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Richting: 2de masterjaar in de biomedische wetenschappen - klinische moleculaire wetenschappen
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Datum: 11.12.2009

Peroneal neuropathy after weight loss: ultrasonographic and histological characterization of the n. fibularis communis

Eline Cardinaels

promotor :
Prof. Dr. Frank WEYNS, Prof. Dr. Herlinde VANORMELINGEN

Eindverhandeling voorgedragen tot het bekomen van de graad master in de biomedische wetenschappen klinische moleculaire wetenschappen

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Table of content

List of Abbreviations

BMI:	Body mass index
cm:	Centimeter
CNS:	Central nervous system
e.g.:	Exempli gratia
EMG:	Electromyogram
FOV:	Field of view
kg:	Kilogram
kHz:	Kilohertz
L:	Lumbal
m.:	*musculus*
mm:	Millimeter
MRI:	Magnetic resonance imaging
n.:	*nervus*
PNS:	Peripheral nervous system
r.:	*ramus*
S:	Sacral
TE:	Echo time
TR:	Repetition time
µm:	Micrometer
US:	Ultrasonography
ZOL:	Ziekenhuis Oost-Limburg

Abstract

Introduction

Peroneal neuropathy is caused by the compression of *n. fibularis communis*, and is associated with weight loss. This condition can be treated by either conservative treatments or surgical decompression. Because it is not yet known how weight loss induces peroneal neuropathy, the best treatment option has yet to be elucidated. It is for this reason important to gain more insight into the pathophysiology of this problem. Basic knowledge of the microanatomy is essential to unravel the development of nerve compression syndromes.

Materials and methods:

In this study the microanatomy of *n. fibularis communis* is examined using in vivo ultrasonographic and histological techniques. During an epidemiological study, the prevalence of peroneal neuropathy after weight loss is addressed in patients, who underwent gastric banding surgery.

Results:

By in vivo ultrasonography, differences in the structure of *n. fibularis communis* can be detected in patients suffering from peroneal neuropathy. On the ultrasound images, *n. fibularis communis* of obese persons appeared thicker than in thin persons. In the histological study, *n. fibularis communis* of thick specimens showed a tendency of containing more intraneural fat in comparison to thin specimens. The epidemiological study demonstrated that 3% of the patients, who underwent gastric banding surgery, developed symptoms of peroneal neuropathy.

Conclusion

From this study we can conclude that in vivo ultrasonography can contribute to the diagnosis of peroneal neuropathy. In obese people *n. fibularis communis* appeared thicker, possibly due to an increase in intraneural fat. It is estimated that weight loss could result in the disappearance of intraneural and subcutaneous adipose tissue. Although further research is necessary, this investigation suggests a possible role for the adipose tissue in the development of peroneal neuropathy. It must be considered that it is likely that multiple factors play a role in the pathophysiology of this condition.

Acknowledgments

This thesis indicates the end of a period of five years of the study Clinical Molecular Life Sciences. For my senior internship during my final master year, I chose the subject "Peroneal neuropathy after weight loss" at the department of Neurosurgery at the Ziekenhuis Oost-Limburg and the department of Basic Medical Sciences at Hasselt University. With great pleasure I look back to this period. In this preface I wish to thank everybody who has contributed directly or indirectly to this work.

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Eline

1. Introduction

Peroneal neuropathy is the most common mononeuropathy in the lower limbs, accounting for 15% of all the mononeuropathies in adults. [1, 2] In most cases peroneal neuropathy results in foot drop. This condition is characterized by the patient's inability to dorsiflex (raise) the foot and ankle, seriously affecting the normal gait. Peroneal neuropathy is caused by the compression or entrapment of *n. fibularis communis* or the (common) peroneal nerve. Predominantly the origin of this nerve compression is known, such as direct trauma to the fibular head or intrinsic masses exerting pressure on the nerve. [3] In other patients no evidence of mechanical compression was present. In these cases weight loss was indicated as a risk factor for the development of peroneal neuropathy. [4] However, it remains controversial how weight loss is associated with this neuromuscular disorder. It is thought that metabolic disturbances could play a significant role in the development of peroneal nerve entrapment. [5] Examining *n. fibularis communis* and the surrounding structures is important in understanding the pathophysiology of this condition. In this study the microstructure of *n. fibularis communis* is visualized, using ultrasonographic and histological techniques. Peroneal neuropathy is a rare condition but the extent of the problem after weight loss is to the best of our knowledge unknown. During an epidemiological study, the prevalence of peroneal neuropathy is evaluated in patients, who underwent gastric banding surgery.

1.1 Microstructure of the peripheral nerves

N. fibularis communis is a peripheral nerve located in the lower leg. Peripheral nerves serve as a communication network between the central nervous system (CNS) and every part of the body. There are two types of peripheral nerves: sensory and motor nerves. Sensory nerves transfer information from the periphery to the CNS, while in motor nerves the information is transferred from the CNS to the periphery. A peripheral nerve is composed of numerous myelinated or unmyelinated axons. The axons are bundled together in fascicles and are surrounded by three different layers of connective tissue. The first layer is called the endoneurium, which surrounds each axon. The perineurium, surrounding the fascicles, is the second layer of connective tissue. Finally, different fascicles are enclosed by the epineurium. Adipose tissue and a rich blood supply are also surrounded by this outermost layer of connective tissue. [6, 7]*(Figure 1.1)*

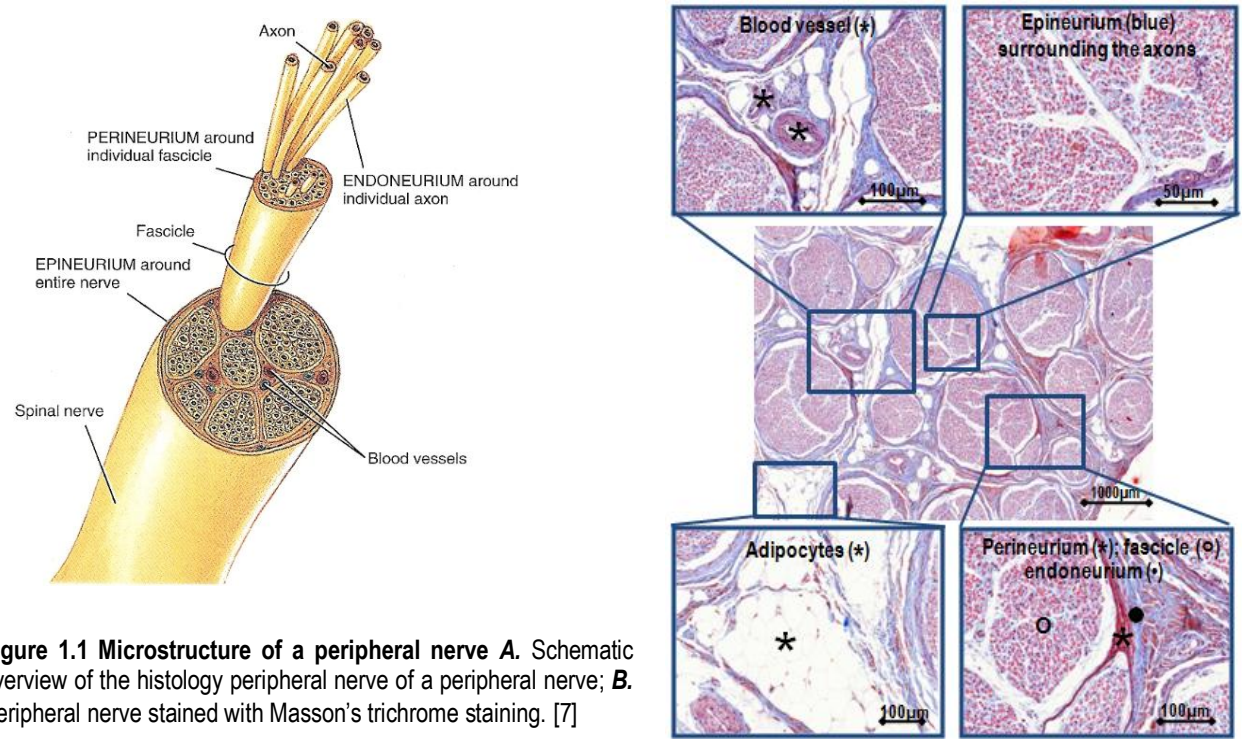
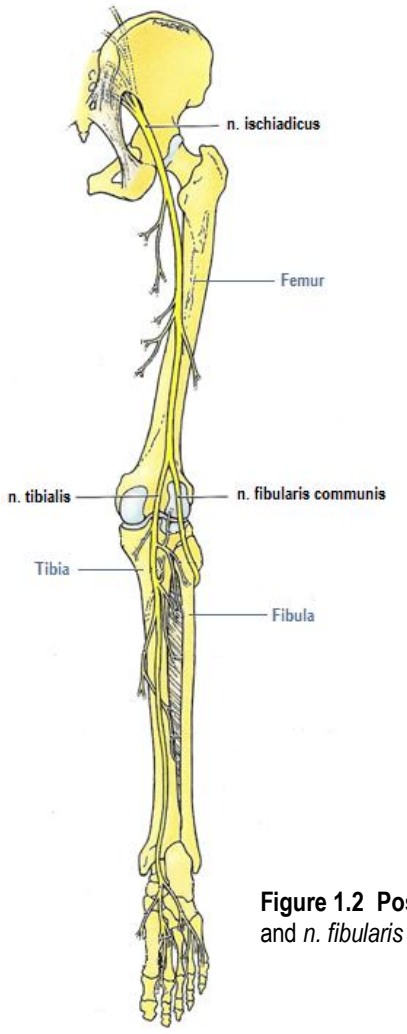


Figure 1.1 Microstructure of a peripheral nerve *A.* Schematic overview of the histology peripheral nerve of a peripheral nerve; ***B.*** Peripheral nerve stained with Masson's trichrome staining. [7]

1.2 Anatomy of the lower limbs



The motor and sensory nerves of the lower limbs are provided by the sacral plexus, with exception of the posterior and medial aspect of the thigh. The sacral plexus is formed by the spinal cord segments from L4 to S4. The largest nerve originating from the sacral plexus is *n. ischiadicus*. *N. ischiadicus* originates from the posterior divisions of the L4, L5 and S1 to S3 nerve roots and bifurcates into *n. fibularis communis* and *n. tibialis* at the posterior aspect of the thigh. *N. fibularis communis* descends obliquely along the lateral side of the *fossa poplitea*, at the back of the knee, to the head of the fibula. *(Figure 1.2)*[8, 9] More distal, *n. fibularis communis* gives off two cutaneous branches innervating the skin and the upper lateral leg, *n. cutaneus surae lateralis* and *r. communicans fibularis.*

Figure 1.2 Posterior aspect of the lower leg: *n. ischiadicus* bifurcates into *n. tibialis* and *n. fibularis communis*. [8]

