-tendon junction a larger surface and thereby greater strength. This rough skeletal surface consists of a layer of compact bone covering cancellous bone whose structure is partly determined by the direction of the most common stress lines. According to Viidik (1969 a) the compact bone at the insertion of the Achilles tendon in rats is thin, but this is the only mention of variations in bone structure at the insertion of the Achilles tendon that I have been able to find in the literature.

The bone is built up over a lattice of collagen fibrils exactly like the collagen found in other sites (Engfeldt 1969). However, the turnover of the bone collagen is considerably more rapid in bones than in tendons (Neuberger & Slack 1953). Within this lattice of collagen there are cells of different types, mucopolysaccharides, and Ca phosphates of varying composition.

The mechanical properties of the bone may be described int. al. by a length-tension diagram which is steeper than that of the tendon (Fig. 2) and also lacks the toe part found on the length-tension diagram of the tendon.

For long bones (femur) the strain at the rupture limit is said to be 1 %, whereas the tensile strength is about 10 kp/mm^2 (Yamada 1970). Lindahl

& Lindgren (1967 b), also studying the femur, reported an ultimate strain of 2 % and a tensile strength of 14 kp/mm^2. These values are highly varying, both within the same bone and between various bones (Sedlin 1965, Evans 1957, Yamada 1970). Cancellous bone, such as the vertebral bodies, has a strain limit of 0.8 % which is reached at a loading of 0.4 kp/mm^2 (Yamada 1970). The posterior part of the calcaneus cannot be suspended in a material testing machine, but has to be studied in association with tendons as described in Chapter 4. In such experiments the tendon has usually proved stronger than the bone, but in rupture experiments on ligaments (Palmer 1938, Viidik 1968 b, Tipton et al. 1967 b) tears often occur at the bone-ligament junction or in the ligamentous substance.

The alteration in straining which arises in a bone on loading (1–2 %) is so slight that it is of no consequence in relation to the straining in the muscle and tendon. Apart from this, the deformation decreases with increasing rate of straining (Sedlin 1965), due to considerable viscous properties.

Like the tensile strength, the strength against pressure and bending varies for the individual bones and for the individual parts of each bone (Sedlin & Hirsch 1966). Lastly – and perhaps mainly in relation to tendon rupture – there are appreciable variations depending on age, diet, and state of training (cf. Chapter 9).

CHAPTER 8

THE MUSCLE

Anatomy

The muscle is made up of muscle fibres, mutually separated by endomysium, collected in bundles which are surrounded by perimysium. Groups of these bundles – fasciculi – make up the muscle which is separated from its surroundings by the muscle fascia – epimysium. At both ends of the muscle perimysium as well as endomysium gradually merge into a strong fibrous structure – a tendon aponeurosis – which converges to form the tendon. At the origin of the muscle the tendon is as a rule very short, unlike the findings at its insertion. It seems expedient to place the heavy motor closer to the joint axis, thus reducing the momentum.

Previously, it was believed that, at the junction of the soft muscle fibre and the relatively stiff tendon fibrils, there was direct fibril continuity. However, Schwarzacher (1960) and Schippel & Reissig (1968), in electron microscopic studies, have shown that this is not so. At each end of the muscle fibre (= muscle cell) there are crypts of a depth corresponding to the length of up to 6 sarcomeres (Gelber et al. 1960). The collagen fibrils enter these crypts where they join the outer layer of sarcolemma, whereas the myofibrils attach to the inner layer.

Hanak & Böck (1971), in their electron microscopic studies, found the collagen fibrils to be fixed in a network of microfibrils issuing from the outer layer of sarcolemma. Only a few collagen fibrils join the lateral surfaces of the muscle fibres, and at this site it has indeed not been possible to demonstrate any connection between the myofibrils and the inner aspect of the cell membrane.

The outer shape of the muscle is highly varying, depending upon the distribution of tendon aponeurosis and the direction of the muscle fibres in relation to the long axis. In a fusiform muscle the fibres are parallel to the axis, whereas in a penniform muscle the fibres form a major or minor angle on the long axis.

Muscle length depends upon the range of movement permitted by the adjacent joints, the fascicular length being about twice that of the range (Weber 1851). In the case of the penniform muscles fascicular length is somewhat less, being dependent upon the angulation in relation to the long axis of the muscle.

Crawford (1954) reported that the fusiform anterior tibialis muscle in rabbits altered its range of movement on transposition and that simultaneously the length of its belly altered according to the above pattern. In a subsequent study in collaboration with Alder and Edwards (Alder et al. 1958) he followed the same muscle throughout the growth period. The range of movement in adult rabbits was found to make up a smaller part of the muscle length than in newborn rabbits, perhaps because the possibility of moving *in utero* had been limited. Hypertrophy induced by the removal of a synergist did not alter the muscle length in the anterior tibialis (Crawford 1961). The length of fasciculi or fibres was not measured.

The penniform muscles have been studied in more detail by Benninghoff & Rollhäuser (1952), Wendt (1953), and Rollhäuser & Wendt (1955), partly by muscle dissection in autopsy series and partly by training experiments on cats. The cross section of the muscle fibres proved to be oval, the longer axis being parallel to the long axis of the muscle. On training, the transverse axis of the muscle fibres increased. Thereby the fibre angle grew larger, and at the same time fascicular length diminished so that the lifting height was still the same. The largest fibre angle in the extended muscle was 30°, corresponding to a fibre angle of 90° at a 50 % shortening. Thus, a larger fibre angle would have prevented the last part of the shortening on contraction. It was emphasized by these authors that the increase in the fibre angle did afford an increase in muscle mass, but there was not an entirely corresponding increase in force, the force resultant in the long axis of the muscle decreasing relatively. Reversely, atrophy would not cause such a marked loss of force as would correspond to the loss of muscle mass. Furthermore, they emphasized that the increasing angulation will make for decreasing force with increasing shortening beyond the loss of force that may be observed in the length-tension diagram for the individual muscle fibres. Fick (1910) pointed out that in the pennate muscles there are, depending upon the fibre angle, very great lateral forces which prevent the juxta-muscular part of the tendon from shifting sideways.

The triceps surae is a multipennate muscle, consisting of 3 components, the soleus which passes 2 joints – the talocrural and subtalar joints – and the 2 bellies of the gastrocnemius which also pass the knee joint. Owing to the size and the situation in relation to the ankle axis the triceps surae is able to contribute about 9/10 of the plantar flexion force (Fick 1911).

The soleus is reported to be the strongest, thereafter the medial gastrocnemius, the weakest one being the lateral gastrocnemius (Lanz & Wachsmuth 1938). The triceps surae is of approximately the same relative importance in supination of the foot.

The soleus belly contains "red" muscle fibres (C fibres) which differ from "pale" fibres in their innervation (Close 1965) and biochemical structure, especially in a high content of oxidative enzymes (Wilkie 1966). The colour is said to be due to a high content of myoglobin (Sissons 1964). The gastrocnemius bellies contain a mixture of A, B, and C fibres (Schmalbruch 1971), A fibres being "pale" and B fibres occupying an intermediate position. The "red" fibres are reported to be slow (Buchthal & Schmalbruch 1970), having mainly a static function, whereas the "pale" ones are concerned mainly with rapid, phasic movements (Woodbury & Ruch 1960). The capillary supply to the soleus is stated to be 2–3 times that to the gastrocnemius (Schmalbruch 1971).

The motor nerves to the muscle are mainly thick nerve fibres each one coming from its appurtenant anterior-horn cell. In the muscle the nerve fibre splits up and supplies a number of muscle end-plates, a few in muscles with delicately graded movements and several thousands in e.g. leg muscles. These jointly innervated muscle fibres constitute a motor unit in which the fibres are indeed separated by other fibres, but all within a fairly narrow area (Buchthal et al. 1957, Sissons 1964). In addition, there are thin motor nerve fibres, gamma fibres, which innervate the motor part of the muscle spindle and which, together with the sensory fibres from the muscle spindle, make up a proprioceptive organ as well as a servo-mechanism (Åstrand & Rodahl 1970).

Ultrastructure of the Muscles

The basic element of the muscle belly is, as already mentioned, the muscle fibre which is collected in bundles – fasciculi – whose size and situation characterize the macroscopic appearances and to some extent function. The muscle fibre is a large cell having a diameter of (10–) 50–100 μ. Its length in the sartorius muscle may be up to 45 cm, but in the triceps surae it is about 5 cm, somewhat greater in the gastrocnemii than in the soleus (Weber 1851). Studying the frog sartorius, Lindhardt (1931) found that as a rule the fibre length somewhat exceeded half the fascicular length. In the abductor muscle of the little finger Sailer (1950) found up to 10 fibres, serially arranged, i.e. with several small intermediate tendons.

The muscle fibre is invested in a cell membrane which constitutes the inner part of the enveloping sarcolemma. It contains nuclei in a very large number depending int. al. upon the function of the muscle fibre. The "red" fibres contain about 3 times as many nuclei as the "pale" ones. The nuclei of the mature muscle cell are peripheral. On the outside of the cell

there is a muscle end-plate with an afferent nerve fibre. On long fibres there may be more than one muscle end-plate.

In addition to the nuclei, the fibre contains densely arranged myofibrils surrounded by sarcoplasm. Owing to its content of myoglobin, the sarcoplasm influences the colour of the muscle, which grows redder with increasing content. Between the fibrils there are mitochondria and vacuoles holding glycogen and fat. There have also been reports of the sarcoplasmic reticulum, believed to be concerned with the spread of impulse from the muscle end-plate (Wilkie 1966).

The myofibril is striated, easily recognized in the ordinary light microscope. The following description is based chiefly upon Hanson & Lowy's review from 1965. The striations are repeated at intervals of 1.65 – 3.65 μ, depending upon the degree of straining of the fibril. The myofibrils are situated close together, the striation proceeding all through the fibre. The myofibril proper is about 2–3 μ thick. On cross section (in the electron microscope) it is seen to contain filaments of two thicknesses, in a pattern so that each of the thick filaments (myosin) is surrounded by 6 thinner filaments (actin) each of which is situated an equal distance from 3 thick filaments. These filaments do not extend throughout the myofibril, ending within each sarcomere. Cross sections at different levels may show exclusively thin or exclusively thick filaments. This is the structure which causes the striation. On the basis of the different refraction a distinction is made between A substance and I substance. In the middle of I substance there is the Z line which extends across the fibre. From both sides of the Z line thin filaments extend through the I band to interdigitate to a major or minor extent with the thick filaments of the A band. When the muscle fibre lengthens the A band is seen to remain unchanged in length, whereas the I band lengthens. It has not been possible to demonstrate any change in the length of the filaments with changes in fibre length. This description forms the basis of the sliding filament theory.

The thick and thin filaments are built up of longitudinally arranged myosin and actin molecules respectively, both of which have a periodicity of around 400 Å. This may entail that invariably two identical groups are face to face, but the nature of the bonds which retain the actin and myosin filaments opposite each other has not been elucidated. The nature of the process which causes mutual displacement of the filaments is also unknown. It seems as if the number of sites of contact between the filaments bear a direct relation to the tension that the muscle is able to develop. The energy source of these processes is ATP, adenosine triphosphate, which appears to enter directly into the contraction process. Within the muscle there are a number of energy sources which may contribute to the synthesis of ATP. Roughly simplified, it may be said

that the presence of ATP renders possible a shift of the filaments and that splitting of ATP causes a shift of actin in between the myosin filaments. The degradation product locks the position, which is unlocked by the resynthesis of ATP. On exhaustion of a muscle it may be imagined that ATP synthesis gradually comes to a standstill, resulting in a state in which the position is partly maintained, a state which must make the muscle more rigid.

When a muscle fibre hypertrophies, its sarcoplasm gets more abundant. Denny-Brown (1961) also found that the number of myofibrils increased, if training was intense, but did not investigate whether they decreased in number during inactivity. He interpreted the amount of sarcoplasm as a sign of metabolic reserves, i.e. determining endurance, whereas the number of myofibrils was decisive to the magnitude of the maximum contraction force.

Mechanical Reaction of the Muscle

Contraction of the muscle is an alteration of the mechanical equilibrium of the muscle fibres. The normal stimulus for the contraction process is the transmission of an impulse through the nerve fibre to the muscle end-plate, where the impulse gives rise to a breakdown of the membrane potential. The local breakdown is transmitted to the entire fibre at such a high rate (5 m/sec) in relation to the contraction process that this process takes place, in the triceps surae, almost simultaneously throughout the entire fibre. Some time has to elaspe until *status quo* has been re-established in the end plate. This period is called the refractory period. The latter, however, is appreciably shorter than the time elapsing, even at maximum contraction, from one impulse hits the fibre until the next one appears. The single contraction is characterized by an increase in tension which is rather more rapid than the decrease in tension. The total duration is 30–100 msec. The sliding which occurs in the sarcomere takes some time and is not completed within the period of a single contraction. If a stimulus is repeated before the previous one has been completed, the tension obtained is greater than that in a single contraction. With increasing frequency the tension increases and gradually gets even. At a frequency of 50/sec the maximum tension for mammalian muscles has been attained. Often, a lower frequency is sufficient, depending upon the individual, muscle group, kind of fibre (red or pale), and temperature. This maximum tension obtained by electric stimulation of the nerve is reported to be of the same magnitude as the maximum voluntary contraction (Merton 1954, Bigland & Lippold 1954, and Buchtal & Schmalbruch 1970). According to Ikai et al. (1967), however, this could only apply in extraordinary situations, e.g. under hypnosis.

Both the individual fibre and the fibres of a motor unit follow the "all

or none" law, if the stimulation is via the nerve, but as already mentioned the resulting tension may be increased by raising the frequency. By increasing the number of working motor units (recruitment), there is furthermore a possibility of grading, and as these motor units work asynchronously, there is also a possibility that even a weak contraction may be smooth, not jerky. In fatigue there may be synchronization of motor units (Bigland & Lippold 1954).

At an unaltered degree of stimulation the tension in the muscle depends upon its length. This dependence has been illustrated in the length-tension diagram (Fig. 3) for the isometrically contracted and the relaxed muscle. On this figure the extreme limits of mobility *in situ* are marked. Alder et al. (1958) found, in newborn rabbits, a considerable shift to the left of the lower limit, whereas the upper limit was constant. In adult animals the fairly horizontal part of the curve representing the contracted muscle was within these limits.

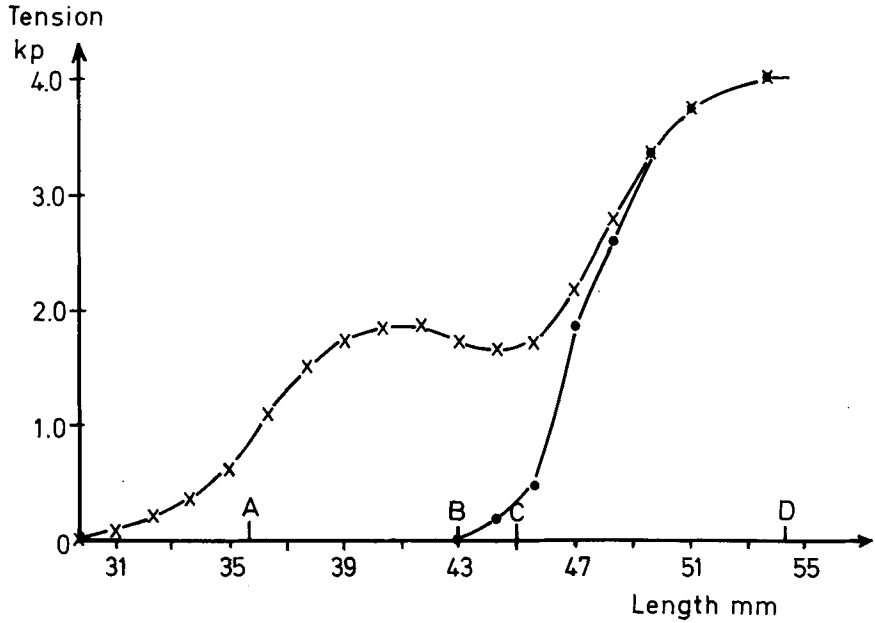

Fig. 3. Length-tension diagram for the triceps surae muscle from a wild rat (♀ 265 g). X–X–X– : The curve for isometric muscle force at maximum stimulation of the sciatic nerve.
. – . – . – : The passive tension curve.
The length represents the length of the muscle + the Achilles tendon. A indicates the length at maximum flexion in the knee, plantar flexion in the ankle. C indicates the length at extension in the knee and dorsiflexion in the ankle. Thus A – C is the greatest possible range of movement in the intact organism. B: Elastic equilibrium length. At D visible rupture of the muscle.

The elastic equilibrium length for the relaxed muscle – the greatest length the muscle can have without the use of force – has been reported to be 15–30 % below the maximum possible length (Lindhard 1931, Åstrand & Rodahl 1970). This length corresponds approximately to that area of the curve which shows the most marked difference in tension between the relaxed and the contracted muscle (Fig. 3).

If the length increases during a contraction (eccentric contraction), the obtained tension will be greater than that corresponding to isometric contraction at the new length. Simultaneous shortening (concentric contraction) causes less tension. After the lapse of a short time the tension will fall, or rise respectively, to the level of isometric contraction, both because of the viscous properties of the muscle. Buchthal (1942) established that this viscosity was 4 times greater for the contracted than for the resting muscle and that viscosity increased with increasing elongation. The rate at which the change in length takes place is decisive to the magnitude of the tension, as illustrated in Fig. 4 (after Asmussen et al. 1965). The ordinate is the force in per cent of the maximum isometric force by which flexion of the arm can be performed, and the abscissa gives

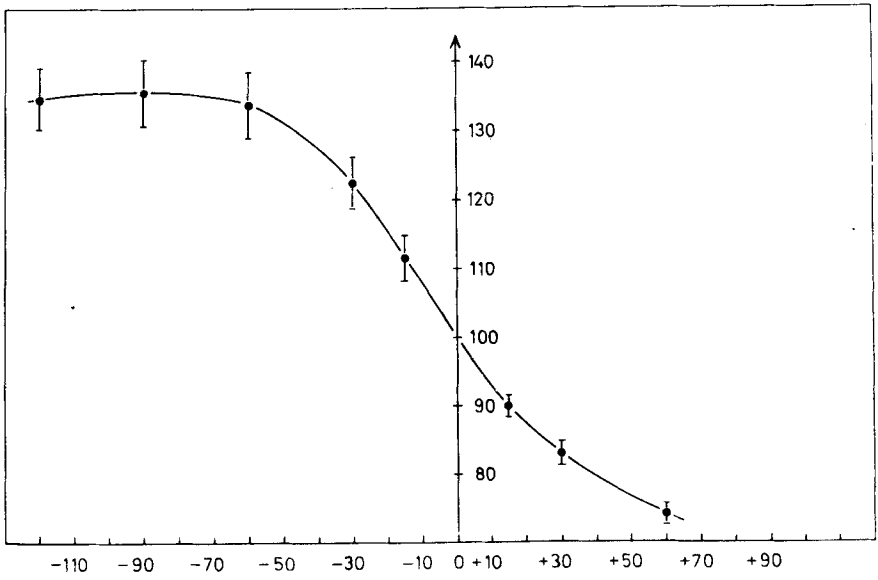

Fig. 4. Force-velocity curve for elbow flexion in 6 men.
Ordinate: Force in % of the maximum isometric force.
Abscissa: Velocity in % of arm length per sec.
Values calculated from the midposition of the total movement (after Asmussen et al. 1965).

the velocity as per cent of the arm length per sec. Asmussen et al. found a 35 % increase of tension in eccentric contraction up to a certain velocity. Thereafter, the curve flattened. In animal experiments, however, a considerably greater increase seems to be obtainable. Asmussen (1968) showed in his Fig. 15 an almost 100 % increase during eccentric contraction in the frog gastrocnemius. Katz (1939) reported an increase of 1.9 x the maximum isometric force. As a possible explanation of this great difference between voluntary human experiments and animal experiments it may be imagined that in man the neurotendinous spindles elicit an extension reflex which inhibits contraction.

If the curve on Fig. 4 had continued to the right, it would have ended in tension O (Abbott et al. 1952) at a contraction velocity which has been reported in rather different ways. For human muscles Inman & Ralston (1954) (kineplasties) reported a rate of 143 cm/sec for the pectoralis muscle and 55 cm/sec for the triceps brachii. For the rat diaphragm Ritchie (1954) found a rate of 20 cm/sec corresponding to 10 x the muscle length per sec. Pertuzon & Bouisset (1971) and Pertuzon (1972) reported a contraction rate of 80 cm/sec in the human biceps brachii upon minimum loading.

The length at which the muscle ruptures has been reported by Triebel (1902) to be 200 %, by Yamada (1970) to be 260 %, both measured on the relaxed muscle. Casella (1950) stated that relaxed frog muscle fibres lengthened 339 % before rupturing, but did not mention lengthening at the rupture limit for whole muscles or for contracted muscles.

The tensile strength post mortem was found by Yamada (1970) to be 2–4 kp/cm^2, less for the gastrocnemius than for e.g. the rectus abdominis and appreciably lower than the 8 kp/cm^2 reported by Triebel in 1902. According to Casella's (1950) calculations, the rupture limit for frog muscle fibres is 3–4 kp/cm^2. It has not been elucidated whether it is the myofibrils, sarcolemma, or the connective tissue of the muscle which determines its properties at the rupture limit.

Muscle Force

The isometric muscle force for man is stated by Ikai & Fukunaga (1968) to be 6.3 kp/cm^2 (4–8 kp/cm^2) and by Buchthal & Schmalbruch (1970) to be 5–6 kp/cm^2. Casella found the force of the entire frog muscle (1.5 kp/cm^2) to be less than that of the muscle fibres (3.3 kp/cm^2).

In measuring human muscle force it is generally necessary to abstain from measuring individual muscles. Instead, the action of a muscle group upon a joint is determined, often as the isometric force at a given position of the joint. The force is then stated in kp x cm. There is a good correlation between the isometric force and the dynamic force (Asmussen et al. 1965, Carlson 1970).

The standard deviation of the results of measurements from the same subject is in the order of ± 20 % (Asmussen et al. 1959). A number of different isometric muscle functions have been investigated in a large material of normal subjects (Asmussen & Heebøll-Nielsen 1961). In general, women – after correction for the difference in height – had only 80 % of the muscle force found in the men. The force increased up to the age of 30 in order to fall slowly thereafter (cf. Fig. 5, p. 65), incidentally more slowly for arm muscles than for leg muscles. The most relevant values from this study were the torque for the maximum isometric, intended plantar flexion of the ankle. In a 35-year-old man, 182 cm tall, it was 1280 kp x cm. The corresponding value for extension of the knee was about 2000 kp x cm. At a distance from the ankle axis to the Achilles tendon of 4.5 cm (cf. p. 36) the isometric force of the triceps surae may be calculated as 289 kp. As the variability is marked, both for the same individual and for different individuals, an appreciably greater individual force can be attained.

Previously Reys (1915) found a muscle force of 560 kp in the triceps surae and Haxton (1944) found 438 kp. Herman & Bragin (1967) reported a torque in the ankle of 1150 kp x cm. These values are given without paying regard to the fact that the triceps surae contributes by only 9/10 to plantar flexion torque.

Voluntary Movements

The work output of which the muscles are capable depends upon the magnitude of the muscle force, the duration of the force, and the condition of the muscle immediately before the movement is started. The muscle requires some time for changing from relaxed to maximally contracted, but if it can start the movement from a maximally contracted state, either against resistance by the antagonists or from a preceding eccentric contraction, better performances can be achieved (Asmussen & Sørensen 1971).

A movement which involves several joints of a limb is performed with more efficiency, if it can be carried out by one pluriarticular muscle than if it is to be carried out by several muscles, each operating only one joint (Elftman 1939 b and 1941). It is also able to operate with less change in length, as the direction of movements in the joints passed by the muscle are usually opposed. The contraction velocity will then become slower and the force greater.

The function of biarticular muscles has been studied in greater detail by Molbech (1965) and Carlsöö & Molbech (1966). In guided movements these muscles may have a paradoxical effect, acting as extensors instead of flexors. The movement may be guided e.g. by gravity or by the inertia of the body. This means that in some situations one possesses greater muscle

force (extensor force + flexor force) to perform a movement. It must mean also that the function and innervation of the muscle are not strictly bound to a given direction of movement. Thereby the possibilities of erroneous innervation seem to be increased.

It is difficult to foretell the interplay between several muscles in a given movement. An electromyographic study is needed to clarify the innervation pattern, either by means of needle electrodes or surface electrodes. At the same time, other principles of measurement are used to clarify the relationship of the movement to EMG, e.g. measurement of forces by a force-plate or measurement of joint angles or angular acceleration by photography or by angle accelerometers.

Thus, Herman & Bragin (1967), in EMG studies, found that the soleus was fairly uniformly activated in different positions of the ankle joint, whereas the gastrocnemius muscles were more forcibly activated when the foot was plantar flexed than when it was dorsiflexed. They also found that the soleus muscle was most active in a slowly initiated, weak contraction, whereas gastrocnemius activity was at its peak in a sudden, vigorous contraction.

A number of authors have studied walking (Eberhardt & Inman 1951, Hirschberg & Nathanson 1952, Sheffield et al. 1956, Houtz & Walsh 1959, Liberson et al. 1962, Carlsöö & Norstrand 1968) and cycling (Houtz & Fischer 1959, Carlsöö & Molbech 1966, David et al. 1968). These studies established the innervation pattern and gave an impression of the contraction force since EMG, apart from demonstrating muscle action, is also applicable – with reserve – quantitatively. A striking finding in these studies was that the maximum function of the quadriceps femoris and the triceps surae was not observed simultaneously. According to Houtz & Fischer the innervation pattern in cycling was preserved, even though the speed and resistance were greatly increased.

Kamon (1971) did EMG studies of jumps from the squatting position. He found a fairly uniform pattern of muscle function in the various subjects. The rectus femoris muscle was active before heel lift-off and continued its action for some time after the heel had been lifted. The gastrocnemius (lateral belly) did not start until the heel had been lifted and continued its full activity until toe lift-off. In other words, extension of the knee started before lifting of the heel. On the accompanying sketches the knee seems to have been flexed about 60° when heel-lifting started. In sprint start (Payne & Blader 1971) and in high jump (Gombac 1971) the movement of the knee had almost ceased at the time of heel-lifting. This sequence would seem more effective, since otherwise the extension of the knee must force the gastrocnemii to isometric or even eccentric function, whereby the gastrocnemii would not contribute to the kinetic energy of the body.

An impression of the loads to which the Achilles tendon is exposed in the course of a movement requires a combination of anatomical data (distances between joint axis, centre of gravity for various parts of the body, and the length of the lever arms through which the muscles act) with data from the movement (joint angle and angular acceleration). In this way Elftman (1941) studied walking and running. He found a maximum tension in the Achilles tendon of 240 kp (walking) and 375 kp (running). On films of double somersaults, Grafe (1969) calculated the tension which arose in the Achilles tendon at take-off to the second somersault. The maximum load on both Achilles tendons amounted to 1070 kp. Grafe emphasized that unintended asymmetry would load one Achilles tendon by more than $\frac{1070}{2}$ kp. During take-off for a high jump Gombac (1971) found a vertical pressure of 250 kp against the ground, but the data are not so accurate that it could be said whether this pressure had been attained during the phase of deceleration or of acceleration; accordingly, it is not possible to calculate the tension in the Achilles tendon. Asmussen & Bøje (1945) found that leg push (the weight that a subject can lift by his knee in a sitting position with plantar flexed ankle) was up to 200 kp. If the ankle axis is considered the axis of this lever system, these 200 kp would have required a force of 540 kp in the triceps surae (cf. p. 36). According to Brouha (1960) the maximum increase in pressure against the ground in standing jump was 145 kp. As take-off was by both legs, the maximum tension in the triceps surae must have been almost 200 kp. In a similar experiment Davies & Rennie (1968) found 236 kp, corresponding to a maximum tension of 318 kp in each Achilles tendon.

By means of Chaffin's computerized static sagittal plane model of the human body (Chaffin 1969), Quigley & Chaffin (1971) studied the tension in the Achilles tendon during skiing. By maintaining a given style – erect or crouched – it was possible to attain theoretically a tension of 700 and 500 kp respectively at 40° slope. In practice they found values of up to 350 kp, because the skiers deviated from the planned style. No regard was paid to changes which might occur because of uneven ground, altered direction, resistance by the wind, or changes in the state of the snow.

All the above-mentioned loads on the Achilles tendon have been found in normally occurring movements produced by concentric, or perhaps almost isometric contractions. If at these times the muscles are exposed to external forces altering the contraction to the eccentric type, the tension in muscles and tendons will become even greater (cf. p. 55).

The role of psychological factors in motor performance may be seen e.g. from the fairly varied results obtained by an athlete under apparently identical conditions. One aspect of this phenomenon has been elucidated

by Roush (1951) and by Ikai & Steinhaus (1961) who found that voluntary, maximum isometric contraction could be increased by 15–20 % by hypnosis and rather less by shots and shouting. This effect is believed to be dependent on the activity in the reticular formation and the connected gamma system (Asmussen 1968, Åstrand & Rodahl 1970). The state of a person's mind, combined with the weightiness of his motive, must be considered of essential importance to his motor performance (Ikai & Steinhaus 1961, Jokl 1968).

Training

Apart from psychological factors, the state of training of course influences the motor performance.

Firstly, training may improve cardiopulmonary function and increase the density of capillaries in the muscles. This increases endurance.

In addition, training brings about an improvement of the innervation, of the local muscle (better distribution of the motor unit innervation) as well as of all muscles involved in the movement, in other words better timing. Metz (1970) found that training in performing one given, simple function caused better timing of the contraction, a reduction of the degree of activity judged by surface EMG, and lastly a better relaxation of the antagonists. All these changes take place *via* the central nervous system and are considered by many the most important effect of training (Rasch & Morehouse 1957, Hansen 1963).

Lastly, there are local changes in the muscle fibre which increase its volume – although this is not necessary for obtaining greater muscle force (Asmussen 1968). As mentioned above, intense training may increase the number of myofibrils, increase the sarcoplasmic content of myoglobin, enzyme systems, and energy depots. According to Man-I et al. (1967), fibre hypertrophy is greater in "pale" than in "red" fibres. They found no evidence of transformation of one fibre type to another. van Linge (1962), Riede (1966 b), and Asmussen (1968) have reported that the muscle content of connective tissue is increased, especially after isometric or eccentric training. However, there have not yet been any quantitative studies of the increase in connective tissue.

An increase in muscle force may be obtained by one daily, maximal isometric contraction (Müller 1970) or by repeated sub-maximal contractions of isometric or dynamic type. Sub-maximal contractions do not afford any training effect, if they are carried out by a force less than 40 % of the current isometric maximal force (Hettinger 1964), not even when repeated many times (Petow & Siebert 1926). The training effect manifests itself mainly in performances of the same nature as the training (Petersen et al. 1963, Hansen 1963).