The nerve winds around the neck of the fibula. At this location *n. fibularis communis* enters a "fibular tunnel" or "fibular arch".[8, 10, 11] The fibular arch is an osteofibrous tunnel, the floor of the tunnel consists of fibular bone and the roof is comprised of muscle fibers and fibrous tissue (aponeurosis) derived from two muscles: *m. peroneus longus* and *m. soleus*. [12, 13] *(Figure 1.3)* Near the fibular tunnel, *n. fibularis communis* bifurcates into its final branches: *n. fibularis superficialis*, a superficial branch, and *n. fibularis profundus*, a deep branch. *(Figure 1.4)*

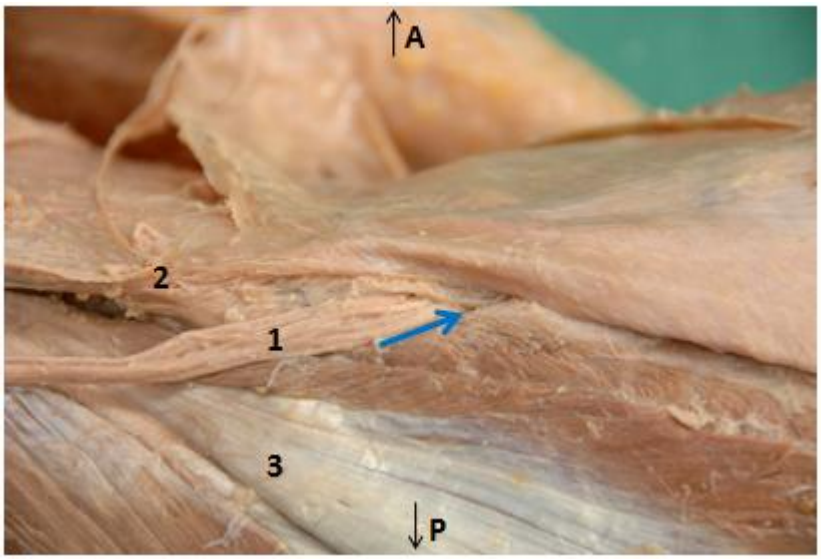


Figure 1.3 Fibular tunnel located on the right lower leg. This figure shows *n. fibularis communis (1)* entering the fibular tunnel (blue arrow); tendons of *m. biceps femoris* (2) and the postior muscles (3) (*m. soleus* and *m. gastrocnemius*) are also visible. (A=Anterior; P=Posterior)

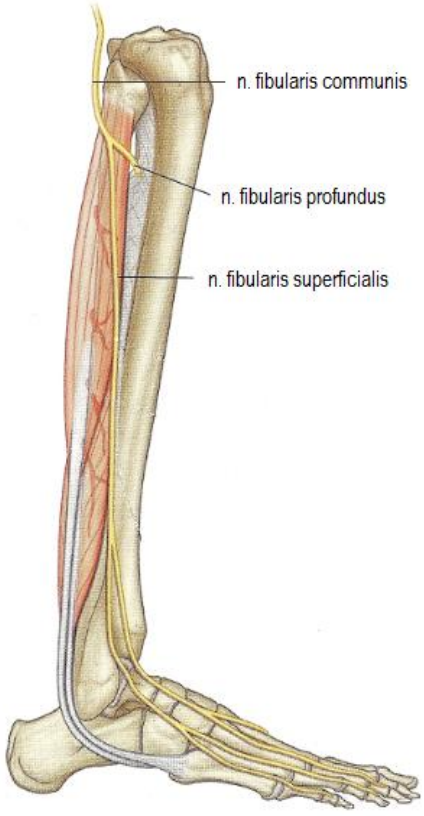


Figure 1.4 Lateral aspect of the right lower leg. At the fibular head *n. fibularis communis* enters the fibular tunnel and bifurcates into its final branches: *n. fibularis profundus* and *n. fibularis superficialis*. [8]

The skin of the lateral lower leg is chiefly innervated by *n. fibularis superficialis.* More specifically, this nerve innervates the skin of the lower two thirds of the lateral aspect of the leg and the dorsum of the foot. *N. fibularis superficialis* also sends motor branches to *m. peroneus longus* and *m. peroneus brevis*, both ankle evertors *(Figure 1.5a, Supplement I)* ***N****. fibularis profundus* is primarily a motor nerve. This nerve innervates the anterior muscles of the lower leg, which control foot dorsiflexion and toe extension. *(Supplement I)* This nerve finally terminates into a cutaneous branch that innervates the skin between the first and second toes. *(Figure 1.5)* [11]

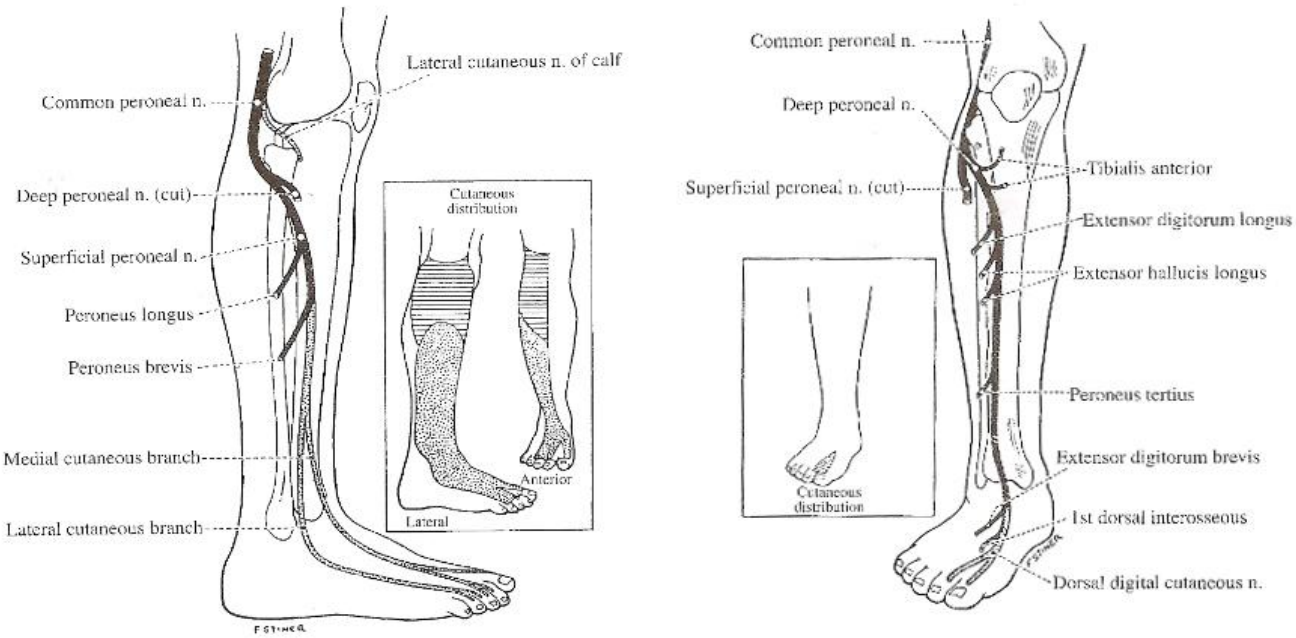


Figure 1.5 Terminal branches and cutaneous innervations of the *n. fibularis superficialis* (***A***), innervating the lateral lower leg, and the *n. fibularis profundus* (***B***), predominantly having a motor function. [11]

1.3 Peroneal neuropathy

Peripheral neuropathy is a condition describing damage to the peripheral nervous system (PNS), due to the entrapment or compression of the peripheral nerve when passing through confined spaces, like for example an osteofibrous tunnel. Because osteofibrous tunnels are narrow anatomic passages, nerves are more vulnerable to compression in these tunnels than at other points along their course. [13, 14] Peroneal neuropathy is caused by the entrapment of *n. fibularis communis*. Peroneal nerve entrapment can occur anywhere along its course, but is most commonly found at the fibular tunnel. [14, 15] The nerve is also sensitive to compression at the fibular head and neck because of its superficial course, only covered by

skin and subcutaneous tissue. Both terminal branches of *n. fibularis communis* may be impaired, but *n. fibularis superficialis* is usually less involved than *n. fibularis profundus*.[16]

1.3.1 Cause of peroneal neuropathy

Over the last decade it has been documented that peroneal neuropathy is a consequence of compression of the nerve at the level of the head of the fibula. Nerve compression can be caused by direct trauma to the fibular head or intrinsic masses, such as ganglion cysts. [3] Squatting and crossing the legs for a long time are also prevalent causes of peroneal nerve compression. [17-19].

However, not every case of peroneal neuropathy can be explained by this theory.[20, 21] In the cases where obvious causes were absent, the speculation has risen that weight loss is associated with the pathophysiology of peroneal neuropathy. Sherman and Easton describe seven patients with peroneal nerve palsy, with a mean weight loss of 19 kg over a period of four to 15 months. Weight reduction was the only obvious causal factor present in these patients. [4] A study, conducted by Sotaniemi, reported ten healthy patients, who developed peroneal neuropathy while they were on a weight reducing diet. [22] The amount of weight that was lost varied from 16 to 44 kg by the time complications appeared. Particular attention was paid at the patient's sitting and lying postures. However no history of habitual leg crossing or other postures, that might have provoked compression of *n. fibularis communis*, were found in these patients. For this reason he concluded that metabolic disturbances, due to slimming, may be involved in the development of peroneal nerve palsy in these patients. Other authors also point towards the contribution of a nutritional aetiology in peroneal neuropathy after weight loss. Evidence of a link between anorexia nervosa and peroneal neuropathy, indicating a direct relationship between weight reduction and peroneal neuropathy, has been frequently described. [23, 24] Peroneal neuropathy was prevalent in cachectic cancer patients. [25, 26] Peroneal nerve palsy was observed after weight loss, due to gastric partitioning for morbid obesity, and has been linked to metabolic disorders, i.e. diabetes mellitus, alcoholism, vitamin B depletion. [27-29]

How weight loss is associated with peroneal neuropathy has not been fully elucidated. The aetiology of peroneal neuropathy, as a consequence of weight reduction, may be multifactorial. As observed in diabetic neuropathy, it is hypothesized that although mechanical factors are important, vascular factors and metabolic changes also play significant roles in the development of peroneal neuropathy after weight loss. From a biochemical point of view, diabetes and weight loss are similar conditions. An example of metabolic factors

related to the development of diabetic neuropathy is the conversion of excess intracellular glucose to sorbitol. The excess of trapped intracellular sorbitol results in hypertonicity which creates an osmotic gradient into the cell. This osmotic gradient leads to cellular swelling and subsequently microvascular damage, resulting in the formation of intraneural oedema and ischemia.[30] The vascularization of the nerve could play an important role as well in the development of peroneal neuropathy. In the past, several anatomical examinations of *n. fibularis communis* were conducted at Hasselt University, at the department of Basic Medical Sciences. [31-33] These studies indicated that *n. fibularis communis* has a limited blood supply. Distally, *n. fibularis profundus* had a minor vascularization in comparison to *n. fibularis superficialis*. Nerves with a limited blood supply are more prone to ischemia than when a higher vascularization is present, explaining the fallout of *n. fibularis communis* in peroneal neuropathy and the frequent involvement of *n. fibularis profundus* in peroneal neuropathy. [31]

1.3.2 Symptoms

Peroneal neuropathy can cause sensory and motor symptoms. *N. fibularis profundus*, which predominantly has a motor function, is usually more affected than the sensory *n. fibularis superficialis*. [16, 34] Therefore motor symptoms, like paresis of the dorsiflexors and evertors of the foot and ankle, are predominantly present in patients suffering from peroneal neuropathy. [11] When the dorsiflexors and evertors of the foot and ankle are affected, the patients are unable to point the toes toward the body (dorsiflexion) or move the foot at the ankle outward (eversion), also known as foot drop. Walking becomes difficult and is characterized by lifting the foot higher than usual, to overcome the problem of dragging the foot on the ground. As a consequence, this condition will lead to functional gait impairment. In some cases, the sensory nerve is also affected. These patients present symptoms like numbness of the dorsum of the foot and the lower lateral leg. Pain is rarely a symptom in peroneal neuropathy. The onset of symptoms can be acute or chronic. When the peroneal lesion occurred quickly, in a few hours or in one day, it is referred to as acute peroneal neuropathy. When the onset of symptoms is recurrent and long-lasting, it is referred to as chronic neuropathy.

1.3.3 Diagnosis

A first phase in diagnosing peroneal neuropathy is an anamnesis. The patients are questioned about the onset of their symptoms and the likely causes such as remote or recent trauma, habitual leg crossing, etc.

Motor and sensory function is tested in a clinical examination. A compressed sensory nerve can be detected by performing Tinel's sign. When gently tapping the compressed nerve results in a tingling sensation, the Tinel's sign is found positive. Motor function, more specifically the ankle and toe dorsiflexion, can also be significantly affected in peroneal neuropathy. The strength of each muscle, innervated by *n. fibularis communis* and summarized in supplement I, can be tested using a manual muscle strength test. In this test, the patient is asked to contract the muscle against a produced resistance. The results can be scored using the medical research council scale of 0, no contraction possible, to 5, a normal contraction. [5]

As a supplement to clinical examination, electrodiagnostic assessment is considered. These evaluations can contribute greatly to the diagnosis and management of entrapment neuropathies and can help to determine the exact location of the problem. The electrodiagnostic examination consists of two main parts: a needle electrode examination and a nerve conduction study. Peripheral neuropathy can lead to two major types of peripheral nerve injury. The first type is axon degeneration or axon loss, which is detected by needle electrode examination. In needle electromyograms (EMG), the physiological characteristics of muscles during rest and contraction are evaluated. An electrode is placed within the skeletal muscle to record the summed action potentials of the skeletal muscle fibers of a muscle unit. These muscle unit action potentials are evaluated in order to assess axon loss. [35] The second type of injury is segmental demyelination, which is a focal conduction disturbance along an intact axon. This can be detected by nerve conduction studies. During this evaluation, the nerve is stimulated electrically and the evoked responses are measured using electrodes. These responses are called compound muscle action potentials. The time it takes from stimulation to generation of the action potential is called the conduction speed. If the conduction speed is abnormal, segmental demyelination occurred. [36]

The compression of *n. fibularis communis* can be visualized by magnetic resonance imaging (MRI), which is a valuable tool in the diagnosis of peripheral nerve lesions. The MR images can play an important role in helping to define the site and aetiology of nerve compression. The images are capable of showing changes in the nerves themselves. In addition, MRI can

detect space-occupying lesions along the nerve passage and reveal the presence of denervated muscles. [3, 14, 37]

1.3.4 Treatment

Peroneal neuropathy can be treated by either conservative or surgical treatment. Conservative or non-surgical treatment options are usually considered first. These include for example the avoidance of further pressure on the nerve, improved nutrition with sometimes supplementation of the diet with vitamins. The use of ankle-foot orthotics (AFO) is applied to support the foot and help the patient during the human gait. Another example of conservative treatment is physiotherapy, that helps strengthen the muscles. [38] On the opposite, peroneal neuropathy can also be treated with surgical decompression. During this procedure, the fascia or the roof of the fibular tunnel is cut, so that the nerve is released from compression. Because peroneal neuropathy is a complex condition, it remains controversial what the best treatment option is. Conservative treatments allow the nerve to spontaneously repair itself. In literature, it has been reported that spontaneous repair could take up to 18 to 24 months and may be incomplete, leading to permanent nerve damage. [39] It has been described that surgical treatment has good outcomes if the period between the onset of the symptoms and the surgery is not too long. [40, 41] Therefore, most authors propose a waiting period of 3 to 4 months for spontaneous recovery before surgical decompression is considered. [39, 42]

1.4 Problem situation

Previous clinical studies have shown that a surprisingly large group of patients (approximately 15% - 20%) suffering from peroneal neuropathy, developed this condition after weight reduction. [1, 22, 28, 43] The pathophysiology of peroneal neuropathy after weight reduction has yet to be unravelled. It is for this reason important to gain more insight into this problem. In literature, it is estimated that more than one factor may be involved in the aetiology of this condition. These authors suggest that next to the mechanical compression of *n. fibularis communis*, the metabolic changes, due to weight loss, could play an important role during in peroneal neuropathy.

Basic knowledge on *n. fibularis communis* and the surrounding structures is important in understanding the pathophysiology of this condition. In this study we therefore explore the internal structure of *n. fibularis communis*, by visualization with ultrasonographic and histological techniques. During the in vivo ultrasonographic explorations, different morphometrical characteristics (e.g. transverse cross-sectional area of the nerve, transverse

cross-sectional area of the fibular tunnel) of the nerve are measured. In literature, peroneal nerve entrapment is the only neuropathy linked with weight reduction. Because weight loss leads to a reduction of adipose tissue, our attention is focussed on the intraneural adipose tissue of *n. fibularis communis*. Another aim is to identify relationships between the measured characteristics of the nerve and the adipose tissue, present at different levels throughout the body.

To the best of our knowledge, the prevalence of peroneal neuropathy is unknown. In an epidemiological study, we try to evaluate the extent of this problem in patients, who underwent gastric banding surgery. Adjustable gastric banding is a surgical operation where a hollow plastic band is inserted around the stomach. Band constriction has a restrictive effect on gastric filling capacity and forces the patient to lower his food intake. As a consequence of a lower food intake, the patients will lose weight. The patients were used for this study because they undergo rapid and marked weight loss, indicated as a risk factor for the development of peroneal neuropathy.

2. Materials and methods

In this study the microanatomy of *n. fibularis communis* is examined. Using in vivo ultrasonography (US), the morphometrical properties of *n. fibularis communis* were assessed in a control sample and in four patients suffering from peroneal neuropathy. The intraneural fat, present in the specimen of *n. fibularis communis*, is explored using Masson's trichrome staining. During an epidemiological study, the prevalence of peroneal neuropathy after weight loss is addressed in patients, who underwent gastric banding.

2.1 In vivo ultrasonographic characterization of *n. fibularis communis*

N. fibularis communis was visualized using US at the department of Radiology at Ziekenhuis Oost-Limburg (ZOL) in Genk. *N. fibularis communis* was visualized in 50 controls as well as four patients, diagnosed with peroneal neuropathy after weight loss.

2.1.1 Controls

A group of 50 healthy volunteers, with no signs of peroneal neuropathy, were asked to participate to this study. The following data were collected for each participant: age, gender, weight and height. Body Mass Index (BMI) was calculated using the simplified formula:

$$BMI = \frac{weight\ (kg)}{\left[height\ (cm)\right]^2}$$

2.1.2 Peroneal neuropathy patients

In four patients, suffering from peroneal neuropathy, the lower leg was examined using ultrasonography. From these four patients, two patients were suffering from acute peroneal neuropathy, one patient was diagnosed with chronic peroneal neuropathy and another with bilateral peroneal neuropathy.

A 12-year old boy (case 1) was referred to the department of Neurosurgery at ZOL after being involved in a car accident. After this accident, the patient slipped into a prolonged coma. During this coma he lost more than 10% of his bodyweight, subsequently leading to atrophy of *n. fibularis communis*. As a result, the patient revealed a complete paresis of the foot dorsiflexion and eversion and a diminished sensation on the common peroneal nerve distribution. The diagnosis of foot drop was confirmed with MRI and electrodiagnostic examination. The patient was treated with surgical decompression, post-operatively followed by physical therapy and electrical stimulation. The patient had a complete recovery.

A year prior to admission to the department of Neurosurgery at ZOL, a 52 year old man (case 2) developed an intermittent dorsiflexion paresis of the left foot. During this period, he experienced difficulties during walking and working. Clinical investigation showed a partial paresis of the dorsiflexors and a positive sign of Tinel at the left side of the leg. This indicated a chronic compression neuropathy of the left *n. fibularis communis* resulting in a reduced motor and sensory function. This diagnosis was confirmed by MR imaging and electrodiagnostic examination. The patient was treated with surgery, leading to a complete decompression of *n. fibularis communis* and its two final branches. Post-operatively the patient underwent electrotherapy and physiotherapy to promote the dorsiflexion of the left foot. At six weeks, the post-operative evolution was positive.

A 46 year old man (case 3) lost within several months 10% of his body weight. After this acute weight loss, the patient developed a sudden paresis of the left foot dorsiflexors, accompanied with a sensory loss along the dorsum of the foot. Two months later the patient was referred to ZOL. Electrodiagnostic examination showed an acute peroneal neuropathy involving *n. fibularis communis* and its two final branches. On MRI, an intraneural oedema was present in the nerve. The patient was treated with decompression surgery. Post-operatively the patient underwent electrotherapy and physiotherapy to promote the dorsiflexion of the left foot. By the time of the last follow-up, the patient had a complete recovery.

A 46 year old woman (case 4) presenting with bilateral peroneal nerve entrapment after losing 24% of her bodyweight during a period of three months. On MRI, the peroneal nerve injury was visible on both sides. The nerve compression was confirmed by MRI and EMG. The MRI of the left side revealed a minor intraneural oedema. The patient was treated with surgical decompression surgery, resulting in a complete decompression of *n. fibularis communis* and its two final branches. Post-operatively the patient underwent electrotherapy and physiotherapy to promote the dorsiflexion of the left foot.