Warming-up before an athletic performance empirically improves the

results. According to Asmussen & Bøje (1945) an increase in body temperature entailed both an increased muscle force (peak value) and an increased working capacity, at least for a short time. When the body temperature rises, the turnover rate of the chemical processes, rate of nerve conduction, and working capacity increase (Åstrand & Rodahl 1970). Asmussen (1966), moreover, reported that warming "softens" muscles, connective tissue, and joints, reducing the risk of rupture. As stated on p. 45 an increase of temperature seems to entail a reduction of the tensile strength of tendon tissue. Thus, more ruptures would be expected after warming up, but an increased temperature seems to involve at the same time an increase in extensibility, and perhaps this greater extensibility is the explanation why warming up may prevent a rupture.

Fatigue

During intense muscle work there may occur a rise of body temperature up to 40°C, a fall of pH to 7.0 (Åstrand & Rodahl 1970), and shifts in the ionic balance with a loss of intracellular K^+ and a gain in Na^+. Systemically there will be dehydration, but locally oedema (Fenn et al. 1938). However, Ahlborg et al. (1967) found an unchanged water content locally. As long as 24 hours after an exertion, tardive oedema may arise and last for up to a week. It is most marked after eccentric work and is presumably localized in the connective tissue (Asmussen 1956, Brendstrup 1962).

The energy depots (glycogen and creatine phosphate) are reduced, but as the turnover rate is extremely high, it has not been possible to demonstrate a reduction in the amount of ATP to less than 40 % of the initial value (Hultman et al. 1967, Karlsson & Saltin 1971). According to Bendall (1960) rigor mortis appeared at the time when the last of ATP had been split.

The exhausted muscle is more rigid, almost doughy, has a longer latent period, slower increase to a lower contraction height, and a protracted relaxation period (Lindhard 1931). EMG shows a certain synchronization (Bigland & Lippold 1954), whereas the integrated EMG shows increased innervation, if the same tension is to be maintained despite fatigue (Knowlton et al. 1951, Jørgensen 1964, Stephens & Taylor 1970, Kuroda et al. 1970, Lloyd 1971).

Merton (1956) demonstrated, among other things, that complicated movements during ischaemia could be carried out with accuracy until total exhaustion. In his opinion, the fatigue was exclusively restricted to the muscle. However, symptoms of fatigue, e.g. lacking coordination, occurring without ischaemia, must be derived from the central nervous system. This fatigue does not occur so suddenly and is to some extent subject to will (Åstrand & Rodahl 1970).

CHAPTER 9

VARIATIONS IN THE LOCOMOTOR APPARATUS

What has been stated in the previous chapters might erroneously lead one to believe that bones, tendons, and muscles are always of the same structure and always possess the same properties – but this is not so. A large number of factors cause changes which may be concurrent in the 3 types of tissue, but frequently they act mainly upon one.

Before discussing which of the 3 tissues is weakest, a further description of these variations will be given. They are related chiefly to age, activity level, sex, and diet and must be considered physiological. Pathological states, such as alterations in the hormone balance (e.g. steroid therapy) and various diseases, local or systemic, may of course also induce changes in the locomotor apparatus. However, any description of such pathological states is outside the range of the present paper, as there is no evidence that they are operative in more than a few cases of A.R.

Relative Growth of Muscles and Tendons

As already mentioned, the length of a muscle is determined by its structure and by the range of movements in the joints on which it acts (p. 50). The length of the tendon will be the remaining distance between origin and insertion (Le Gros Clark 1958).

Increase in muscle force during growth depends int. al. upon sex and body dimensions and is modified by training intensity (p. 60). This increase continues up to the age of about 30 (Fig. 5), but there is probably no age limit to the ability of training to increase muscle force (Müller 1970). How the tensile strength of muscles alters during growth we do not know. As mentioned on p. 60, there are signs that connective tissue increases on training. Assuming that the content of connective tissue in the muscle is decisive to the tensile strength, it must be expected that tensile strength increases on training.

Rollhäuser (1953–54) and Viidik (1967), training adult guinea-pigs and young rabbits respectively, found no change in tendon weight, but altered mechanical properties (p. 69). Tittel & Otto (1970), training 4-month-old

rats in running, found an increase in tendon cross section/100 g body weight, but in absolute values a minor fall as compared with the control group.

The growth of tendons plus muscles has been investigated by Ingelmark (1945, 1948) and Elliott (1964). In human subjects Ingelmark (1945) found the cell count per cubic mm tendon to fall from the neonatal period up to the age of 25 years. The total cell count per tendon cross section rose up to the age of 10 years, whereupon it fell. The increase in tendon cross-sectional area ceased at the age of 20. Training young rabbits (1945) and rats (1948) Ingelmark found that in Achilles tendons training entailed an increase in cell count and tendon cross section as compared with untrained animals of the same age. The muscle/tendon weight ratio was the same for trained and untrained rats. Training caused no change in the water content in relation to unexercised animals of the same age. If training was not started until growth had been practically completed, mice showed no increase in tendon cross section, but an increase in the physiological cross sectional area of the muscle (Ingelmark 1945).

As stated by Ingelmark, the cell count is not *per se* decisive to the growth of the tendon, but Ingelmark's demonstration that tendons can hypertrophy only up to a certain age accords with the findings of Holmes (1971). He made a morphological distinction between tenoblasts and tenocytes. In the rat Achilles tendon he found tenoblasts to predominate until the rat weighed 250 g. Thereafter the number of tenocytes increased. In human tendons – age not stated – more than 90 % of the cells were tenocytes.

Elliott (1964, thesis and 1965, a review) studied muscles and tendons from rabbit hind legs, partly the semitendinous muscle and partly the anterior and lateral hind-leg muscle groups. Tendon growth was assessed on the basis of dry weight per mm tendon or collagen content per mm tendon, whereas muscle weight was assessed on the basis of isometric muscle force or physiological cross sectional area. He found that penniform and fusiform muscles (predominantly with "pale" fibres) having a muscle force of 1 kp transmitted this force by 0.33 and 0.20 mg dry substance respectively per mm tendon. The fusiform semitendinous muscle, which contained predominantly "red" muscle fibres fell outside this pattern, containing more dry substance per mm tendon than the above-mentioned penniform muscles. Regrettably, the triceps surae-Achilles tendon were not studied.

Converted to muscle force per mm^2 fresh tendon, the isometric muscle force was 2.3 kp/mm^2 fresh tendon, and in Elliott's opinion there must be a wide margin of safety for tendons, if their tensile strength is calculated as being between 5 and 10 kp/mm^2. Elliott did not pay regard to the increase in tension in eccentric contraction. Nor did he himself measure

the tensile strength of the tendons, and lastly the measurement of isometric strength appears to be low compared with the results of a similar muscle force investigation (Alder et al. 1958). The cause was possibly a fairly long-lasting stimulation of the muscle which, combined with the anaesthesia, may reduce muscle force (Sirnes 1954).

In a series of experiments Elliott studied tendon thickness after changes in muscle function (hypertrophy, denervation, cutting of tendon, immobilization). He concluded (Elliott 1965 b) that the final thickness of collagen to be found in a tendon was a reflexion of the history of the tension transmitted by it and not necessarily related to the force of its muscle.

Summing up, it may be said that the muscle is able to hypertrophy for a larger number of years than the tendon, that this hypertrophy of the muscle is induced by repeated vigorous contractions, and that hypertrophy of the tendon is determined by the added tensions to which the tendon has been exposed.

Age

Age changes of the muscle consist mainly in a reduction of muscle mass, presumably because of reduced function. According to Rubinstein (1960) the muscle fibre proper did not change with age. Ikai & Fukunaga (1968) found muscle force in relation to muscle cross sectional area to be independent of age. Rubinstein (1960) felt that the electrolyte changes occurring with increasing age were due to changes in the connective tissue of the muscle. These changes consist in an increase of the extracellular phase, less increase of total water content, Na^+ increase and loss of K^+ (Lowry et al. 1942 a, Friedman et al. 1963). However, Horvath (1946) and Ingelmark (1948) reported a constant total water content, also for rat muscles. With advancing age the lipid content increases (Frantzel & Ingelmark 1950–51). Lowry et al. (1942 a) found that with age there was a tendency to increasing total muscle collagen.

According to Asmussen & Heebøll-Nielsen (1961) the isometric muscle force increased up to the age of 30, whereupon it fell – somewhat more rapidly in the legs than in the arms (Fig. 5). The curve corresponds approximately to that published by Ufland (1933) and Hettinger (1964). Changes in the tensile strength of muscles with advancing age are shown, together with those in the tendon and bone, in Fig. 6 (after Yamada 1970). Unfortunately, the curves represent different sites in the body: the muscle was the rectus abdominis, the bone was the femur, and the tendon was the Achilles tendon. Nevertheless, the figure gives the impression that the changes occurring with age are not parallel in the various types of tissue.

The tensile strength of the bone and its elongation at the rupture limit

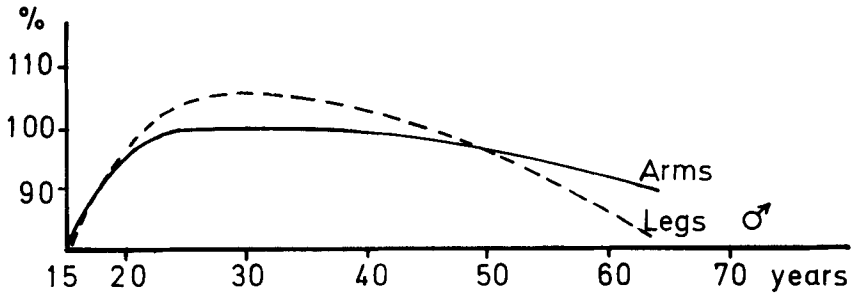

Fig. 5. Percentage isometric muscle force for muscle groups of arms and legs in relation to age, stated in per cent of the force at the age of 21 years (after Asmussen & Heebøll-Nielsen 1961).

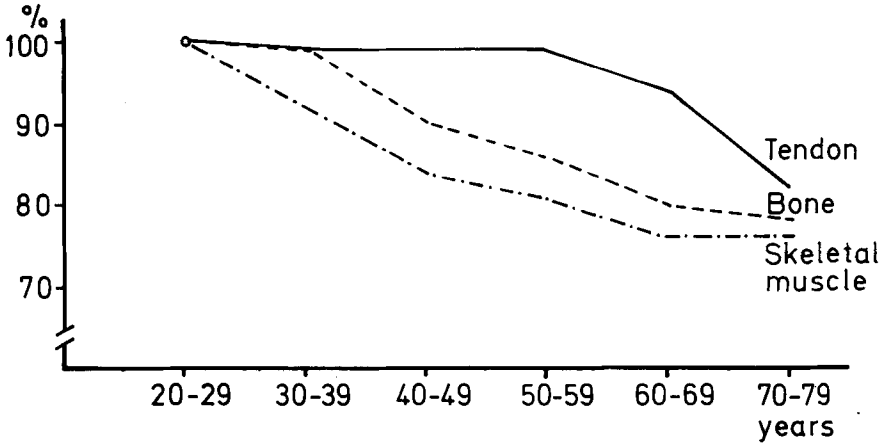

Fig. 6. Tensile strength of muscles (rectus abdominis), tendons (Achilles tendon), and bones (femur) in per cent of the strength in the age groups 20–29 years (after Yamada 1970, p. 273).

decrease with advancing age (Lindahl & Lindgren 1964, Yamada 1970). In 1967 Lindahl & Lindgren found that the specific gravity rose at the same time. Nilsson (1966) found an unchanged density – representing the mineral content – up to the age of 50, and thereafter a fall, more marked for ♀ than for ♂. However, his material was selected, the measurements being derived from the uninjured leg after fracture in the other. Vose et al. (1961), in senile osteoporosis, found an increase in strength per mm^2, but a decrease of 40 % for the total bone. In addition, they found that it was particularly the proximal and the distal ends of the femur which weakened. With advancing age there also occurs a fall in the hexosamine/collagen ratio, due especially to reduced hexosamine (Sobel et al. 1954).

Hübscher (1901), using perimetry, found reduced joint mobility with advancing age, but Ahlberg (1940) found only a slight reduction of mobility in the subtalar joints. Widén (1954) observed a decrease of from 25°.3 in young persons to 22°.9 after the age of 46.

The mechanical properties of tendon tissue rapidly change during growth, the tensile strength increasing and the elongation at the rupture limit decreasing (Rollhäuser 1951). The elongation proved to fall from 18 % to about 12 % and the tensile strength increased from 3.0–4.5 kp/mm^2 to 7.0–11.0 kp/mm^2. In human Achilles tendons Karasev (1969) found the tensile strength to double from the age of 10 to 30 (3.5 to 7.83 kp/mm^2). Thereafter, he observed a gradual fall down to 4.8 kp/mm^2 at the age of 70. Blanton & Biggs (1970) found only a slightly greater tensile strength in adult than in foetal human tendons, perhaps because the adult tendons were from elderly individuals. The exact relationship between these changes and biochemical changes has not been clarified. Reduced vascularization (Håstad et al. 1958/59, Rothman 1965) due to advancing age presumably plays an important role combined with a reduction in the cell count in the tendon (Ingelmark 1945, Zs-Nagy et al. 1969). With advancing age (and with straining of the tendon) Rollhäuser (1951) found better longitudinal orientation of the elements from which the tendon is built up, assessed by X-ray diffraction and birefringence.

The collagen fibrils grow thicker (Pahlke 1954, Schwartz 1961, Vogel 1964), and at the same time the variability of the diameters increases. The increase in thickness is tantamount to a reduced fibril surface. Jackson (1956) believed that this was the cause of the oft-described reduction in mucopolysaccharides (Boas & Foley 1954, Verzar 1957 a, Kao et al. 1960, Nemeth Csoka 1965, Bihari-Varga & Biro 1971). Schmitt et al. (1970 b), however, observed a slightly increasing mucopolysaccharide content. The glycoproteins also decrease, especially during growth (Robert et al. 1971). Elden (1964, 1968) believed that the electrostatic forces between collagen and mucopolysaccharide influenced the hydration and elasticity. The water content is rapidly falling during growth, later more slowly (Ingelmark 1948, Schmitt et al. 1970 b, Boas & Foley 1954). Takacs & Verzar (1968) reported a constant water content of 60 % in rat-tail tendons. According to Bihari-Varga & Biro (1971) water binding to tendon tissue is firmer with advancing age. This they found to be correlated to the number of cross links. Assessed on the basis of the shrinkage temperature the number of cross links has been found to increase with age (Brown & Consden 1958, Chvapil & Jensovsky 1963). Takacs & Verzar (1968) found similar changes, and Verzar (1967 b) reported that the shrinkage temperature could be utilized for determining age, but with decreasing accuracy as age advanced.

Simultaneously with the increase in the number of cross links the

solubility decreases (McGavack & Kao 1960, Takacs & Verzar 1968, Jackson & Steven 1969, Piez 1969 a). The last-mentioned author emphasized that growth changes are marked and age changes slight. Bihari-Varga & Biro (1971) pointed out that tendon tissue matures quickly, but ages slowly.

The explanation of the increased number of cross links is to be sought in an increasing amount of fluorescent substances (La Bella & Paul 1965, Zs-Nagy et al. 1969). Bjorksten (1962) considered the cross links to have arisen on the basis of metabolic products, especially in connection with anoxia. In his opinion, the cross links entailed a "frozen metabolic pool" which is incompatible with life when attaining a certain size. However, Deyl et al. (1971 a), studying various animal species, demonstrated that the increased number of cross links with advancing age were determined by the numerical age not by the relative age, i.e. age in relation to expected lifespan.

Like the muscle, the tendon also shows increasing lipid deposits with advancing age (Moissejeff 1921, Chaletzkaja 1934, Lang 1951).

Sex

Women's muscle force is below that of men (Hettinger 1964), also after correction for the difference in height (Asmussen & Heebøll-Nielsen 1961). This is presumably due to less muscle mass, as Ikai & Fukunaga (1968) found no significant sex difference in the maximum, isometric, voluntary muscle force per anatomical cross-sectional unit in the same muscle groups. There have apparently been no investigations on sex difference in tensile strength.

Locally in and around the birth canal the mechanical properties of the connective tissue are affected during pregnancy, but in a review on this subject (Harkness 1964) it is not stated whether connective tissue far away from the birth canal is affected too. According to Arvay et al. (1963) repeated pregnancies may render the connective tissue biologically older than in females of the same age but with a history of fewer pregnancies.

In human femora Lindahl & Lindgren (1967 a, b, 1968) found the density of the compact bone to increase gradually with advancing age in men, whereas it remained unchanged, or even fell, in women, especially after the age of 50. The cross-sectional area was significantly smaller in women, but no correction had been made for the difference in height. There was no sex difference in tensile strength or deformation, but the compressive strength was greater in males. Incidentally, Lindahl & Lindgren reported extremely great individual differences in the parameters measured.

Diet

The diet affects experimental animals, and presumably also human beings, causing quantitative as well as qualitative changes.

In several experimental series it has been found that restriction of a qualitatively sufficient diet increases longevity, reduces morbidity (Hansen & Steensberg 1950, McCay 1952, Berg 1967) and gives biologically younger muscle tissue (Lowry et al. 1942 b) and connective tissue (Deyl et al. 1971 b, Everitt 1971). The same changes are obtained by exposing rats to intermittent food intake (sufficient diet *ad libitum* once or twice weekly) (Holecková & Chvapil 1965).

Qualitative changes in diet may be int. al. lack of vitamin and insufficient supply of Ca. Severe avitaminosis C can hardly be an undiagnosed cause of A.R., but milder deficiency of vitamin C may possibly contribute to altering the strength of connective tissue. Vitamin C is not only necessary for collagen formation, but also influences the composition of the ground substance (Kirchheiner 1968) and is necessary for preserving new-formed collagen (Gould et al. 1960).

A diet with a Ca content of less than 0.36 %, despite a sufficient supply of vitamin D, reduces the strength of rat bones on bending and torsion (Bell et al. 1941). At the same time, the ash content is reduced and the straining is increased (Bell et al. 1947, Weir et al. 1949).

Training

The role of training has previously been discussed on pp. 60 and 62 as regards its influence upon muscle force, muscle hypertrophy, and endurance. The influence of training upon the growth of tendons has also been mentioned.

In experimental animals capillary density has proved dependent on the state of training (Petrén et al. 1937), but in muscle biopsies from trained and untrained persons Hermansen & Wachtlova (1971) detected no significant difference in capillary density. Wendt (1952) found the muscle to contain 2 % more water during training-induced hypertrophy, but after training was stopped the water content returned to normal (74.5 %). As already mentioned the connective tissue in the muscle appears to increase on training.

The number of capillaries in the tendon also increases on training of rats (Maurer et al. 1969). Wrete (1951) reported that the dry substance of the tendon increased from 60 to 77 per cent while the content of mucopolysaccharides increased. After training mature quinea-pigs Rollhäuser (1954 a, b, 1953–54) observed first a slight fall in the birefringence and tensile strength of the Achilles tendon. At the end of two weeks there was an increase to 120 % of the initial value. The water content rose a bit in order to drop insignificantly later. The girth of the

tendon and the histological appearances were unchanged. The tensile strength increased, and this was interpreted as an improved structure of the tendon, i.e. a qualitative change. The effect of the excessive action to which Borsay et al. (1952) exposed tendons (cf. p. 22) cannot be compared to any training programme.

Training immature rabbits, Viidik (1969 a) found that in loading experiments the bone-muscle-tendon-bone chain broke as avulsion of bone. The elongation was measured only for the tendon. On training, the elastic stiffness tended to decrease. In experiments on isolated tendons (Viidik 1967) a higher ultimate tensile strength was found after training. Several different tendons from the lower leg were investigated. The elastic stiffness of the tibialis posterior muscle decreased, whereas an increase was found in the peroneus longus, tertius, and quartus. Investigation of water content, dry substance, and collagen content revealed no significant differences, only a tendency to a greater collagen content per 100 mg dry substance after training. Viidik, like Rollhäuser, concluded that the changes induced in the tendon by training were qualitative rather than quantitative.

Tittel & Otto (1970) carried out a 117-day training programme on 4-month-old rats. One group ran daily for 60 min. 40 m/min., another group 20 times daily 1 min. 70 m/min., and the last group 4 times 4 min. 80 m/min. The body weight increased appreciably more in the control group than in the exercised groups. As mentioned on p. 62, the cross-sectional area of the tendon increased in relation to body weight, but in absolute figures there was a minor fall. Thus, it was not possible to demonstrate any definite quantitative difference, but qualitatively there were changes, the training giving rise to a significant increase in the tensile strength, and a simultaneous decrease in ultimate straining. The changes were most marked in the first-mentioned group (slow, sustained running). Tittel & Otto recommended training of this nature to strengthen the Achilles tendon in athletes. However, their description gives no impression of the effect of this type of training upon the muscle or the effect of training, if the running had been started at an age so early that an absolute increase in the cross-sectional area of the tendon could have been anticipated (cf. Ingelmark 1945, 1948, p. 63).

On knee ligaments several rupture experiments have been conducted after training. Adams (1966) reported that female rats developed stronger ligaments when trained on a rough than on an even surface. Training inmature rabbits Viidik (1968 b) found a significant increase in the tensile strength and "failure energy" of the anterior cruciate ligament, whereas the ultimate straining tended to increase. Tipton et al. (1967 a, b) and Tipton et al. (1970) trained partly rats and partly mature dogs. In the knee ligaments they found an increase in tensile strength as well as of

elongation. Four weeks after the training (Tipton et al. 1967 a) the tensile strength was still increased, but the elongation was less than in the control group. The water content was unchanged, whereas the collagen content increased. Zuckerman & Stull (1969) training domesticated rats in running, found a significant increase in the tensile strength of the knee ligaments, but no difference between animals kept in cages and animals kept in runs. In all Tipton's experiments the rupture occurred as avulsion of bone. A few of Viidik's experiments ended in rupture in the ligamentous substance – without any relation to the training.

In general, it may be said that changes in elongation reflect properties of the tendon, whereas changes in tensile strength in the above-mentioned experiments have reflected mainly properties of the bone.

The influence of training upon the bone has been studied in a more direct way by Saville & Smith (1966) and by Smith & Saville (1966). Their experimental animals were rats whose forelegs had been amputated at an early age. The increased load upon the hind legs of these bipedal rats caused increased specific gravity, Ca content, and tensile strength of the bone. At the same time, muscle mass increased. Lindahl & Lindgren (1967 a) believed that the mechanical stress was the factor which determined the thickness and strength of the bone. Nilsson & Westlin (1971), using a photon absorption method, investigated bone density (mineral content g/cc) in femoral condyles. Their material consisted of normal adult persons in different states of training. They found increasing bone density with increasing degree of training, the highest values in weight-lifters.

Inactivity

After 6 weeks' immobilization of rabbit legs in plaster Rothman & Slogoff (1967) observed a decrease of capillary volume, assessed by the content of Cr^{51}-labelled erythrocytes, in tendons, but unchanged in muscles. As a control they used the healthy leg, and this is not satisfactory, as the other legs must be considered trained. This method also fails to afford any information about a possible closed capillary reserve. According to Maurer et al. (1969) the rat Achilles tendon, after being kept in plaster, contained more vessels, but the tendons of trained rats more capillaries.

Eccles (1944) found a 60 % weight loss in muscles 3 weeks after denervation. Fischer & Ramsey (1945/46) reported that in rabbit legs denervation resulted in a greater loss of myosin than did immobilization. Kohn (1964) as well as Brooke & Bewick-Slack (1959) stated, for rats and rabbits respectively, that the re-building of muscle fibres and the connective tissue of the muscle continued despite denervation, but that breakdown increased. In a histological study Patel et al. (1969) found no changes in the fibrils of denervated and immobilized muscles from human subjects. However, in some of the cases the diameter of the fibres was

smaller. Both Hines & Knowlton (1937) (rats) and Eichelberger et al. (1958) (dogs) described the same changes in the muscle after denervation and after immobilization. The water content was unchanged or slightly increasing, the Cl^- and Na^+ content rose, and the K^+ content fell. According to Eichelberger et al. (1956) and Ferolla (1963) the collagen content rose. The content of fat rose from 2 – 15 % of the muscle weight after immobilization for 11 weeks (Eichelberger et al. 1958). In the opinion of Hines & Knowlton these changes were due to the greater share of connective tissue in muscles, whereas Eichelberger stated that the changes represented extracellular oedema.

Three weeks after denervation of rat muscles Stolov & Weilepp (1966) observed that the stiffness of the muscles increased, whereas that of the tendon remained unchanged. In their measurements they used only rather slight loading, and the tensile strength was not measured. In a subsequent study (Stolov et al. 1970) the elastic equilibrium length was found to increase following denervation. The hydroxyproline content exhibited an absolute fall, but a relative increase. Since the hydroxyproline determination was carried out on the gastrocnemius muscles and Achilles tendon combined, it is not possible to evaluate whether the change occurred in the muscle only or in the muscle plus tendon.

Rollhäuser (1953–54) observed, 6 weeks after having destroyed the sciatic nerve of mature guinea-pigs, a fall in the birefringence of the Achilles tendon and an increase in its water content. The tensile strength fell by 20 %. Microscopy showed no changes, and according to Rollhäuser a less adequately arranged structure on the molecular plane was responsible for the changes. The tendons gradually returned to normal, so that the changes were reversible. Akeson & La Violette (1964), after immobilizing dog knees, studied the adjacent tendons. After an initial increase in the mucopolysaccharide content, stated in relation to dry tendon weight, a drop to 30 % was observed. The water content also decreased. In a subsequent study (Akeson et al. 1968) the changes due to immobilization were compared to changes of ageing.

After immobilization of joints the thickness of cartilage is diminished (Holmdahl & Ingelmark 1948) and the adjacent ligaments frayed (Mathiass & Glupe 1966). Peacock (1963, 1966) found the paraarticular connective tissue to be unchanged as regards the number of cross links assessed by shrinkage temperature. The amount of collagen tissue was greater, and in Peacock's opinion this was the cause of joint stiffness.

During disuse of the bone because of denervation of the muscles or immobilization, the strength of the intact bone is reduced (Allison & Brooks 1921, Adams 1968), but the bone strength per mm^2 remains unchanged (Allison & Brooks 1921, Gillespie 1954, Semb 1966). Thus, the change in the bone is quantitative rather than qualitative.

Wild – Domesticated

Richter (1954) has published a characteristic of wild rats and their domesticated albino forms: "The wild Norway rat lives in an environment in which it must constantly be on the alert and ready to fight for its existence. It has to defend itself against all kinds of enemies: rats, dogs, cats, owls, and snakes, as well as man. It is a fierce, aggressive, suspicious animal that attacks at the least provocation and in captivity takes advantage of the least opportunity to escape, remains suspicious and tense, and breeds poorly.

In marked contrast, the healthy domesticated Norway rat is tame, gentle, and trusting, does not bite unless frightened or hurt, and makes no attempt to escape. It lives placidly in the controlled environment of the laboratory where food, water, shelter, and safety are constantly assured. Its only contribution to its own survival are its feeding, drinking, grooming, and mating activities. It reproduces at an early age and with a rapid rate".

King, in 1923, gave a similar description. These two types of rats mate mutually, but not with other species of rats, e.g. the Egyptian rat (Donaldson 1924, Richter 1954).

Richter (1954) has described a number of differences between wild and domesticated rats, int. al. larger adrenals in the wild rats and a larger pituitary gland in the domesticated ones. In his opinion, some of the existing differences might be due to a selection of experimental animals, the most aggressive domesticated rats biting their young to death.

King (1923) found growth to be more rapid in the domesticated rats up to the age of one year, but thereafter growth was more marked in wild rats.

Comparison of the femoral bones of wild and domesticated rats (Vostal et al. 1961) has shown the same Ca content, but a high, constant, collagen content in wild rats, while in domesticated rats the collagen content fell after the rat had attained a body weight of 60 g. According to Chvapil & Roth (1964) the named differences in collagen content were diminished by training of the domesticated rats. At the same time they found that the collagen content in lungs from wild rats was constant, whereas it gradually increased in the domesticated ones. For this parameter there were sex differences only among the domesticated rats. The tendons were biologically younger, judging by a greater speed of relaxation following chemical contraction. Comparison of hare/rabbit, wild/domesticated rat, and wild/domesticated duck has shown the same pulmonary changes as mentioned above, and a larger heart with less collagen content in the wild animals; however, this was not significant in the case of the ducks (Chvapil et al. 1966). This team did not study the tensile strength of bones or tendons in these groups of animals.

Chvapil (1967) wrote: "In several respects domestication is unfavourable for full functional development and ageing of the body. Relative overfeeding and restriction of movement are very probably responsible for accelerated ageing of many structures and functions in the domesticated rat". Chvapil gave many examples how differences between these groups of animals might be diminished or abolished by approaching the conditions of living for the two groups to each other. This accords with the view advanced by Donaldson as early as 1924.

CHAPTER 10

PRESENT INVESTIGATIONS, RESULTS, AND DISCUSSION

From the preceding chapter it was apparent that age and conditions during growth are important factors in the development of tendons, muscles, and bones. As these factors are also essential in the occurrence of human A.R. (p. 17), they must clearly be borne in mind in tendon rupture experiments on animals. This was attempted in the present study by comparing wild with domesticated animals. Rats were chosen for experimental animals, as wild rats could be procured in reasonable numbers. A further description of the experimental animals is necessary to evaluate the results of the actual rupture experiments.

The Rat as an Experimental Animal

The wild rats, captured in 3 different places in this country, behaved as has been described on p. 72. They were healthy in appearance and had a thick fur. Only a few old rats had tumours, and the mortality after capture was low. The rats were grouped by weight, as an age determination would have been very inaccurate, especially in the case of the old animals. Since the age criterion could not be applied to the wild rats, no attempt was made to use it in the other groups.

All the domesticated albino rats were obtained from a drug factory where they had been kept in cages of about 30 x 50 cm. They were healthy, and none of them had been used in trials of drugs. They had received a sufficient diet *ad libitum.*

The rats kept in runs (hereafter called run-kept rats) were the 1st or 2nd generation of wild brown rats which had grown up in a run measuring 4 x 3.5 m. These rats too had received a sufficient diet *ad libitum.* They had been used in a trial of rat poisons, but were in good health at the time of the experiment. They behaved almost like the wild rats, and in the run they kept up a pecking order as known from other animal communities.

In using an experimental model composed of these groups of different rats one must abstain from the uniformity obtained by inbreeding and identical diet. According to Chvapil (p. 73), however, there is reason to

expect that the three groups would have become uniform in most respects, if they had grown up under the same conditions. The intermittent food intake by the wild rats may have entailed some of the differences found, but not the differences between the domesticated albino rats and the "run-kept" rats. It must be reasonable to consider the difference in activity level as the predominant difference between the three groups.