2.1.3 Technique

In US high frequency sound waves (above 20 kHz), inaudible to human ears are used. These sound waves have shorter wavelengths and can be formed into a narrow beam. Ultrasound waves are produced by a transducer, also called an ultrasonic sensor, that converts an electrical signal into an ultrasound beam. *(Figure 2.1)*These sound waves pass through the body part of interest, up to a point where they are reflected by the tissue. This reflected sound wave is called an “echo”. The transducer evaluates the echo that is received and transforms it

into an electrical signal. The same ultrasonographic sensor can transform electrical signals into sound energy and vice versa. In the transducers, pressure is generated when electrical energy is applied to the disc. This pressure wave causes the disc to contract (or to expand), and subsequently create sound waves. The reflected sound waves or echoes can reverse the pressure wave. When the pressure is reversed, the voltage is reversed as well. [44]

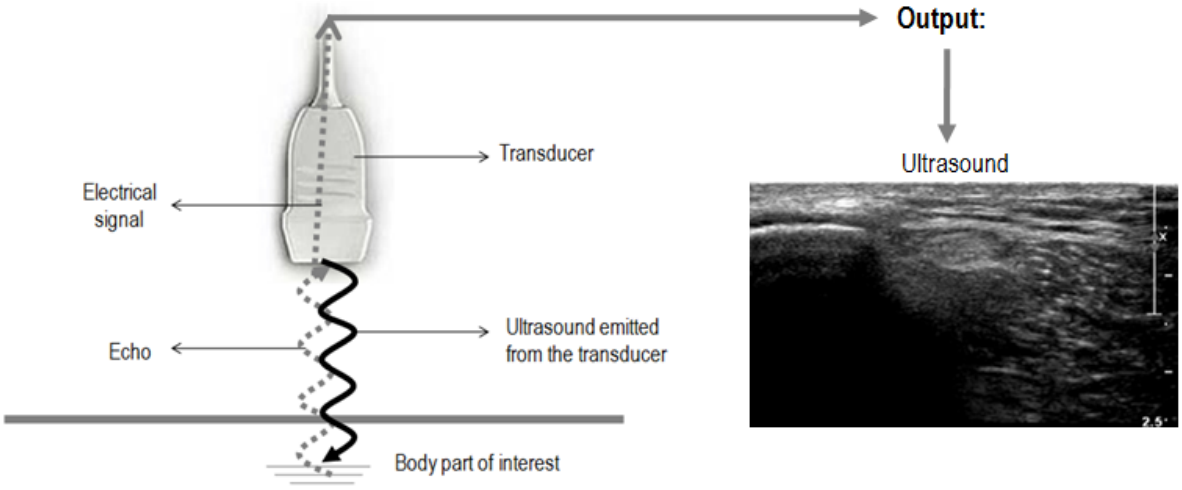


Figure 2.1. Basic principle of ultrasound imaging: The transducer emits ultrasounds at the body part of interest. A reflected ultrasound is called an "echo". The transducer transforms the received echoes into an electrical signal. These electrical signals can be converted to an ultrasound image.

Different structures can be visualized through the difference in echogenicity or the ability of the structure to create an echo that can be detected. Water is referred to as hypoechoic, as a consequence water containing structures, such as muscles, appear darker on the ultrasound. Adipose tissue has a higher echogenicity than water and is therefore referred to as hyperechoic, as a consequence structures with a higher adipose tissue content appear brighter on the ultrasound. [45]

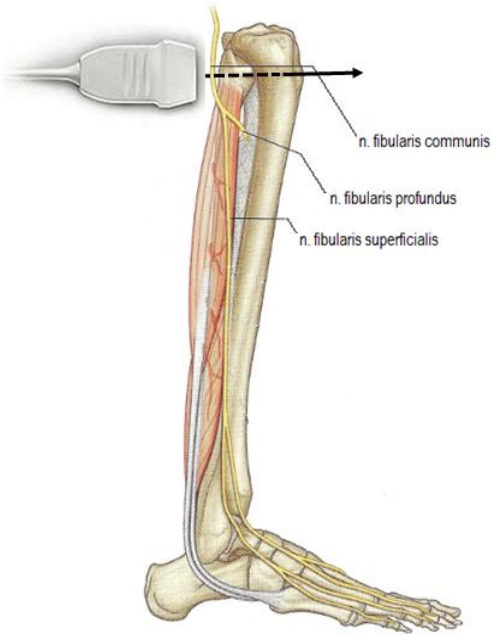


Figure 2.2 The ultrasound images were taken at the level of the fibular head.

N. fibularis communis was visualized bilaterally in each patient using US with a 12,5 Mhz transducer (iU22, Philips Medical Systems, Bothell, Washington). The ultrasound images were taken at the position where the nerve has the closest proximity to the fibular head. The legs of each person were positioned in a fully stretched position with the foot turned inward, so that the fibular head is reached easily. (*Figure 2.2*)

The morphometrical characteristics of *n. fibularis communis*, illustrated in figure 2.3, were assessed by the same observer. The first examined characteristic is the subcutaneous fat, measured superficial to the nerve. Also the transverse cross-sectional area of the nerve was determined, comprised of a long and short transverse diameter. To describe the fibular tunnel, the distance between the fibular bone and *n. fibularis communis* and the distance between the fibular bone and the *m. peroneus longus* was measured. The transverse cross-sectional area of the fibular tunnel was assessed as well.

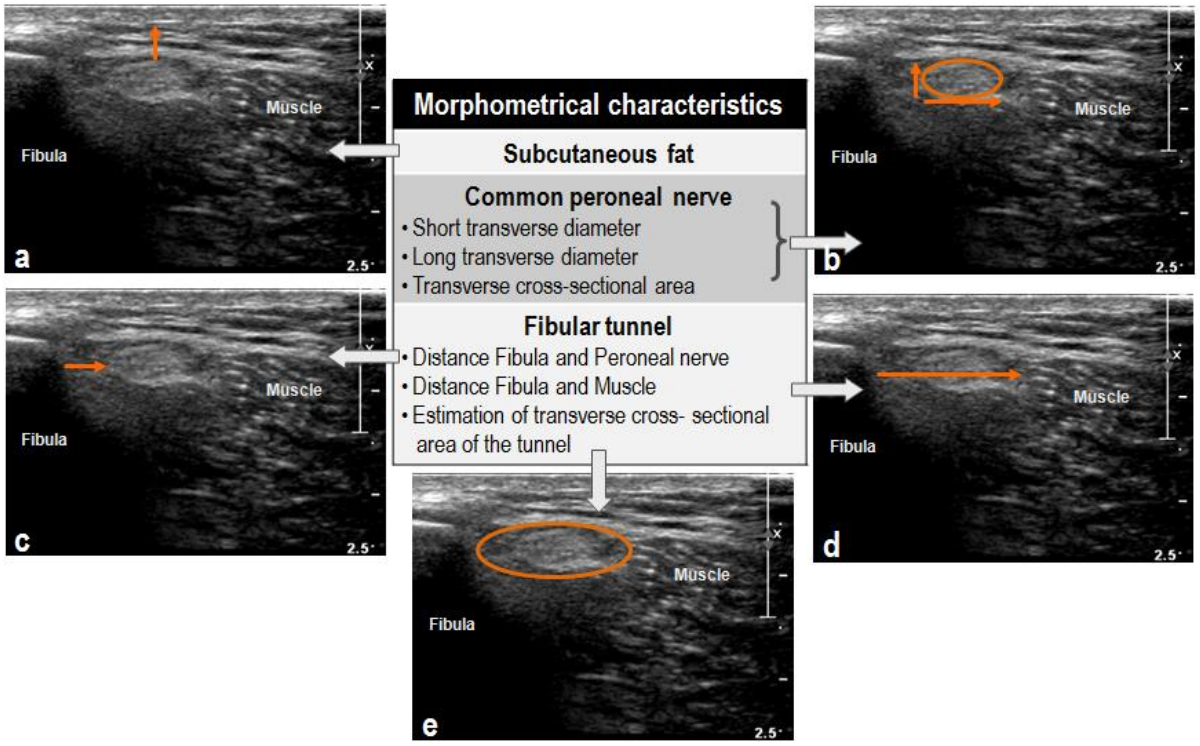


Figure 2.3 Morphometrical characteristics of *n. fibularis communis*: *A*. Subcutaneous fat superficial to the nerve; ***B.*** Transverse cross-sectional area of the nerve; ***C.*** Distance between the fibular bone and the nerve; ***D.*** Distance between the fibular bone and the *m. peroneus longus*; ***E.*** Transverse cross-sectional area of the fibular tunnel.

2.1.4 Statistical analysis

Using the students T-test, it was investigated whether there were significant difference in the measured characteristics between the right and left legs of the controls. Next, correlations

between these characteristics and BMI were analyzed using Pearson's correlation and multiple regression models. Only the P-values under a threshold of 0,05 were found significantly different.

2.2 Histology

The materials for the histological study were specimens of *n. fibularis communis* prelevated from eight adult cadavers (six males and two females) at the department of Basic Medical Sciences at the University of Hasselt in Diepenbeek. The human cadavers were balmed in a classical balm-fluid (containing alcohol, formalin, glycerol, phenol and thymol). During the embalming procedure, all of the blood is removed and replaced by balm-fluid.

2.2.1 Specimens

The specimens of *n. fibularis communis* was derived from the left lower limbs of the cadavers. *(Figure 2.1)* All specimens contained the distal part of *n. fibularis communis*, located at the fibular head, and its bifurcation into its two final branches, *n. fibularis profundus* and *superficialis*. The mean length of the specimen was 11 ± 1,7cm. *(Figure 2.1) N. ulnaris* was prelevated at the level of the left elbow from one of the three thick cadavers. The thickness of the subcutaneous fat was measured at two different levels: at level of the iliac crest and superficial to *n. fibularis communis* at the level of the fibular head. Using these adipose tissue measurements, the eight cadavers can be divided into thin cadavers (N=5) and thick cadavers (N=3).

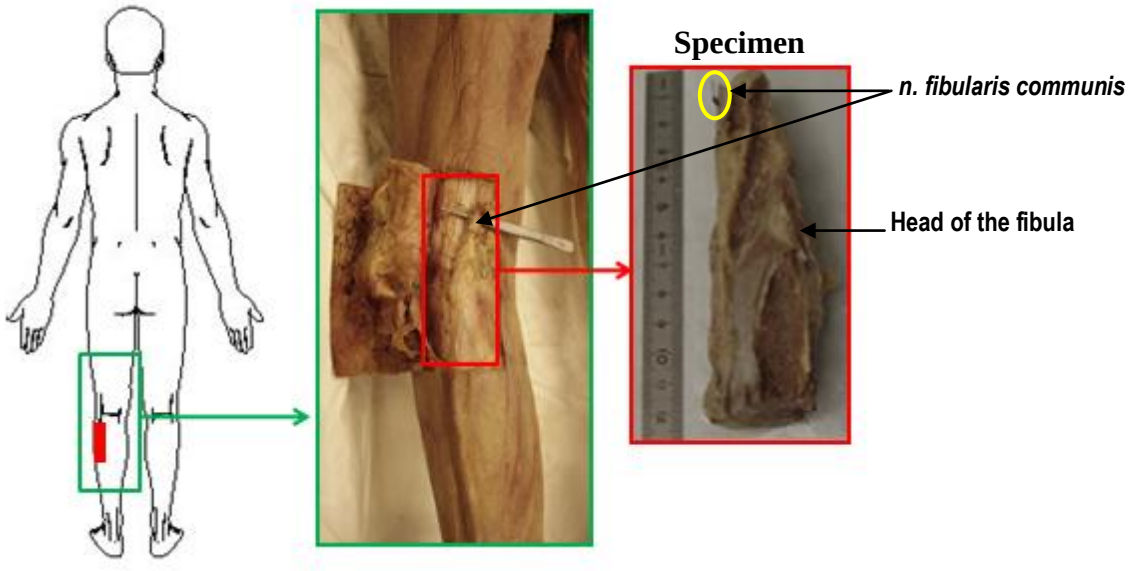


Figure 2.4 The specimens were taken from the left lower limbs of the cadavers.