The fourth group, called the inactivity group, consisted of wild rats which for 3 months had been kept in cages where they received a sufficient diet *ad libitum*. Although a change in diet may have affected these animals, the altered (restricted) possibilities of moving must be considered the main difference from the other groups.

As in man, the rat triceps surae originates from the posterior surfaces of the femoral condyles. The soleus part originates with a flat tendon high on the posterior tibial surface. The muscles join and gradually merge into the Achilles tendon which twists about 60° before inserting on the posterior surface of the calcaneus – mainly on 2 facets, medially and laterally. In the rat it is very easy to separate the tendons of the individual muscle fractions right down to the calcaneus. In other words, there is very sparse interdigitation. Mobility in the talo-crural joint and in the subtalar joints permits an approx. 30° supination and a 20° pronation of the calcaneus. In close contact to the Achilles tendon there is the strong plantaris tendon whose muscle belly originates from the lateral femoral condyle, extending down between the gastrocnemii and the soleus, gradually shifts to the posterior aspect of the Achilles tendon in order to continue in a groove on the posterior aspect of the calcaneus and further down into the sole. This tendon was resected in all the rupture experiments.

Grossly, the rat anatomy differs from human anatomy in the strong plantaris muscle, in a more punctate insertion, and in less interdigitation of the tendon.

As the junction from tendon to muscle is poorly defined owing to the large aponeurosis, this length was not routinely measured. The total length of muscle plus tendon was about 5 cm and that of the free tendon in the order of 1.5 cm.

Foot length (the distance from the heel to the longest toe), muscle-tendon length (the distance from the origin of the muscle to the insertion of the tendon with extended knee and the foot in maximum dorsiflexion), and excursion (the difference between muscle-tendon length and length with maximally flexed knee and plantar-flexed foot) were measured on all the rats (Barfred 1971 c). These 3 parameters increased at a decreasing rate in relation to body weight. Longitudinal growth in the foot decreased at a lower weight than did the muscle-tendon length. Foot length as well as muscle-tendon length were significantly greater in the wild than in the

domesticated rats and greater in males than females of the same body weight. This may be of genetic nature, but may also be due to the domesticated rats being overweight as compared with the wild rats. In that case the run-kept group and the inactivity group must have been underweight, as their longitudinal measurements were greater than those of the wild rats. Lastly, the walking function among domesticated rats as opposed to the more jumping function among the wild ones may have affected the longitudinal growth. The increase in the excursion appears to accompany the foot length, and the relationship between these parameters was constant for all groups apart from the run-kept rats in whom the excursion was greater.

Before each rupture experiment muscle force was measured as the isometric force at elastic equilibrium length following maximum stimulation of the sciatic nerve. The muscle force-body weight relation decreased with increasing weight, so that the muscle force-log. body weight relation may reasonably be described as linear (Fig. 10, Barfred 1971 c). The degree of muscle force depends upon the physiological cross section, and according to Asmussen & Heebøll-Nielsen (1955) the force might have been expected to show a linear relationship to the longitudinal measurement (e.g. muscle-tendon length) in the second power. However, the muscle force-muscle-tendon length relation proved to be linear and even identical for all groups. The only deviation was that the muscle force of the pregnant rats was lower in relation to the muscle-tendon length. Since the muscle-tendon length does not alter during pregnancy, the reduction in force must be interpreted as being caused by the pregnancy, an observation which has not been reported by others.

Force in relation to weight was less in domesticated than in wild rats, but when considering only the large rats there was merely a tendency to less force in the domesticated ones. The inactivity group had not suffered any loss of force during the period in cages. Muscle force was measured during brief isometric contraction and thus cannot disclose any difference in endurance, in the ability to carry out work.

On a small number of rats from the various gruops, the weight of the m. triceps surae and of the Achilles tendon was determined. In addition, dry weight, fat-free weight, Na^+ and K^+ content, and hydroxyproline content in the tendon as well as muscle were determined (Barfred & Langgård 1973). Muscle weight was the same in relation to body weight in the various groups at the same body weight, but decreased with increasing body weight. The same applied to the tendon weight in the younger rats, whereas the tendon weight (free tendon + aponeurosis) was significantly greater in the large wild rats. Muscle length in the wild rats must be expected to be increased, as the range of movement is greater (cf. p. 50). Thus, although the muscle-tendon length was slightly increased, there is

no reason to believe that the tendon had grown longer. It is likely, therefore, that the increased tendon weight represents a greater cross-sectional area. This was only to be expected, as the increased muscle force in the small wild rats must be interpreted as a sign of increased training, which is a stimulus to the growth of the tendon cross section (p. 64). Thus, the muscle weight-tendon weight ratio was highest in the domesticated rats, lowest in the wild ones. The run-kept rats occupied an intermediate position, whereas the ratio for the inactivity group was the same as for the wild rats. (Table 7, Barfred & Langgård 1973). The muscle weight-tendon weight ratio decreased with increasing activity.

No studies of the bone were made, but it is worth mentioning that on drilling with a manual drill into the calcaneus considerably greater force and patience were required in the wild than in the domesticated rats to get through the bone.

Barfred & Langgård (1973), like Elliott (1964), found no significant difference in the hydroxyproline content per 100 mg fat-free dry substance in the tendons of the various groups. Viidik (1967) observed a tendency to a higher collagen content in the tendons of exercised rabbits. The muscular content of collagen, assessed on the basis of the content of hydroxyproline, showed such marked variability that no differences between the groups could be disclosed. The hydroxyproline determination was not performed on the entire muscle, but on a part of it which was, as far as possible, free of tendon tissue. Total determination would have been preferable.

Investigation of the electrolyte and water content of the muscles revealed but few differences. The K^+ concentration fell with increasing body weight. This fall was most marked in the wild rats, a difference which cannot be explained. The most striking finding was dehydration of the muscles in the inactivity group.

The water and Na^+ content of the tendons was constant in the wild rats, but decreasing in the domesticated ones. This corresponds to the fact that the tendons of the wild rats were what Chvapil (1967) would call biologically younger. The high K^+ content in the tendons of the young wild rats must be interpreted as a sign of a higher cell count and thus an indication of greater training (cf. Ingelmark 1945, 1948). It is not known why the tendons in the inactivity group had a significantly higher K^+ content than those of the other large rats. Histological investigation (Barfred 1971 a) did not demonstrate any cellular infiltration in these tendons. The values for water and electrolyte content in the run-kept group were intermediate between those in the domesticated and in the wild rats.

The tendons in the inactivity group, like the muscles, were dehydrated compared to the other two groups. It is difficult to make any comparison

with other investigations, as in our case there was a question of restriction of total activity, whereas in previous studies (p. 70) activity has been restricted in only one limb. Such studies have shown an increased water content in muscles (Hines & Knowlton 1937, Eichelberger et al. 1958) and tendons (Rollhäuser 1953–54). However, Akeson & La Violette (1964) found a decreasing water content in tendons around immobilized dog knees and simultaneously a 30 % decrease in the mucopolysaccharide content.

Histological investigations of Achilles tendons (Barfred 1971 a) revealed parallel tendon bundles separated by slender fibrocytes which, however, were ever more reminiscent of chondrocytes as the insertion was approached (Fig. 1, Barfred 1971 a). This appearance corresponds to other authors' descriptions of the free tendon and tendon insertion. Moreover, halfway up the tendon, about half the investigated tendons showed foci of cartilage-like metachromatic tissue which in some cases contained calcium, but never bony tissue. As the occurrence of these foci was independent of age and group of rat examined, they cannot be interpreted as degenerative changes, but have probably been caused by pressure forces from the crossing plantaris tendon. In a few cases the peritendineum was thick and cellular, but in these cases too the underlying tendon was normal. Grouping was not possible on the basis of the histological appearances, and the investigation prior to the rupture experiment revealed no signs of degeneration.

Since most of the differences between the groups of rats may reasonably be explained as being due to differences in activity level, it is considered justified to use these animals as an experimental model in studying the influence of the activity level in rupture experiments on the bone-muscle-tendon-bone unit.

The Rupture Experiments

The rupture experiments were carried out under pentobarbitone sodium or ether + pentobarbitone sodium anaesthesia after the rats had been fastened in a material testing machine the details of which have been described previously (Barfred 1971 c). In all the rats the lower leg, apart from the Achilles tendon, was severed to secure rupture at some site of the bone-triceps surae muscle-Achilles tendon-bone chain. Fastening was done by wire traction in the calcaneus and a combination of wire traction and clamp in the condyles. Unless otherwise stated, the rupture experiment was performed by a quick manual elongation of the muscle-tendon unit at an initial length corresponding to the elastic equilibrium length. During the rupture experiment the muscle was maximally stimulated by a double electrode on the sciatic nerve. The calcaneus was 30° rotated into supination on the left leg, pronation on the right.

The increase in tension and the elongation were recorded simultaneously on graph paper moving at a constant speed. On the graphs the tension and elongation at the rupture limit were measured. In addition, elongation at an increase in tension up to 1.5, 2.0, 2.5, and 3.0 times the isometric muscle force was measured. Thus, $L_{2.0}$ was the elongation at an increase in tension up to 2 x the isometric force. $\Delta L_{1.5}$ signifies elongation from $L_{1.5}$ to $L_{2.0}$ and thus affords an impression of the inclination of the steep part of the length-tension diagram.

In the rupture experiments rupture occurred either at the site of fixation in the calcaneus or femoral condyles ("fixation"), or as tendon rupture ("tendon"), or as muscle rupture ("muscle"), or combined in the group "elsewhere" as avulsion of a chip of bone from the calcaneus or femoral condyle and in rare cases as rupture at the muscle-tendon junction (Figs. 7 and 8). The fixation ruptures predominated among the domesticated and the young rats. The small domesticated rats, not shown on the figures, invariably exhibited fixation ruptures. Of course, a fixation rupture suggests an insufficient method, but yet affords an impression of the strength, in particular the strength of the bone. Therefore, these experiments were not rejected. From Fig. 11 (Barfred 1971 c) it is apparent that several of the fixation ruptures among the large domesticated rats had required greater force than the only tendon rupture which occurred in this group. It has been mentioned above (p. 77) that the bones of the wild rats appeared to be stronger than those of the domesticated rats.

All the tendon ruptures were close to the calcaneus, as a rule at a distance of 0–2 mm and frequently oblique, so that the tendon remnant on the calcaneus might be up to 4 mm in length (Fig. 6, Barfred 1971 c).

The side on which the tendon remnant was situated was not mainly the medial side or the lateral side, and it did not depend on whether the trauma was inflicted in a position of pronation or supination. The criterion of a tendon rupture was that no gross or microscopic bony tissue could be found in the proximal tendon stump. In all cases the proximal tendon stump was studied histologically, but it was in only one case that this was necessary for classification. The constructions described on p. 37 in the human Achilles tendon and at the junction from tendon to bone, and which may be imagined to protect the tendon from uneven weight-bearing, were int. al. fan-shaped spread at the insertion, interdigitation of the fibres, twisting of the tendon, and gradual transition from tendon to bone. Among these constructions only the last two are present in the rat. Therefore, it cannot be taken for granted that the site of rupture must be the same as in man. Owing to the lack of interdigitation, in particular, the oblique traction may be transmitted direct down to the insertion, so that rupture would be more apt to occur in this site. The

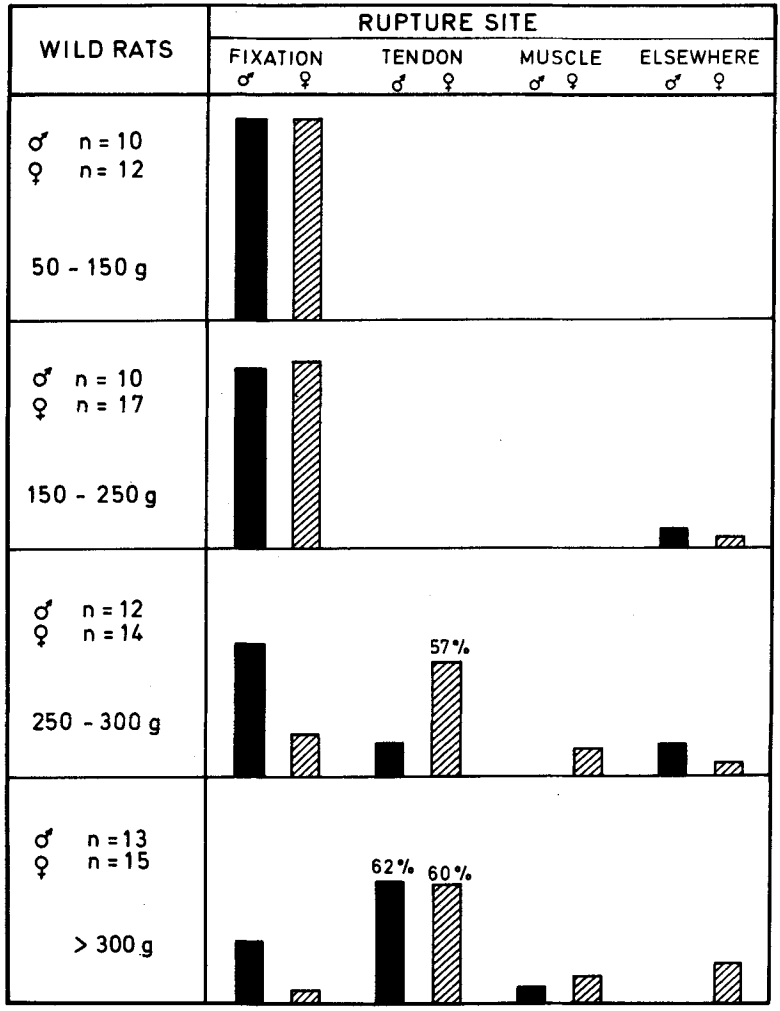

Fig. 7. Distribution of rupture site by weight and sex of recently captured wild rats. Height of columns determined by the percentage number of ruptures. It will be seen that tendon ruptues occur with the same frequency in females weighing 250–300 g and in males as well as females weighing more than 300 g (Barfred 1971 c).