Two different levels of the course of *n. fibularis communis* were examined in these specimens: at the level of the fibular head and at the bifurcation into *n. fibularis superficialis* and *n. fibularis profundus*. It has been described that the course of *n. fibularis communis* and its two final branches is variable between different individuals. [46] To ensure that the levels of interest were correctly obtained for analysis, the intact specimens were visualized using high-field MRI. The specimens were analyzed over a length of 30 mm using MRI performed in a 9,4 tesla vertical scanner (Varian® Inova 400 Spectometer, Palo Alto, CA, USA), with a probe diameter of 40 mm. Axial proton density images of the specimens were obtained with a repetition time (TR) and an echo time (TE) of 3000 ms and 18 ms, respectively. The images were taken as consecutive 1 mm slices with an in-plane resolution of 140 µm x 140 µm (FOV of 35 mm x 35 mm and imaging data matrix of 256 x 256).The acquired images had a number of averages (NA) of 4, resulting in a total acquisition time of approximately 50 minutes. Because the course of *n. ulnaris* was not as variable as *n. fibularis communis*, it was not necessary to perform MRI on this nerve.

After MRI, the fibular bone was carefully removed from all the specimens. Only soft tissues of the specimen were embedded in paraffin. Using the proton density images, a fragment of approximately 0,5 cm of the two levels of interest were embedded in paraffin. Two fragments of approximately 0,5 cm, taken at different levels along the course of *n. ulnaris*, were also embedded in paraffin.

2.2.2 Masson's trichrome staining

The paraffin blocks were cut into sections with a thickness of 5-7 µm. After deparaffinizing the sections with xylene and rehydrate them with ethanol, the sections are stained with the trichrome staining of Masson. The Masson's trichrome staining is a three-color staining protocol used to differentiate the connective tissue from other structures. In the first step "Mayer's hematoxylin" colours the nuclei of the sections dark red. Secondly, a combination of ponceau de xylidine, fuchsine and acidphloxine is used to stain the cytoplasm of the cells and the muscle tissues red. The third dye is anilin blue, staining the connective tissue blue. Phosphomolybdic acid was used after every staining, to hold the colour.

2.2.3 Analysis

The obtained histological sections were analyzed under light microscope and scanned using the MIRAX desk (Zeiss, Oberkochen, Germany). Morphometrical exploration was carried out at a low magnification of 1x using Axiovision software (Zeiss, Oberkochen, Germany). The

main trunk of peripheral nerve is generally composed of fascicles, containing the nerve fibers, connective tissue, blood vessels and intraneural adipose tissue. To quantify the amount of intraneural adipose tissue present in the nerve, the surface area of the nerve (A_n), the fascicles (A_f) and blood vessels (A_b) were measured. The surface area of the intraneural fat (A_i) was calculated as follows: $A_i = A_n - A_f - A_b$. The amount of intraneural fat present in the nerve was defined as the ratio of A_i/A_n. The intraneural fat percentage was quantified using an average of three to four consecutive sections at each level. A mean value for the thick (N=3) and thin cadavers (N=5) was calculated.

2.3 Epidemiological study

For this study 373 patients, who underwent gastric banding surgery at ZOL in Genk, were used. A survey, concerning problems in their lower legs, was send to each participant, to evaluate the prevalence of peroneal neuropathy. *(Supplement II)* Based on these completed surveys the participants were divided in a control group, including patients that did not have any symptoms of peroneal neuropathy, and a group of patients that complaint about symptoms of peroneal neuropathy (such as numbness, tingling or motor symptoms affecting their lower legs). The latter group was contacted by telephone and invited for a consultation, organized at ZOL, to clinically evaluate the patients. Not all contacted patients were able or willing to come for a consultation. These participants were evaluate on the telephone. From the surveys, it was assessed whether the patients received treatment for the neuropathy symptoms. In addition, the treatment outcome was examined. Based on their patient file at the department of Abdominal Surgery at ZOL, the mean amount and rapidity of weight loss was determined.

3. Results

The purpose of this study was to examine the internal structure of *n. fibularis communis*. Because weight loss leads to depletion of fat stores, the attention was focussed on the intraneural adipose tissue. To our knowledge, the prevalence of peroneal neuropathy after weight loss is unknown. This problem is therefore addressed in an epidemiological study.

3.1 In vivo ultrasonographic characterization of normal *n. fibularis communis*

N. fibularis communis was examined in 50 controls using in vivo US. Morphometrical characteristics of the nerve were assessed on the ultrasound images. In this study correlations between the BMI and the measured characteristics were investigated, using correlation studies and a multiple regression model.

3.1.1 Sample analysis

For this study, 50 volunteers were used, involving 25 females (50 % of the participants) and 25 males (50% of the participants). Among the female participants, the mean age was 49 years (ranging from 22 to 76 years), the mean weight was 66 kg (ranging from 48 to 92,5 kg), the mean height was 163 cm (ranging from 150 to 178 cm) and the mean BMI was 24 (ranging from 18 to 36). Among the male participants, the mean age was 46 years (ranging from 18 to 80 years), the mean weight was 85 kg (ranging from 63 to 112 kg), the mean height was 180 cm (ranging from 165 to 200 cm) and the mean BMI was 26 (ranging from 19 to 33).

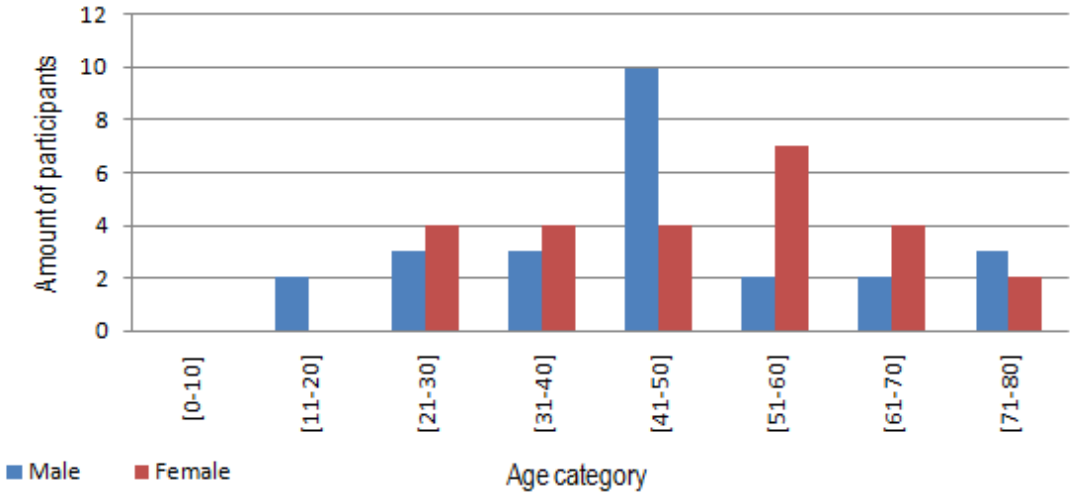


Figure 3.1 Age category of the 50 participants, in red: females, in blue: males.

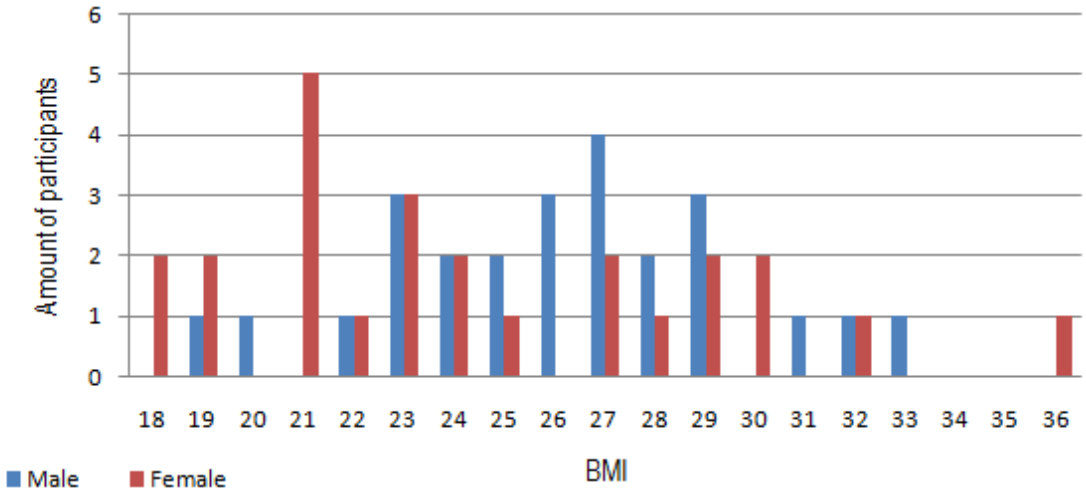


Figure 3.2 BMI of the 50 participants, in red: females, in blue: males.

3.1.2 Mean values of the measured characteristics

The mean values of the measured characteristics are represented in table 3.1. There were no significant differences between the values of the characteristics measured on the right and on the left side (P>0,05). The mean value measured for the subcutaneous adipose tissue, superficial to *n. fibularis communis*, was 0,44 ± 0,20 cm. The distance between the fibular head and *n. fibularis communis* was variable and ranged from 0,08 cm to 0,36 cm. The distance between the fibula and the muscle tissue was by average 1,10 ± 0,26 cm. We found that *n. fibularis communis* had a mean transverse cross-sectional area of 0,18 cm² ± 0,5 cm². For the transverse cross-sectional area of the fibular tunnel, we measured a mean value of 0,44 ± 0,16 cm².

Table 3.1 Mean values of morphometrical characteristics measured on the ultrasounds of *n. fibularis communis*.

Parameter	**Total (N=50)**	
	Mean	**Standard deviation**
Subcutaneous fat (cm)	0,44	0,20
Transverse cross-sectional area of nerve (cm²)	0,18	0,05
Distance between fibula and nerve (cm)	0,22	0,14
Distance between fibula and muscle (cm)	1,10	0,26
Transverse cross-sectional area of tunnel (cm²)	0,44	0,16

3.1.3 Correlation of the measured characteristics with BMI

Significant positive correlations were found between the BMI and three important parameters: the subcutaneous fat, superficial to the nerve (Pearson's coefficient (R) =0,437; P<0,001) *(Figure 3.3A)*, the transverse cross-sectional area of the nerve (Pearson's coefficient (R) =0,430; P<0,001) *(Figure 3.3B)* and the transverse cross-sectional area of the fibular tunnel (Pearson's coefficient (R) =0,391; P<0,001). *(Figure 3.3C)* These correlations imply that an increase in BMI is accompanied with a significant increase in the amount of subcutaneous fat, and an increase in the cross-sectional area of the nerve and fibular tunnel.

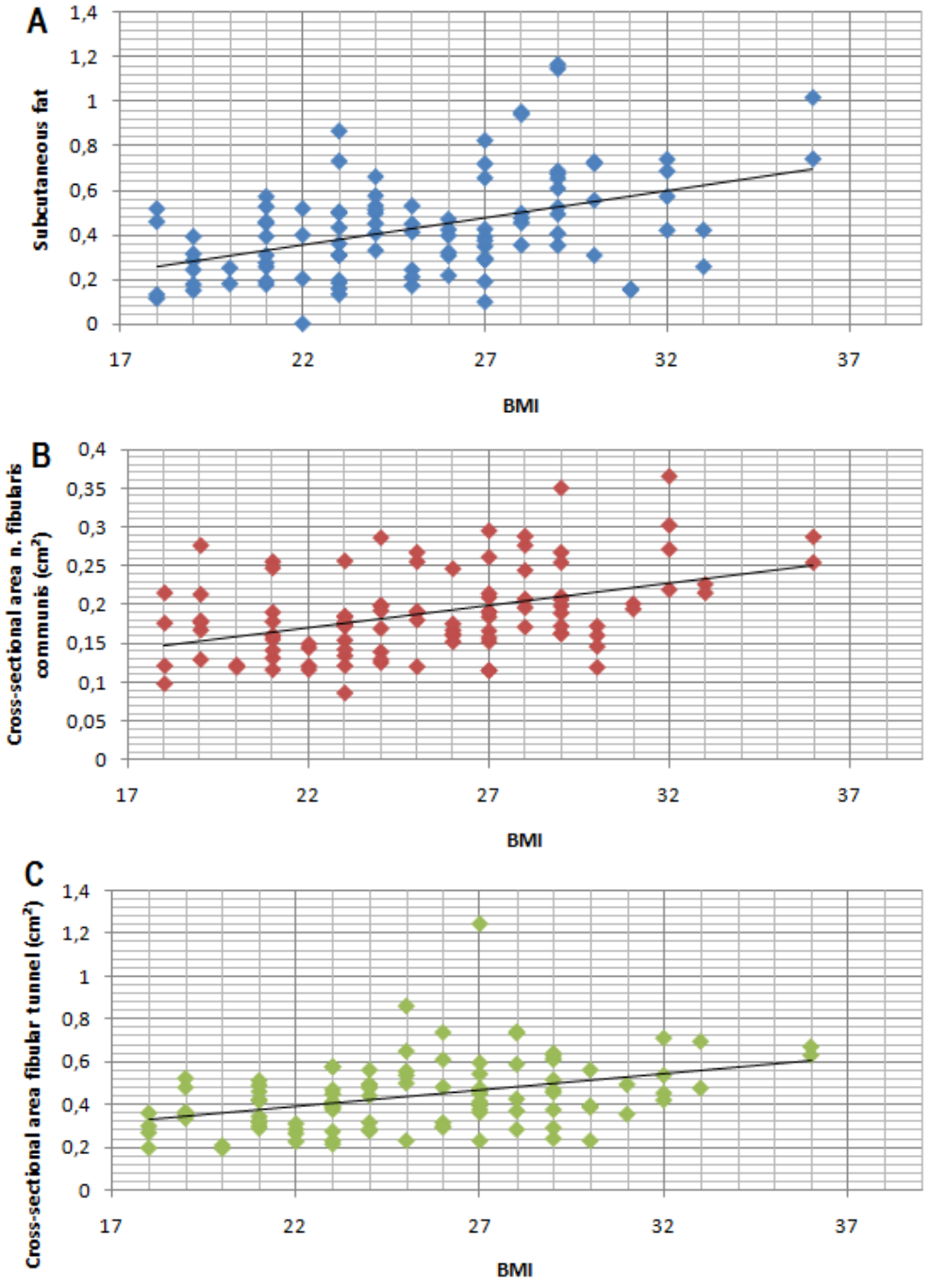


Figure 3.3 A. Relationship between BMI and the subcutaneous fat, superficial to the *n. fibularis communis*. Pearson's correlation (R) = 0,437; P<0,001. **B.** Relationship between BMI and the transverse cross-sectional area of the *n. fibularis communis*. Pearson's correlation (R) = 0,430; P<0,001. **C**. Relationship between BMI and the transverse cross-sectional area of the fibular tunnel. Pearson's correlation (R) = 0,391; P<0,001.

Using the same multiple regression model, significant correlations between the subcutaneous fat and the cross-sectional area of *n. fibularis communis* (Pearson's correlation (R) = 0,290, p = 0,003) were found. Upon an increase of the subcutaneous fat, superficial to *n. fibularis communis*, the cross-sectional area of the nerve increases as well. *(Figure 3.4)*

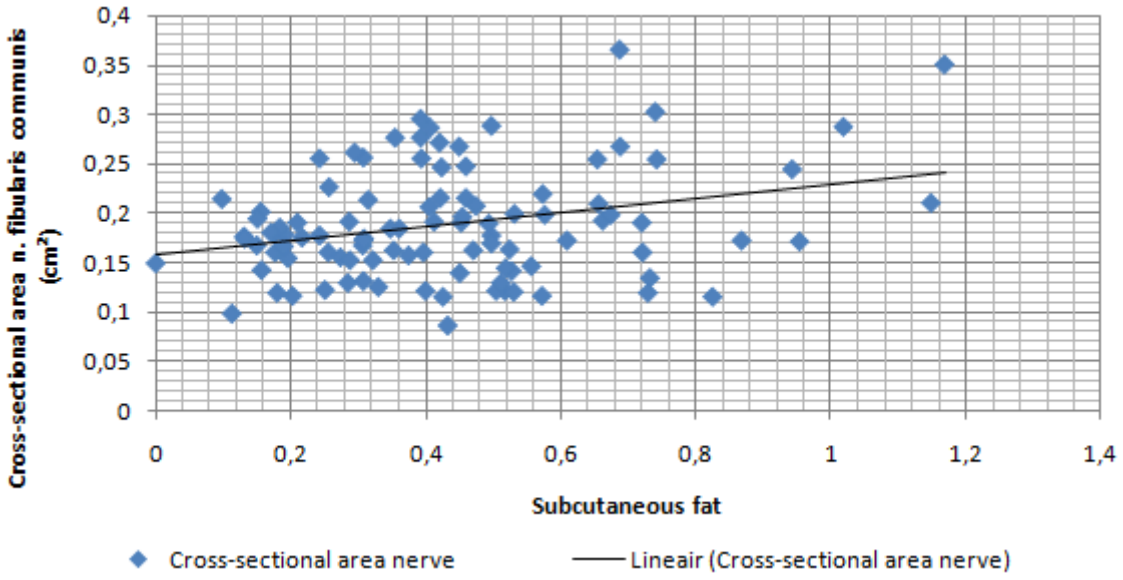


Figure 3.4 The relationship between subcutaneous fat, superficial to the peroneal nerve and the transverse cross-sectional area of the *n. fibularis communis*. [Pearson's correlation (R) = 0,290; P=0,003.]

Figure 3.5 illustrates ultrasound images of a person with a high BMI (right image) and a low BMI (left image). A clear difference in echogenicity was observed between the two ultrasound images. The nerve of the obese person appeared bilaterally thicker, hyperechoic and lighter. The US images of the thin control showed a thinner and darker (hypoechoic) nerve in both legs.

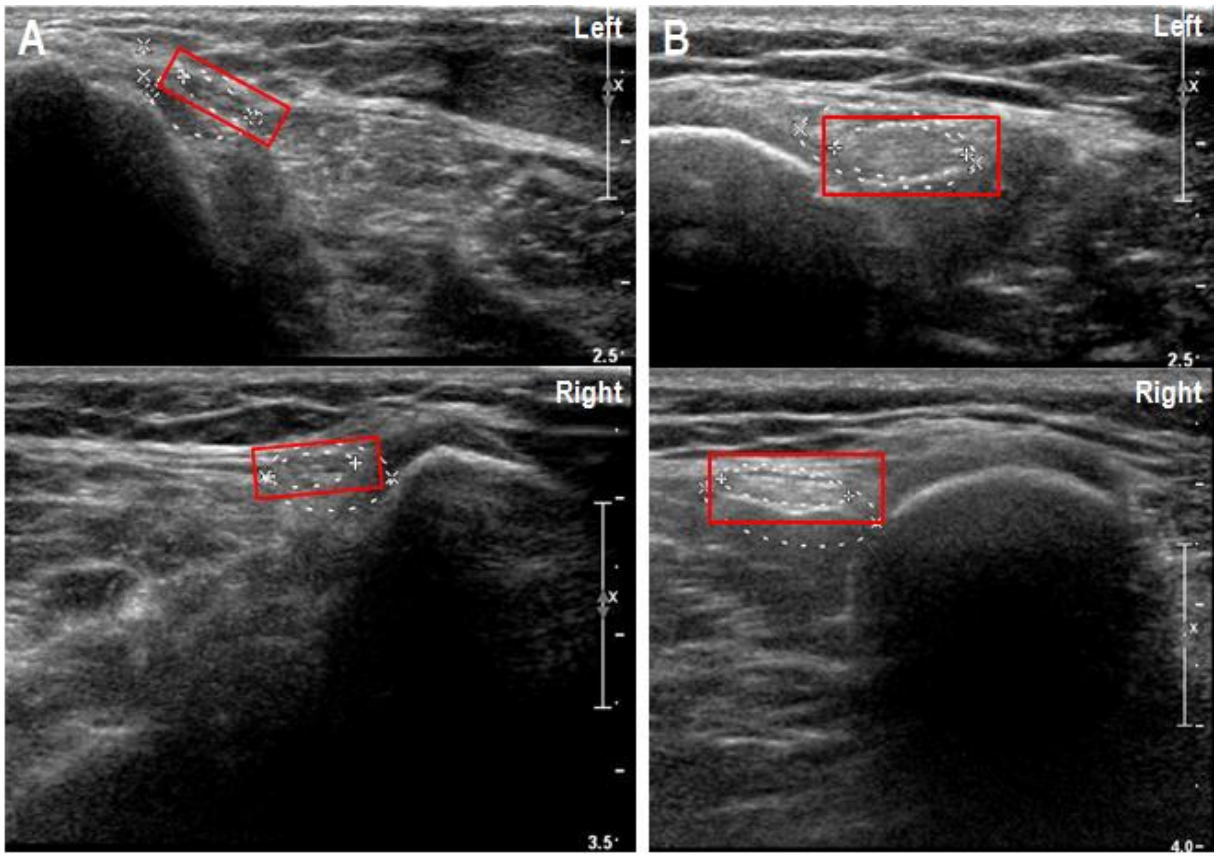


Figure 3.5 A. On the ultrasound of a patient with a low BMI, the nerve (indicated by the red rectangle) appears more hypoechoic and thinner. **B.** The ultrasounds of a patient with a high BMI shows a hyperechoic and thicker nerve (indicated by the red rectangle).

3.2 In vivo ultrasonographic characterization of pathological *n. fibularis communis*

N. fibularis communis was visualized in patients, having a history of weight loss, diagnosed with peroneal neuropathy. This condition resulted in a foot drop, seriously affecting the patient's ability to walk. Herein, we report four cases of patients suffering from a foot drop, resulting from peroneal neuropathy.

3.2.1 Case 1

The ultrasound showed a denser and hypoechoic *n. fibularis communis* on the pathological side (right side). Contralaterally, the nerve appeared hyperechoic. *(Figure 3.6)* Morphometrical analysis showed that on the pathological side, the nerve had a transverse cross-sectional area of 0,071 cm^2, which is two times smaller in than the cross-sectional area of the left nerve (0,151 cm^2). *(Table 3.1)* The atrophic nerve appears thinner in comparison to the non-affected side on the US images.