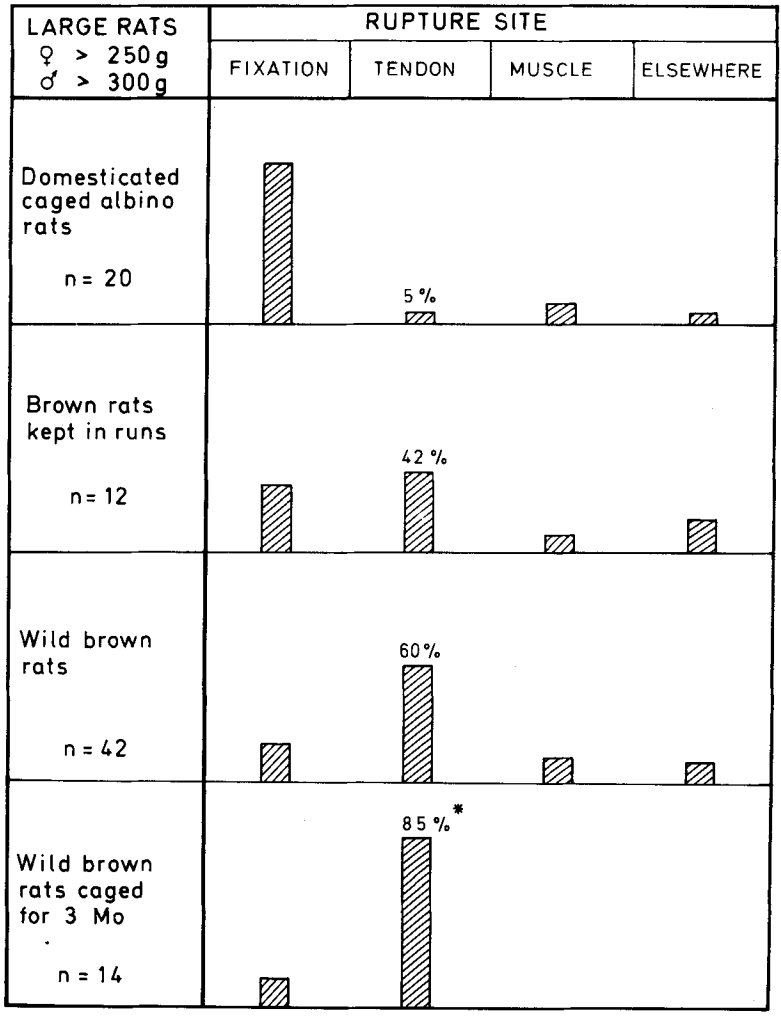

Fig. 8. Distribution of rupture site by large rats' conditions of living (o > 300 g, ♀ > 250 g). Height of columns determined by the percentage number of ruptures (stated for the tendon ruptures only).
*) significantly different from "run-kept" rats (p = 4%), but not from the recently captured wild rats (p = 12%) (Barfred 1971 c).

absence of fan shape may also mean that the force reaches the insertion directly, not being concentrated at the narrowest site.

The distribution of the rupture site revealed that with advancing age and increasing activity the bone grew so strong that it could "withstand" the fastening in the machine. From then on the rupture occurred mostly in the Achilles tendon. Tendon ruptures occurred most often (85 %) in the group which had passed from the highest degree of activity to inactivity. Next in order were the wild rats (60 %), the run-kept rats (42 %), and finally the domesticated rats (5 %). The difference between the inactivity group and the run-kept group was significant, but not that between the inactivity group and the wild rats. This distribution is shifted only slightly, if the fixation ruptures are excluded (100 %, 74 %, 63 %, and 25 %). Even though tendon weight was highest in the wild rats, the number of tendon ruptures was also greatest. Accordingly, the other structures, viz. bones and muscles, must have been strengthened more than the tendon.

The separation force in relation to muscle force was increasing from domesticated to run-kept to wild rats, but was significantly lower in the inactivity group than in the wild group. The elongation at rupture limit in relation to muscle-tendon length was the same in the domesticated, run-kept, and wild groups, but significantly lesser in the inactivity group. $L_{2.0}$ showed no difference in these groups, whereas $\Delta L_{1.5}$ (the inclination of the steep part of the length-tension curve) was somewhat less marked for the domesticated rats.

Histological examination of the ruptured tendons showed no degeneration, but slight fraying and a more wavy structure in the tendon fibres without these changes permitting a classification of the individual specimens in relation to the microscopic appearances prior to the rupture experiments.

It was emphasized on p. 24 that the results of a histological examination of biopsies from ruptured Achilles tendons were not consistent and that, in particular, it does not appear to be realized which changes the rupture episode can entail in a healthy tendon. After rupture experiments resulting in tendon ruptures skin suture at the level of a low-level lower-leg amputation was sometimes performed after the proximal tendon stump had been bent upwards between the soleus muscle and the deep calf muscles. 1, 2, 4, or 8 days later the tendon stump was removed and studied histologically (Barfred 1971 a). Already after the lapse of 24 hours there was reduced stainability of the fibres which were frayed and the fibre bundles split up. The cell nuclei became irregular and pyknotic. The cell count seemed unchanged, and the stainability with alcian blue and toluidine blue also appeared to be unchanged. These changes did not increase in the older specimens. The experimental model

was satisfactory in so far as the tendon studied had been torn apart and not, as in previous experiments (p. 23), merely cut. It was unsatisfactory that the tendon end was so thin that it might almost be able to survive by diffusion and that it was situated in loose connective tissue and not in a fairly unyielding tendon sheath entailing a possibility of oedema and congestion as after human A.R. On the basis of these objections the changes described above must be considered the mildest ones that may be expected after rupture of a healthy tendon.

The changes described at an early stage after human A.R. are of the very same nature, and even though these changes are more pronounced it seems difficult to accept that they should represent pre-existing degeneration. When occasionally an early biopsy shows more severe changes with cellular infiltration, the possibility cannot be ruled out that these are changes left by a previous partial rupture in a healthy tendon.

Although the investigations have shown that the healthy tendon may be the weakest link in the chain in certain age groups and at a given activity level, subcutaneous tendon rupture is rare. The following experiments were carried out to detect under which conditions the risk of tendon rupture is at its height. These experiments were concerned with variations in the position of the knee and ankle joints, in the speed of the movement, and lastly changes in the contraction state and exhaustion of the muscle. The method and results have been described in detail by Barfred 1971 d. The significance of the variations was assessed partly by differences in the distribution of the site of rupture (Fig. 9) and partly by differences in the parameters of the rupture experiment. Only wild rats weighing more than 250 g (females) and 300 g (males) were included in these experiments. The control group was made up of the wild rats (Fig. 8) fulfilling the same conditions and subjected to the experimental conditions as described on p. 78.

At a neutral position of the calcaneus (straight traction) instead of a 30° angulation as in the control group, the number of tendon ruptures fell from 62 % to 37 %. The number of other types of rupture, apart from fixation ruptures, increased, and it was found that in this group muscle ruptures occurred in rats with great muscle force, an occurrence not seen in the control group (Fig. 6, Barfred 1971 d). Although the difference in the frequency of tendon ruptures was not significant ($p = 13$ %), the distribution of the rupture site and the occurrence of muscle ruptures in strong rats at straight traction seems to support the assumption that the oblique traction weakens the tendon selectively.

A reduction in the speed of traction (slow traction) caused neither a change in the distribution of the rupture site nor in the parameters of the rupture. Owing to the viscous properties of the tendon the elongation might have been expected to become greater and the separation force

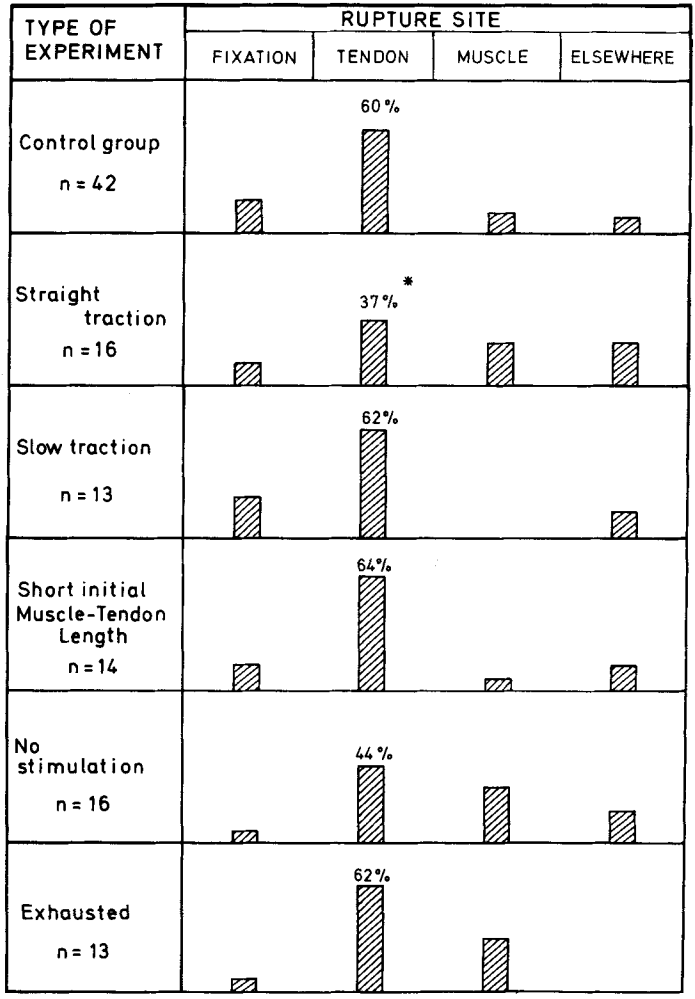

Fig. 9. Dependence of distribution of rupture site upon type of experiment on recently captured large, wild rats. The height of the columns is determined by the percentage number of ruptures.
*) On comparison with the tendon ruptures in the control group p = 13%. (Barfred 1971 d).

lesser at slow traction (Welsh et al. 1971), but no such difference was demonstrable by the present measuring technique, or else the difference in speed was too slight.

The elongation needed to induce rupture was greater if the initial length was short (short initial muscle-tendon length) corresponding to flexed knee and plantar flexed foot, but the difference in elongation was less than the difference in initial length. The final length, i.e. the total length at rupture, was significantly shorter and the chance of obtaining rupture within the length obtainable in the intact organism greater.

It is essential to emphasize that in all cases the rupture experiments necessitated an elongation exceeding that which could have been obtained in the organism. In a few experiments this excess amounted to only a few mm, and by combining the circumstances at which the final length was least (experiments using a short initial length on rats from the inactivity group), it might have been expected that the final length might have become lesser than the muscle-tendon length (= greatest possible length in the intact organism).

The greatest elongation occurred in the group where stimulation of the muscle was omitted (no stimulation). In this group it is difficult to imagine that rupture could take place at all before the muscle-tendon length had been exceeded. The separation force in the non-stimulated group was significantly lower than in the control group, indicating that the tensile strength of the muscle is influenced by the state of the muscle fibre proper, not merely by that of the sarcolemma and the connective tissue present in the muscle.

The results after exhaustion of the muscle (exhausted) were, both in respect to separation force and elongation, between the values for the no stimulation group and the control group without differing significantly from either. The frequency of tendon ruptures was the same in the exhausted and in the control group, somewhat lower in the no stimulation group. This was not significant, nor was the fact that the number of muscle ruptures increased in these 2 groups. On the other hand, muscle force was significantly greater in the exhausted and non-stimulated groups than in the control group, if the comparison included only cases in which the rupture experiment resulted in muscle rupture.

Comparison within the control group of tendon ruptures with muscle ruptures revealed that muscle force was significantly lesser in cases where the subsequent rupture experiment resulted in muscle rupture than when it resulted in tendon rupture. However, this did not mean that a weak muscle was tantamount to muscle rupture (Fig. 5, Barfred 1971 d), but that the individual muscle force contributes to deciding where the rupture occurs, tendon rupture occurring mainly when the muscle is strong.

Conclusion of the Animal Experiments

In the animal experiments it was found

that the muscle weight/tendon weight ratio fell with increasing activity,

that age-conditioned changes in the water and electrolyte content of the tendons was least marked at greatest activity level,

that inactivity entailed dehydration of muscles as well as tendons,

that the activity level influenced the tensile strength of the various tissues,

that previous damage was not necessary for obtaining tendon rupture in experiments on rats,

that tendon rupture occurred mainly in the large wild rats,

that the frequency of tendon rupture was highest after a period of inactivity,

that the risk of tendon rupture increased on oblique traction,

that the risk of tendon rupture increased when the muscle was exhausted,

that the elongation at the rupture experiment always exceeded the muscle-tendon length,

that the tensile strength and the elongation at rupture limit were least in the inactivity group,

that the muscle which had great force also had a greater separation force than the tendon, especially if it was contracted at the same time,

that the elongation was considerably greater when the muscle was not contracted,

that the histological changes which may be observed following A.R. in rats were present 24 hours post rupture and that they resembled the changes seen following human A.R.

The above results are not at variance with the knowledge which has accumulated on muscles, tendons, and bones or variations in these tissues or with some of the previously reported tendon rupture experiments (p. 28), but they are in sharp conflict with the claim that tendon rupture in a healthy tendon cannot be induced experimentally. Another argument for the degeneration theory – the histological changes in biopsies following A.R. (p. 24) – loses part of its value when considering the nature of the changes observed in the present study after rupture in rat tendons.

The experience from the animal experiments certainly does not permit the conclusion that rupture of a healthy tendon in man is possible, let alone common, but it is likely that the tendencies found in the animal experiments affect the human bone-muscle-tendon-bone chain in the same way. This applies both to the conditions connected with the rupture episode proper and, as also described in Chapter 9, to structural changes occurring in the course of a longer period.

CHAPTER 11

ANALYSIS OF RUPTURE EPISODE

The last of the three main arguments claiming that A.R. cannot take place in a healthy tendon is the feeling that the trauma described by the patient cannot have been adequate.

The movements most often reported in connection with A.R. are landing, impending fall, or push-off. The latter seems least acceptable as adequate trauma, and therefore it is reasonable to try analysing the course of this movement. By way of example, let us consider a badminton player who is to reach a ball in front of him and on his right.

The push-off is done by the left leg, while the right leg is raised, ready to take the next step. The centre of gravity of the body has to be carried forward, upward, and to the right. The movement to the right may take place partly by the aid of gravity, the centre of gravity being on the right of the supporting surface, and partly by the aid of the foot supinators, viz. apart from the m. triceps surae the m. tibialis anterior and posterior, the m. flexor digitorum longus, and the m. flexor hallucis longus. The forward movement is effected partly by flexion in the hip, extension in the knee, and dorsiflexion in the ankle, and partly by the inertia of the body, if the person is already moving forward. The upward movement is effected by extension in the hip and knee and by plantar flexion in the ankle. Here, no regard has been paid to movements of the trunk, head, arms, and the contralateral leg.

In a number of the joints the named movements are opposed, and clearly the muscle function has to be adapted to a given sequence. Apart from avoiding direct antagonism, this sequence must secure that joints close to the floor are fixed, so that they are able to determine the direction of the movement in the joint concerned.

When the body has attained a certain speed, and the movement of the knee and hip has been completed or almost completed, the heel is raised from the floor by inertia. At that time the movement is assisted by the m. triceps surae. If this function sets in too late, the movement will not be accelerated – and if it sets in too soon, it will either inhibit the effect of

the other muscles or be insufficient to raise the heel. In early push-off the heel is pressed down to the floor with a force equal to that which induces the opposed acceleration. If triceps function sets in too early, this will be at a time when the knee is extending and at the same time the heel is making resistance, so that the triceps contraction will become eccentric.

According to Arner & Lindholm (1959a) the traumatizing part of the push-off occurs after the heel has been raised and is caused by knee extension. But by then the speed is presumably so great, and acceleration so slight, that the resistance to plantar flexion in the foot cannot appreciably exceed the isometric contraction force of the triceps surae. Moreover, extension of the knee, when the ankle is either fixed by the triceps surae or is by way of being extended, will primarily result in the foot tilting down to the floor on an axis through the metatarsal heads.

In my opinion the dangerous part of push-off is to be sought rather in the above-mentioned phase in which the foot is pressed to the floor by the reaction to the great acceleration.

This pressure has been calculated as 164 kp on the basis of the magnitude of the acceleration in a filmed running step in which partial A.R. occurred (Barfred 1971 b). Since the distance from the ankle axis to the metatarsal heads is 2.7 times the distance from the ankle axis to the Achilles tendon (p. 36), the force of 164 kp has to be multiplied by 2.7 to arrive at the force that the triceps surae has to exceed in order to raise the heel, i.e. 442.8 kp.

By isometric contraction a person of this patient's height ought to be able to accomplish a torque in the ankle of about 1280 kp x cm, corresponding to a muscle force of about 290 kp in the triceps surae, but by eccentric contraction this force may be increased by 50–100 % (p. 56). Considering that the knee may be simultaneously extended by a torque of about 2000 kp, corresponding to extension of the triceps surae by a force of 670 – 1000 kp, it is realistic to believe that a tension of 442.8 kp might arise in the Achilles tendon.

In coordinated movements tensions of 375 – 535 kp have previously been found in the Achilles tendon (p. 59), and at isometric contraction tensions of up to 560 kp (p. 57). As the tensile strength of the Achilles tendon is "only" 4 – 500 kp, the problem of describing the trauma must mainly be that of explaining why A.R. does not occur more often.

One reason, as emphasized already on p. 85, is that the elongation which was needed in the rat experiments to reach the rupture limit was great compared to the elongation that could be obtained in the intact organism. Regrettably, it is not known how great this elongation would have to be in man. It is reasonable to assume that the stiffness demonstrated in the animal experiments after inactivity will occur also in persons who are out of training. Possibly, exhaustion of the muscle or the

tardive oedema after exertion may increase the stiffness, so that the elongation needed to attain the rupture limit is the least possible under these circumstances. Moreover, an oblique traction may require less elongation to initiate rupture, although the final elongation at total rupture was greater in the animal experiments (Barfred 1971 d).

The increasing number of A.R. in skiiers is presumably due to the elongation getting very great in the skiing situations, often so great that associated injuries are sustained by bones and ligaments.

Although great force is available, there are several reasons why it is not put into action. In part, both learning and motive are required to perform a maximum muscle contraction, and in part the m. triceps surae as well as the m. quadriceps femoris have to be maximally innervated at the same time. The latter does not appear to be the case (p. 58) in movements such as walking, cycling, and jumping, also not when these movements are intensified.

Disturbance of the innervation pattern requires the occurrence of a situation which necessitates an alteration – a stumble, a sudden obstacle, or a new goal to be attained. The new innervation pattern may involve a risk of over-strain and will in that case be inhibited, unless the motive is so strong that it overcomes the defence mechanisms or has arisen so quickly that the defence mechanisms do not have the time to come into action.

A contributory cause of erroneous innervation of the triceps surae may be that the patient tries to use this muscle as a knee extensor, cf. the paradoxical function described on p. 57.

In the situation described on p. 88 the patient reported that he was just running along, but scrutiny of the film disclosed that the running step differed considerably from the others in being longer, higher, and more sideways (Barfred 1971 b). In this case the history had not yielded information which permitted an evaluation of the nature of the trauma, a situation which will presumably often arise, considering how complicated the movements are and how quickly they are effected. Even top athletes are unable to give any accurate account of their movements in connection with an athletic performance (Rasch & Burke 1963).

According to what has been stated above, the problem as to whether the trauma has been adequate does not seem to be whether the force needed has been available, but rather whether the circumstances have made this force be utilized to the full in an unfortunate coordination pattern.

CHAPTER 12

CONCLUSION AND PERSPECTIVE

The problem posed in Chapter I was primarily whether experimental rupture could occur in a healthy tendon. The answer is definitely in the affirmative, both on the basis of the present studies on rat Achilles tendons and of some of the previously reported experimental investigations.

No degenerative changes were demonstrable in the Achilles tendons of the animals studied, regardless of their age and way of living. The tendons examined at varying periods after tendon rupture exhibited changes of the same nature as the changes which have been described following rupture of the human Achilles tendon, but less marked. In assessing biopsies, it must be realized that even after rupture in a healthy tendon severe changes will soon appear.

The rupture experiments revealed that it was not until a certain body weight (250–300 g) had been exceeded that tendon rupture could be induced, and that tendon ruptures were the more common the greater the animal's possibilities for activity. However, the largest number of tendon ruptures occurred in wild rats which had been caged for 3 months. The present experiments as well as studies of the literature emphasize that within the normal range there are wide limits to the mechanical and structural properties of the bone, muscle, and tendon and that these properties do not change in a parallel way. It is important to bear this in mind when assessing the nature of the aetiological factors, as there is then no need to believe that increased athletic activity – or such activity replaced by inactivity – entails degeneration arising in some way or other.

Increased activity is also an aetiological factor in a different way, as training entails an increased ability for strong innervation of the muscles, so that the resulting force approaches more the theoretical maximum. At the same time, training involves learning of a movement pattern, a wider movement repertoire.

Describing the pathogenesis in rough outline is easy (stumbling, push-off, landing), but evaluating whether the mode of origin may be

considered an adequate trauma is very difficult, int. al. because of the sudden course of events. However, a review of the literature shows that the muscle force which may be involved in the occurrence of Achilles tendon rupture is so great that it approaches or exceeds the reported ultimate tensile strength of the human Achilles tendon. With better training the ability to perform muscle contractions with greater force increases, but the magnitude of this force is also dependent upon psychological factors, which may be called motivation, although the motive need not have been conscious. It must be expected that the suddenness at which the motive arises, and the movement is performed, does not allow time for adapting the movements harmoniously and affords no possibility for mobilizing the defence mechanism, e.g. relaxation of the muscle.

The conditions which must be assumed to increase the risk of Achilles tendon rupture are previous training replaced by inactivity, an age of 30–40 years, a rapid, impulsive movement, exhausted muscles, an oblique movement, and a great elongation (short muscle-tendon unit at initiation of the movement).

The present study does not afford a basis for answering the question whether degeneration does occur in the tendon, and if so how often. Nor is it possible to prove that human subcutaneous tendon rupture can arise in an indirect trauma to the healthy tendon. But it has disproved or weakened the majority of the arguments which have been adduced in favour of the degeneration theory. The only essential reserve to rupture occurring in a healthy tendon in man is – on the basis of the animal experiments – that it is doubtful whether a sufficient possibility of elongation exists for the muscle-tendon unit. However, the animal experiments revealed circumstances which reduce the elongation needed to reach the rupture limit.

In my opinion subcutaneous rupture of the Achilles tendon must be considered, in a large number of the cases, to be due to a combination of various non-pathological factors all of which need not be present at the same time.

Perspective

This interpretation of the origin of Achilles tendon rupture may have consequences, therapeutic as well as from the point of view of insurance.

With respect to treatment, the recognition that rupture may occur in a healthy tendon combined with the regeneration ability of the tendon must justify a more conservative treatment. This may be either in the form of simple suture or perhaps a purely conservative treatment, using a plaster cast in equinus position instead of subjecting the tendon segment – in which vascularization has already been impaired at rupture – to the

further trauma of major exposure as needed by various types of tenoplasty.

When insurance is involved a considerably more liberal attitude must be recommended in deciding whether rupture of the Achilles tendon is to be considered an accident.

There is finally the question whether some prophylaxis against Achilles tendon rupture may be imagined on the basis of the above review. In the case of skiing accidents, a better design and better check on safety bindings will no doubt reduce the number of accidents. In football players lower knobs under the boots, which do not retain the foot so firmly on the ground might reduce the number of Achilles tendon ruptures as well as of other injuries to the lower limbs – at the cost of velocity in push-off, it is true.

Other prophylactic measures require more prolonged efforts which will probably be difficult to carry through. Concerning the tendon ruptures which arise on resuming sports after a period without training, there is reason to recommend a gradual, not self-overestimating re-training. According to Elliott's opinion of the added tension as a stimulus to tendon growth (p. 64), it is likely that prolonged, not particularly intense training at an early stage of life, while the tendon is still capable of increasing its girth, is the most important prophylactic measure. In that case the tendon will be able to meet the demands placed by subsequently further trained muscles. Possibly, this training should be carried out largely on rough ground in order to strengthen the tendon to withstand different directions of traction.

SUMMARY

Chapter 1

The impetus to the present study was the contrast between the clinical data concerning rupture of the Achilles tendon (age and state of training) and the experimental procedures used for clarifying its pathogenesis. On the basis of the author's previous investigations and a study of the literature, it was the main object of the present paper to discuss whether the healthy tendon can rupture on indirect violence. A secondary object was to investigate the circumstances under which the tendon is most apt to rupture, and lastly to elucidate whether prophylactic measures could be suggested on the basis of the findings.

Chapter 2

Human subcutaneous Achilles tendon rupture (A.R.) is defined as rupture in the tendon substance arising without preceding breach of continuity in the skin or subcutaneous tissue.

On the basis of descriptions by previous authors it may be established briefly that A.R. seems to be increasing in frequency and that it occurs most often in men aged 30–50 years. In general, it is stated that A.R. is not preceded by systemic or local diseases, but usually arises in connection with athletic performances. The patients are, or have been, in a state of training above average. A younger age group sustains the injury while in top training whereas the somewhat older patients sustain A.R. when out of training. In many cases the trauma does not seem adequate, but owing to its sudden occurrence it is difficult to elucidate.

After completed treatment a large number of the patients, many of whom belong to the world élite, can resume their sports.

Chapter 3

Histological findings in normal materials, in biopsies following Achilles tendon rupture, and in tendon tissue following experimental procedures are reported. Concerning the diagnosis of pre-traumatic degeneration, the results are so conflicting that the divergences cannot be due merely to

differences in the patient materials, but must relate also to deficient knowledge about the speed at which changes may arise in tendon tissue following severe trauma. The results of some of the reported experimental studies indicate that this speed may be very great.

Chapter 4

In the most commonly quoted rupture experiments on the bone-muscle-tendon-bone chain it has not proved possible to induce tendon ruptures. However, in a number of investigations it has been possible. These investigations have differed from the former in the rupture experiments, being performed on mature experimental animals, after training or using oblique traction on human tendons.

Chapter 5

The two main views (the degeneration theory and the mechanical theory) concerning the origin of Achilles tendon rupture are briefly reviewed. The former view, that the healthy tendon never ruptures, has been supported by the results of histological studies and by rupture experiments whose results have, however, not been consistent, as already mentioned. The degeneration has usually been ascribed to reduced vascularization, but tendinitis-peritendinitis, and added minor ruptures have also been mentioned as being responsible.

Adherents of the latter view, that the healthy tendon may rupture, have adduced age-conditioned stiffness, contracture (e.g. by the use of high-heeled shoes), oblique loading of the tendon, reflex defence movements, and lastly a disproportion between muscle force and tendon strength. Strange to say, no information has been sought in muscle physiology. Only one author has calculated the stress on the Achilles tendon in the course of an athletic performance and this stress proved very high.

It is mentioned that the insurance companies are adherents of the degeneration theory, although in some cases they accept A.R. as an accident.

In the theories listed only little regard has been paid to the structure and function of the tissues included in the bone-muscle-tendon-bone chain. Therefore, these three types of tissue are described in some detail in the subsequent chapters.

Chapter 6

Joint movements in relation to the Achilles tendon and the relevant lever arms are described. The twisting and interdigitation of the Achilles tendon are described and so are the special conditions at its insertion: fan-shaped spread, gradual transition from tendon to bone, and further interdigitation

of the tendon fibres. The Achilles tendon appears to be amply compensated for the stress concentrations which may occur on alterations in the direction of pull entailed by flexion-extension movements in the ankle. On the other hand, the stress can only partially be evenly distributed in the tendon on pronation-supination in the subtalar joints.

The tendon is composed of parallel collagen molecules kept together by cross links between the molecules and to the interfibrillar substance. There is evidence that the molecular ends do not reach each other, so that the strength is not conditioned by the collagen molecule, but by the named cross links. Collagen in mature tendon tissue has a very slow turnover, but the interfibrillar substance may alter in the course of a few days.

The mechanical properties of the tendon may be described partly by tensile strength and elongation at the rupture limit and partly by the shape of the length-tension curve. Tendon tissue acts as a visco-elastic material in the major part of the length-tension diagram, but with a preponderance of plastic properties at the very beginning and at the end of the curve. The tensile strength is stated to range from 5 to 10 kp/mm^2 and the elongation at rupture limit to be about 10 % of the elastic equilibrium length. In the case of the Achilles tendon this means a tensile strength of 4–500 kp and an elongation of 1–2 cm. It seems possible that physiological increases in body temperature may affect the tendon tissue, so that the tensile strength decreases and the elongation at rupture limit increases.

Chapter 7

The bones are important in tendon rupture owing to their external shape, which determines the length of the levers, and owing to their internal structure, which determines the strength of the bone. The yielding of bone is about 1/10 of that of the tendon. Relevant data on the tensile strength are not available apart from the named rupture experiments on the bone-tendon-muscle-bone chain.

Chapter 8

The gross and microscopic structure of the muscle is described in relation to function. The length-tension diagram and the force-velocity diagram are described with special emphasis on eccentric contraction during which the tension in the muscle-tendon unit may increase by 50–100 % in relation to the tension at maximum isometric contraction.

The rupture limit for the muscle has been poorly elucidated, both with respect to tensile strength and elongation at the rupture limit as well as with respect to differences in these parameters between relaxed and contracted muscle.

The voluntary, maximum isometric contraction force of the m. triceps surae has been found to be in the range 250–500 kp. Values in the same order have been found by kinesiological evaluation of the course of various movements.

EMG investigations of innervation patterns in various movements have revealed that normally the m. quadriceps femoris and the m. triceps surae are not maximally innervated at the same time. The function of pluri-articular muscles may give rise to the peculiar phenomenon that a muscle which normally functions as a flexor may act as an extensor, and this opens up a possibility of disturbance in the innervation pattern.

The possibility of achieving maximum contraction of isometric or dynamic nature depends upon training and psychological factors. Muscle training entails partly quantitative and partly qualitative changes in the muscle, but part of the effect of training is the learning which takes place in the central nervous system.

Chapter 9

By a number of examples it is emphasized how tendons, bones, and muscles may change during growth, training, inactivity, and dietary alterations, without there being any question of pathological changes.

That these changes are not parallel may be seen int. al. from the fact that the growth of tendons and muscles is stimulated by various factors and that the ability of the tendon for growth ceases at the same time as growth is completed, whereas the ability of the muscle for hypertrophy persists.

Changes of ageing after completion of growth appear to entail mainly increased stiffness, although these changes are considerably slighter than the changes occurring during growth. Training as well as wild animals' conditions of living appear to make the changes of ageing proceed more slowly.

Chapter 10

The author's experimental studies on rats, consist partly in determinations of body weight, length of muscle-tendon unit, foot length, excursion of movements, and muscle force as well as muscle and tendon weight, a number of biochemical parameters, and histological examinations before and after the rupture experiments. From an account of the first-mentioned measurements in the various groups of rats – domesticated and wild rats, rats kept in runs, and wild rats caged for 3 months – it is likely that the difference between the groups may be considered an expression of changes caused by differences in activity level.

The rupture experiments proper were performed on anaesthetized rats fastened in a material testing machine. Differences between the groups

were evaluated on the basis of a standard rupture experiment performed by quick manual elongation of the muscle-tendon unit with simultaneous electric stimulation of the muscle via the sciatic nerve. In the testing machine the calcaneus was rotated 30°, corresponding to supination of the left leg or pronation of the right leg.

Differences in the procedure of rupture experiments were investigated on large, wild rats by changing as far as possible only one factor. The factors investigated were straight/oblique traction, quick/slow traction at short or long initial length, traction when the muscle was relaxed or stimulated, and finally traction on exhausted or fresh muscle.