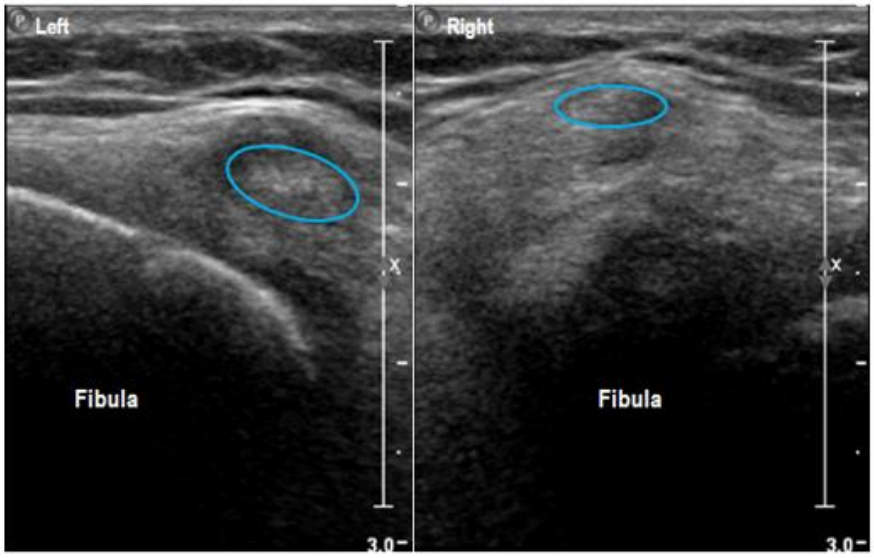


Figure 3.6 Ultrasound case 1: On the right side (pathological side) *n. fibularis communis* (blue) is denser en more hypoechoic than on the nerve on the left side (blue).

Table 3.1 Morphometrical characteristics of *n. fibularis communis* in case 1: The transverse cross-sectional area of the left nerve appeared two times as thick than on the right side (pathological side)

	Left side	Right side (Pathological side)
Subcutaneous fat (cm)	1,46	0,785
Long diameter *n. fibularis communis* (cm)	0,662	0,514
Short diameter *n. fibularis communis* (cm)	0,291	0,181
Transverse cross sectional area nerve (cm²)	**0,151**	**0,073**
Distance between fibula and nerve (cm)	0,775	0,449
Distance between fibula and muscle (cm)	2,41	1,58

3.2.2 Case 2

On the ultrasound image, *n. fibularis communis* (pathological side) appeared to a little extent hypoechoic on the left side compared to the non-affected side. *(Figure 3.7)* Morphometrical analysis showed a minor increase in the transverse cross sectional area of the left nerve (0,235 cm²) and the left fibular tunnel (0,520 cm²) in comparison to the right leg (0,162 cm² and 0,302 cm² respectively). *(Table 3.2)*

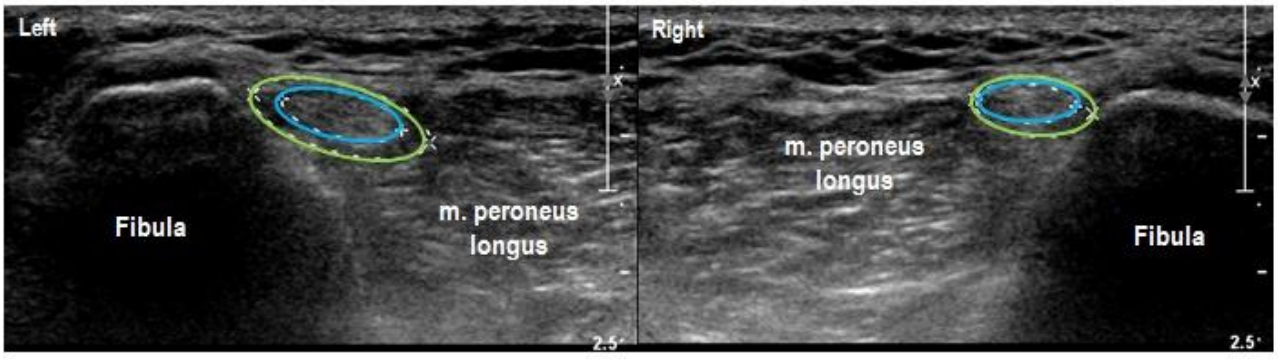


Figure 3.7 Ultrasound case 2: On the left side *n. fibularis communis* appeared hypoechoic, in comparison to the contralateral leg.

Table 3.2 Morphometrical characteristics of *n. fibularis communis* in case 2: The transverse cross-sectional area of the nerve and fibular tunnel were slightly larger on the pathological side compared contralaterally.

	Left side (Pathological side)	Right side
Subcutaneous fat (cm)	0,387	0,302
Long diameter *n. fibularis communis* (cm)	0,861	0,861
Short diameter *n. fibularis communis* (cm)	0,303	0,303
Transverse cross sectional area nerve (cm²)	0,235	0,162
Distance between fibula and nerve (cm)	0,288	0,150
Distance between fibula and muscle (cm)	1,41	1,05
Transverse cross-sectional area tunnel (cm²)	0,520	0,302

3.2.3 Case 3

The transverse cross-sectional area of the left *n. fibularis communis* (0,303 cm²) appeared approximately two times larger in comparison to the cross-sectional area of the non-affected nerve (0,174 cm²). *(Table 3.3)* On the image he nerve also appeared darker or hyporeflective on the left side compared to the nerve on the right side. *(Figure 3.8)*

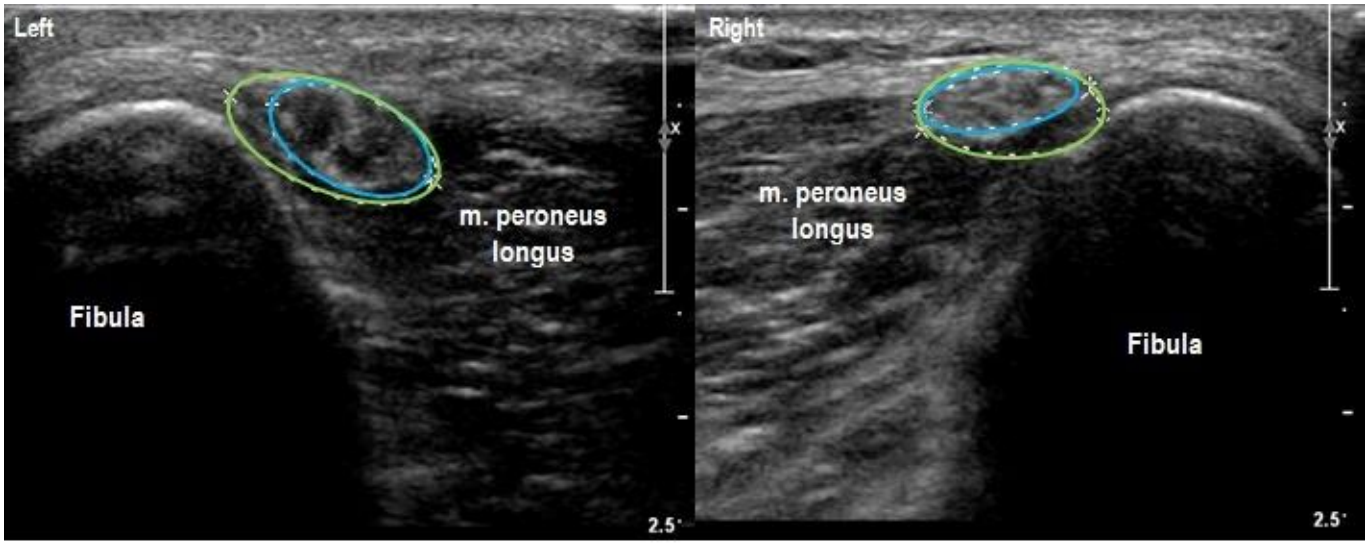


Figure 3.8 Ultrasound case 3: The left *n. fibularis communis* appears hypoechoic in comparison to the contralateral side.

Table 3.3 Morphometrical characteristics of the *n. fibularis communis* in case 3: the transverse cross-sectional area of the nerve was two times larger on the pathological side compared contralaterally.

	Left side (Pathological side)	Right side
Subcutaneous fat (cm)	0,334	0,137
Long diameter *n. fibularis communis* (cm)	0,720	0,831
Short diameter *n. fibularis communis* (cm)	0,459	0,253
Transverse cross sectional area nerve (cm²)	0,303	0,174
Distance between fibula and nerve (cm)	0,111	0,232
Distance between fibula and muscle (cm)	1,14	1,25
Transverse cross-sectional area tunnel (cm²)	0,431	0,304

3.2.4 Case 4

The ultrasound image showed no apparent differences in echogenicity and the transverse cross-sectional area between the right and the left nerve. Morphometrical analysis of *n. fibularis communis* revealed that the transverse cross-sectional area of the left nerve (0,240 cm^2) is approximately equal to the transverse cross-sectional area of the right nerve (0,298 cm^2).

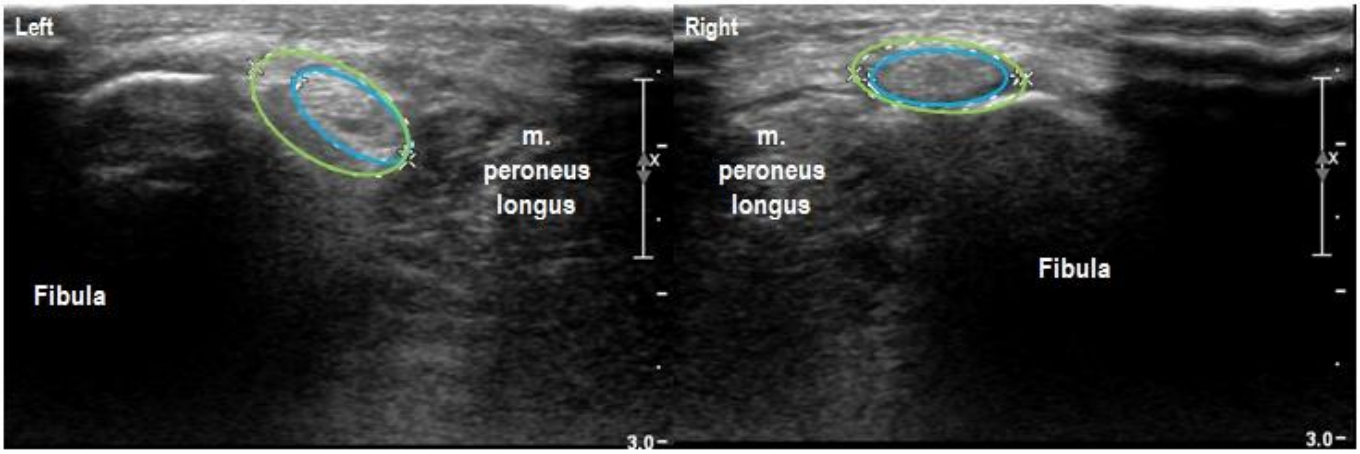


Figure 3.9 Ultrasound case 4: no obvious differences between the left and right nerve, due to the fact that both nerves were affected.