In the animal experiments it was found

that the muscle weight/tendon weight ratio fell with increasing activity level,

that changes of ageing in the water and electrolyte content of the tendons were least marked at the greatest activity,

that inactivity entailed dehydration of muscles as well as tendons,

that the activity level influenced the tensile strength of the various tissues,

that preceding damage was not needed to attain tendon rupture in experiments on rats,

that tendon rupture occurred mainly in the large wild rats,

that the frequency of tendon rupture was highest after a period of inactivity,

that the risk of tendon rupture increased on oblique traction,

that the risk of tendon rupture increased when the muscle was exhausted,

that the elongation at rupture always exceeded the muscle-tendon length,

that the tensile strength and elongation at the rupture limit were least in the inactivity group,

that the muscle which had great force also had greater separation force than the tendon, especially if it was contracted at the same time,

that the elongation was considerably greater when the muscle was not contracted,

that the histological changes which may be seen following A.R. in the rat were present 24 hours post-rupture and that they were reminiscent of the changes observed following human A.R.

The experience from the animal experiments certainly does not permit the conclusion that rupture in a healthy tendon in man is possible, let alone common, but it is likely that the tendencies observed in the animal experiments will be found also in the human bone-muscle-tendon-bone chain, both with respect to structural changes occurring in the course of a long period and the conditions connected with the rupture episode proper.

Chapter 11

Among the types of trauma which have been described in connection with rupture of the Achilles tendon, the push-off, in particular, is difficult to recognize as adequate trauma. The push-off, therefore, is described in some detail, partly with a view to the forces involved and partly with a view to the innervation pattern. On the basis of these considerations, the time at which the knee is extending and the heel still pressed against the ground must be regarded the time at which the traumatizing forces are at their maximum.

It is emphasized that this presupposes erroneous innervation, but in that case there seems to be sufficient muscle force to exceed the tensile strength of the tendon.

Chapter 12

From the present study it is concluded that experimental rupture can occur in healthy tendons in the rat.

None of the arguments previously adduced in favour of the degeneration theory can be attributed with much weight. The only important reserve to rupture occurring in a healthy tendon in man is – on the basis of the animal experiments – that it is doubtful whether a sufficient possibility of elongation exists for the muscle-tendon unit.

In my opinion subcutaneous rupture of the Achilles tendon must be considered, in a large number of the cases, to be due to a combination of various non-pathological factors all of which need not be present at the same time.

A consequence of this view must be a more conservative attitude in selecting the suture method (simple suture) and a recommendation to the insurance companies to assume a more liberal attitude in deciding whether Achilles tendon rupture is to be accepted as an accident.

By way of prophylaxis, it may be recommended – as has been done by previous authors – to be careful in sports performances when out of training. On the basis of the literature it seems likely that long-lasting training in early life is able to strengthen the tendon.

RESUMÉ

Kap. 1

Baggrunden for dette arbejde har været modsætningen mellem de kliniske oplysninger om achillessenerupturer (alder og træningstilstand) og tilrettelæggelsen af eksperimentelle procedurer til belysning af achillessenerupturens patogenese. På basis af egne undersøgelser og litteraturgennemgang har det været hovedformålet at tage stilling til, om den sunde sene kan briste ved indirekte vold. Dernæst at undersøge under hvilke omstændigheder senen vil være mest udsat for bristning, og sidst om man på basis heraf kan foreslå profylaktiske forholdsregler.

Kap. 2

Den humane subkutane achillesseneruptur (A.R.) defineres som bristning i senesubstansen opstået uden forudgående kontinuitetsbrud i hud og subcutis.

På basis af tidligere forfatteres beskrivelse af A.R. kan man summarisk fastslå, at A.R. synes at være tiltagende hyppig, at den forekommer hyppigst hos mænd i 30–50 års alderen. Almindeligvis angives, at A.R. ikke er forudgået af generelle eller lokale sygdomme, men som regel opstået i forbindelse med en sportspræstation. Patienterne er eller har været i en bedre træningstilstand, end tilfældet er i normalbefolkningen. En yngre aldersgruppe rammes, mens de er i toptræning, mens de ældre får A.R., når de er ude af træning. I mange tilfælde forekommer traumet ikke adækvat, men er på grund af den pludselige opståen dårligt belyst.

Efter endt behandling kan en stor del af patienterne, i mange tilfælde tilhørende verdenseliten, genoptage deres tidligere sportsgren.

Kap. 3

Resultaterne af histologiske undersøgelser af normalmateriale, af biopsier efter achillesseneruptur og af senevæv efter eksperimentelle procedurer gennemgås. Hvad angår diagnosticering af prætraumatisk degeneration er resultaterne modstridende i en sådan grad, at det ikke blot kan skyldes forskelle i patientmaterialerne, men må skyldes et manglende kendskab til

den hastighed, hvormed forandringer kan opstå i senevæv efter et svært traume. Resultaterne af nogle af de refererede eksperimentelle arbejder tyder på, at denne hastighed kan være meget stor.

Kap. 4

I de hyppigst refererede brudforsøg på knogle-muskel-sene-knogle kæden har det ikke vist sig muligt at fremkalde senebrud, men der findes dog flere arbejder, hvor dette har været muligt. Disse undersøgelser afveg fra de førstnævnte ved, at brudforsøgene var foretaget på udvoksede forsøgsdyr, efter træning eller med skæv belastning af humane sener.

Kap. 5

De 2 hovedsynspunkter (degenerationsteorien og den mekaniske teori) for opståelsen af achillesseneruptur gennemgås kort. Det ene synspunkt, at den sunde sene aldrig brister, er blevet underbygget med resultaterne af histologiske undersøgelser og af brudforsøg, der dog som omtalt ikke er éntydige. Som årsag til degeneration har man hovedsageligt angivet nedsat karforsyning, men også tendinit – peritendinit og summerede småbrud har været anført som årsag til degeneration.

Tilhængere af det andet synspunkt, at den sunde sene kan briste, fremfører dels aldersstivhed, dels kontraktur (f.eks. ved brug af høje hæle), dels skæv belastning af senen, refleksbetonede afværgebevægelser og endelig misforhold mellem muskelkraft og senestyrke. Mærkelig nok har man ikke søgt oplysninger i muskelfysiologien. Kun én forfatter har i et bevægeforløb beregnet belastning af achillessenen og fundet denne belastning meget høj.

Det nævnes, at forsikringsselskaberne er tilhængere af degenerationsteorien, selv om de i nogle tilfælde anerkender A.R. som ulykkestilfælde.

I de anførte teorier har man kun i mindre grad beskæftiget sig med struktur og funktion af de væv, der indgår i knogle-muskel-sene-knogle kæden, hvorfor man i de følgende kapitler har beskrevet disse 3 vævstyper.

Kap. 6

Ledbevægelserne i relation til achillessenen samt de relevante vægtstangsarme gennemgås. Achillessenens snoning og interdigiteren beskrives sammen med de specielle forhold ved insertionen: vifteformet udbredning, gradvis overgang fra sene til knogle og yderligere interdigiteren af senefibrene. Achillessenen synes velkompenseret over for de belastningskoncentrationer, der kunne forekomme ved de ændringer i trækretning, som fleksions-ekstensionsbevægelser i ankelleddet kan medføre. Derimod kan belastningen kun delvis fordeles jævnt i senen ved pronation-supination i de subtalare led.

Senen er sammensat af parallelle collagenmolekyler, som holdes sammen af tværbindinger mellem molekylerne og til interfibrillærsubstansen. Der er holdepunkt for, at molekylenderne ikke når hinanden, således at styrken ikke betinges af collagenmolekylet, men af de nævnte tværbindinger. Kollagenet i modent senevæv har en meget langsom omsætning, men interfibrillærsubstansen kan ændres i løbet af nogle dage.

Senens mekaniske egenskaber kan dels beskrives ved brudstyrke og forlængelse ved brudgrænse samt ved formen af længde-spændingskurven. Senevæv fungerer som et visko-eleastisk materiale i størstedelen af længde-spændingsdiagrammet, men med overvægt af plastiske egenskaber i kurvens allerførste og sidste del. Brudstyrken angives fra 5–10 kp/mm^2 og forlængelsen ved brudgrænsen til ca. 10 % af hvilelængden, for achillessenen en brudstyrke på 4–500 kp og en forlængelse på 1–2 cm. Der synes at være mulighed for, at fysiologiske stigninger i legemstemperaturen kan påvirke senevævet, således at brudstyrken falder, og forlængelsen ved brudgrænsen stiger.

Kap. 7

Knoglerne har betydning for senerupturerne ved deres ydre form, som bestemmer vægtstængernes længde samt ved deres indre struktur, der bestemmer knoglens styrke. Knoglens eftergivelighed er ca. 1/10 af senens. Relevante oplysninger om brudstyrken findes ikke udover de tidligere nævnte brudforsøg på knogle-sene-muskel-knogle kæden.

Kap. 8

Muskelens makroskopiske og mikroskopiske struktur beskrives i relation til funktionen. Længde-spændingsdiagrammet og kraft-hastighedsdiagrammet gennemgås med særlig vægt på den excentriske kontraktion, under hvilken spændingen i muskel-seneapparatet kan stige med 50–100 % i forhold til tensionen ved den maksimale isometriske kontraktion.

Brudgrænsen for muskelen er dårligt belyst både hvad angår brudstyrke og forlængelse ved brudgrænsen og hvad angår forskelle i disse parametre mellem slap og spændt muskel.

Den volontære, maksimale, isometriske kontraktionskraft for m. triceps surae er fundet af størrelsesordenen 250–500 kp. Tal af samme størrelsesorden er fundet ved kinesiologisk vurdering af forskellige bevægeforløb.

Undersøgelser ved hjælp af EMG af innervationsmønstre ved forskellige bevægelser har vist, at m. quadriceps femoris og m. triceps surae ikke normalt er maksimalt innerverede samtidigt. De flerleddede musklers funktion kan give anledning til den ejendommelighed, at en muskel, der normalt fungerer som flexor kan virke som ekstensor, hvilket åbner mulighed for forstyrrelse af innervationsmønstret.

Muligheden for at præstere en maksimal kontraktion af isometrisk eller dynamisk karakter er afhængig af træning og psykologiske faktorer. Muskeltræning medfører dels kvantitative dels kvalitative forandringer i muskelen, men en del af træningsvirkningen er den indlæring, der sker i centralnervesystemet.

Kap. 9

Det understreges gennem en lang række eksempler, hvorledes sener, knogler og muskler under opvækst, træning, inaktivitet og ændringer i kosten kan ændres, uden at man kan tale om patologiske forandringer.

At forandringerne ikke forløber parallelt ses blandt andet af, at seners og musklers vækst stimuleres af forskellige faktorer, og at senens vækstevne samtidig med vækstens ophør forsvinder, mens muskelens evne til hypertrofi fortsætter.

Aldersforandringerne efter vækstens afslutning synes særlig at medføre øget stivhed, om end disse ændringer er væsentlig mindre end ændringerne under opvæksten. Både træning og de vilde dyrs livsform synes at medføre, at aldersforandringerne sker langsommere.

Kap. 10

Forf.'s forsøgsrække, der er foretaget på rotter, består dels af en række målinger af dyrevægt, cruslængde, fodlængde, bevægeudslag og muskelkraft samt muskel- og senevægt, en række biokemiske parametre samt histologiske undersøgelser før og efter brudforsøg. Hertil kommer de egentlige brudforsøg. En gennemgang af de først omtalte målinger i de forskellige dyregrupper, tamme og vilde rotter, løbegårdsrotter og vilde rotter holdt i bur i 3 måneder sandsynliggør, at man kan betragte forskellene mellem grupperne som udtryk for forandringer fremkaldt af forskelligt aktivitetsniveau.

Selve brudforsøgene er alle foretaget på bedøvede rotter efter indspænding i en materialprøvemaskine. Forskelle mellem grupperne er vurderet på grundlag af et standardbrudforsøg foretaget med en hurtig, manuel forlængelse af muskel-sene gruppen under samtidig elektrisk stimulation af muskelen via n. isciadicus. Calcaneus var ved indspændingen drejet 30° svarende til en supination på venstre ben eller en pronation på højre ben.

Forskelle i brudforsøgsproceduren blev undersøgt på store, vilde rotter ved, så vidt muligt, kun at ændre én faktor. De faktorer, der blev undersøgt er lige/skævt træk, hurtig/langsomt træk, træk ved kort eller lang udgangslængde, træk når muskelen var slap eller stimuleret og endelig træk på udtrættet eller frisk muskel.

Man har i dyreforsøgene fundet,
at muskel/sene ratio faldt med stigende aktivitet,
at aldersforandringer i senernes vand- og elektrolytindhold var mindst udtalt ved størst aktivitet,
at inaktivitet medførte dehydrering såvel af muskler som af sener,
at aktivitetsniveau'et indvirkede på de forskellige vævs brudstyrke,
at forudgående beskadigelse ikke var nødvendig for at opnå senebrud i forsøg på rotter,
at senebrud fortrinsvis optrådte hos de store vilde rotter,
at senebrudsfrekvensen var størst efter en periode med inaktivitet,
at risikoen for senebrud øgedes ved skæv belastning,
at risikoen for senebrud øgedes, når muskelen var udtrættet,
at forlængelsen ved brudforsøget altid overskred muskel-senelængden,
at brudstyrken og forlængelsen ved brudgrænsen var mindst i inaktivitetsgruppen,
at den muskel, der havde stor kraft også havde større brudstyrke end senen, særlig hvis den samtidig var kontraheret,
at forlængelsen var væsentlig større, når muskelen ikke var kontraheret,
at de histologiske forandringer, man kan se efter A.R. hos rotter, allerede opstod efter 1 døgn, og at de mindede om de forandringer, der ses efter human A.R.

De erfaringer, man har fra dyreforsøgene, betyder ingenlunde, at man kan slutte, at ruptur i sund sene hos mennesket er mulig endsige almindeligt forekommende, men det er sandsynligt, at de tendenser, man har fundet i dyreforsøgene, vil påvirke menneskets knogle-muskel-sene kæde i samme retning, både hvad angår strukturelle ændringer på længere sigt og hvad angår forholdene i forbindelse med selve brudepisoden.

Kap. 11

Af de traumetyper, som beskrives i forbindelse med achillesseneruptur, er specielt afsættet svært at erkende som adækvat traume, hvorfor dette gennemgås nøjere, dels med henblik på de deri indgående kræfter, dels med henblik på innervationsmønster. Ud fra disse betragtninger må man anse tidspunktet, hvor knæet er under strækning, og hvor hælen endnu er presset mod underlaget, som det tidspunkt, hvor de skadevoldende kræfter er størst.

Det understreges, at dette forudsætter fejlinnervation, men at der i så tilfælde synes at være tilstrækkelig muskelkraft til at overskride brudgrænsen.

Konklusionen af dette arbejde er, at eksperimentel seneruptur i sund sene er mulig på rotter.

Ingen af de holdepunkter, der tidligere har været fremført for degenerationsteorien kan tillægges større værdi. Det eneste væsentlige forbehold over for, at ruptur i sund sene kan forekomme hos mennesker er, at det på basis af dyreforsøgene er tvivlsomt, om der er tilstrækkelig forlængelsesmulighed for muskel-senegruppen.

Det er forf.'s opfattelse, at den subkutane achillesseneruptur i en stor del af tilfældene må betragtes som forårsaget af en lang række ikke patologiske forhold i en kombination, der ikke behøver at være fuldstændig.

Konsekvensen af denne opfattelse er en mere konservativ indstilling over for valg af suturmetode (simpel sutur) og en anbefaling til forsikringsselskaberne om at anlægge en mere liberal holdning ved afgørelse af, om achillesseneruptur skal betragtes som ulykkestilfælde.

Profylaktisk anbefales, som tidligere forfattere har gjort det, at være varsom med sportsudøvelse, når man er ude af træning. Desuden er det på grundlag af litteraturgennemgang sandsynligt, at en langvarig træning tidligt under opvæksten særlig vil kunne styrke senen.

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SUBJECT INDEX