Table 3.4 Morphometrical characteristics of the *n. fibularis communis* in case 4: no obvious differences between the left and right nerve, due to the fact that both nerves were affected.

	Right side	Left side
Subcutaneous fat (cm)	0,256	0,373
Long diameter *n. fibularis communis* (cm)	1,07	0,689
Short diameter *n. fibularis communis* (cm)	0,316	0,354
Transverse cross sectional area nerve (cm^2)	0,298	0,240
Distance between fibula and nerve (cm)	0	0,14
Distance between fibula and muscle (cm)	1,13	0,940
Transverse cross-sectional area tunnel (cm^2)	0,427	0,402

3.3 Histological characterization of *n. fibularis communis*

The gross appearance of *n. fibularis communis* was examined in specimens, prelevated from human cadavers. Attention was focused on the intraneural fat of the nerve. The intraneural adipose tissue percentage was quantified on histological sections, visualized by the Masson's trichrome staining procedure.

3.3.1 Microanatomical structure of n. fibularis communis

By staining with Masson's trichrome procedure, the different components of the nerve, such as muscles, nerves, blood vessels, connective tissue and adipose tissue, were visualized. Using light microscopy, intraneural adipose tissue can be compared in the different specimens. Figure 3.10A represents *n. fibularis communis* at the level of the fibular head in the thick specimen. Figure 3.10B represents the same level in the specimen of a thin cadavers. In thin specimens, more intraneural adipose cells between the fascicles were observed. On the opposite, The histological sections of the thin specimens demonstrated a low content of the interfascicular fat. In comparison to the obese specimens, the nerves of thin cadavers appeared to be denser and the fascicles were structured closer together.

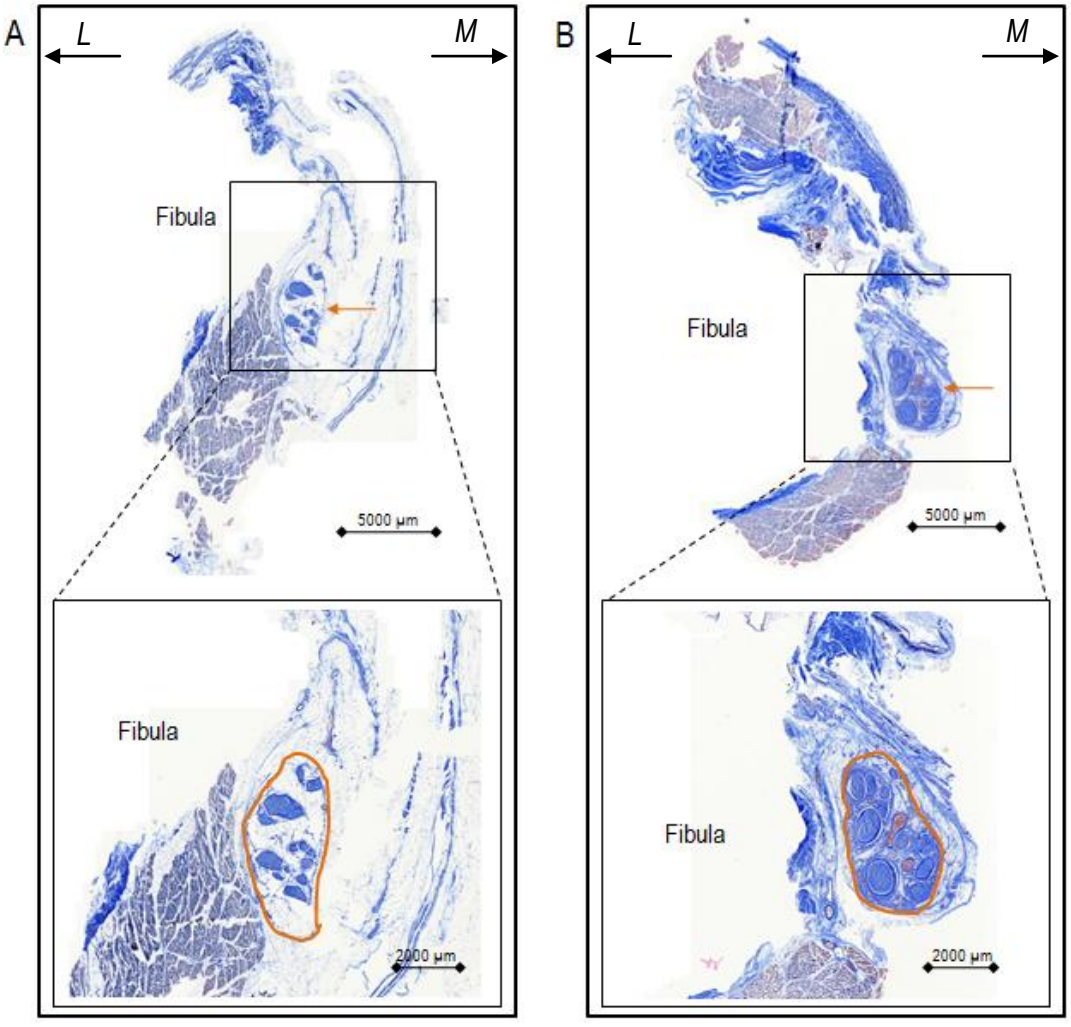


Figure 3.10 Masson's trichrome staining of *n. fibularis communis* in thick specimens (A) and thin specimens (B). (L= Lateral; M= Medial)

In figure 3.11A, a representation of the second level of interest in the specimen shown for the thick cadavers. The same level in the specimen for the thin cadavers is represented in figure

3.11B. In both *n. fibularis superficialis* as *n. fibularis profundus*, more intraneural fat was observed in the thick specimens. Here, the fascicles were surrounded with more adipose tissue. In the thin cadavers, the fascicles were located closer together, due to less intraneural adipose tissue. It was visible that *n. fibularis profundus* contained in most cases less interfascicular fat and appeared denser compared to *n. fibularis superficialis*.

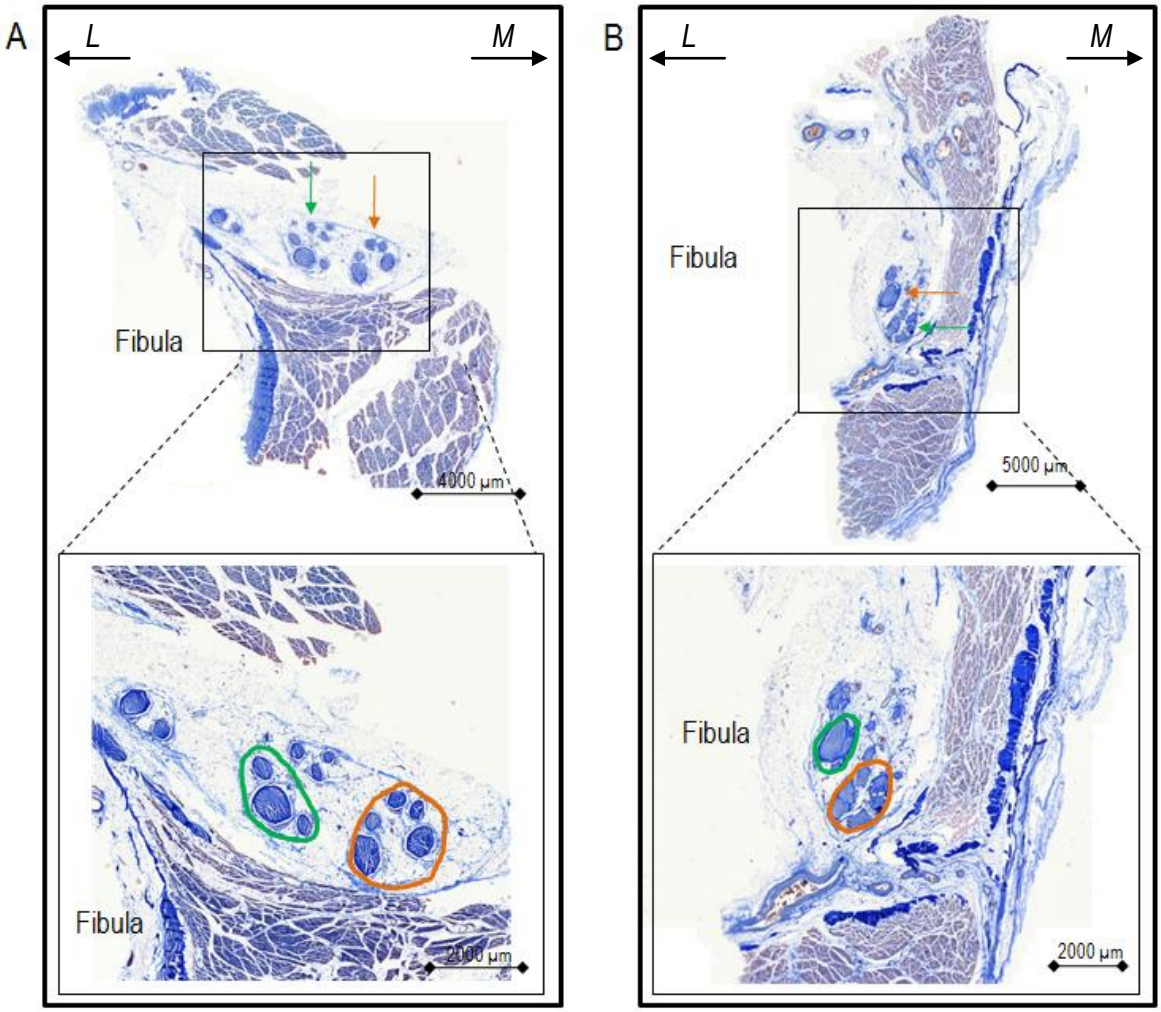


Figure 3.11 Masson's trichrome staining of *n. fibularis superficialis* (indicated in orange) and *n. fibularis profundus* (indicated in green) in thick specimens (A) and thin specimens (B). (L= Lateral; M= Medial)

3.3.2 Morphometrical analysis of intraneural adipose tissue

The amount of intraneural fat was quantified on the histological sections. The results for *n. fibularis communis, n. fibularis superficialis and n. fibularis profundus* are represented in figure 3.12. For *n. fibularis communis*, a mean value of 60% and 33,8% was measured for the intraneural fat ratio in thick (dark blue bars) and thin cadavers (light blue bars), respectively. Quantification of intraneural adipose tissue in *n. fibularis superficialis* and *n. fibularis*

profundus revealed a higher percentage of intraneural fat in the thick cadavers compared to the thin cadavers. The mean values of the intraneural fat ratio obtained for *n. fibularis profundus*, were 46% for the thick cadavers and 28% for the thin cadavers. For *n. fibularis superficialis*, the mean values of the intraneural fat ratio were 56% and 28%, respectively. In general, the quantification of intraneural fat ratios confirms that a higher percentage of intraneural fat was present in the thick cadavers compared to the thin cadavers.

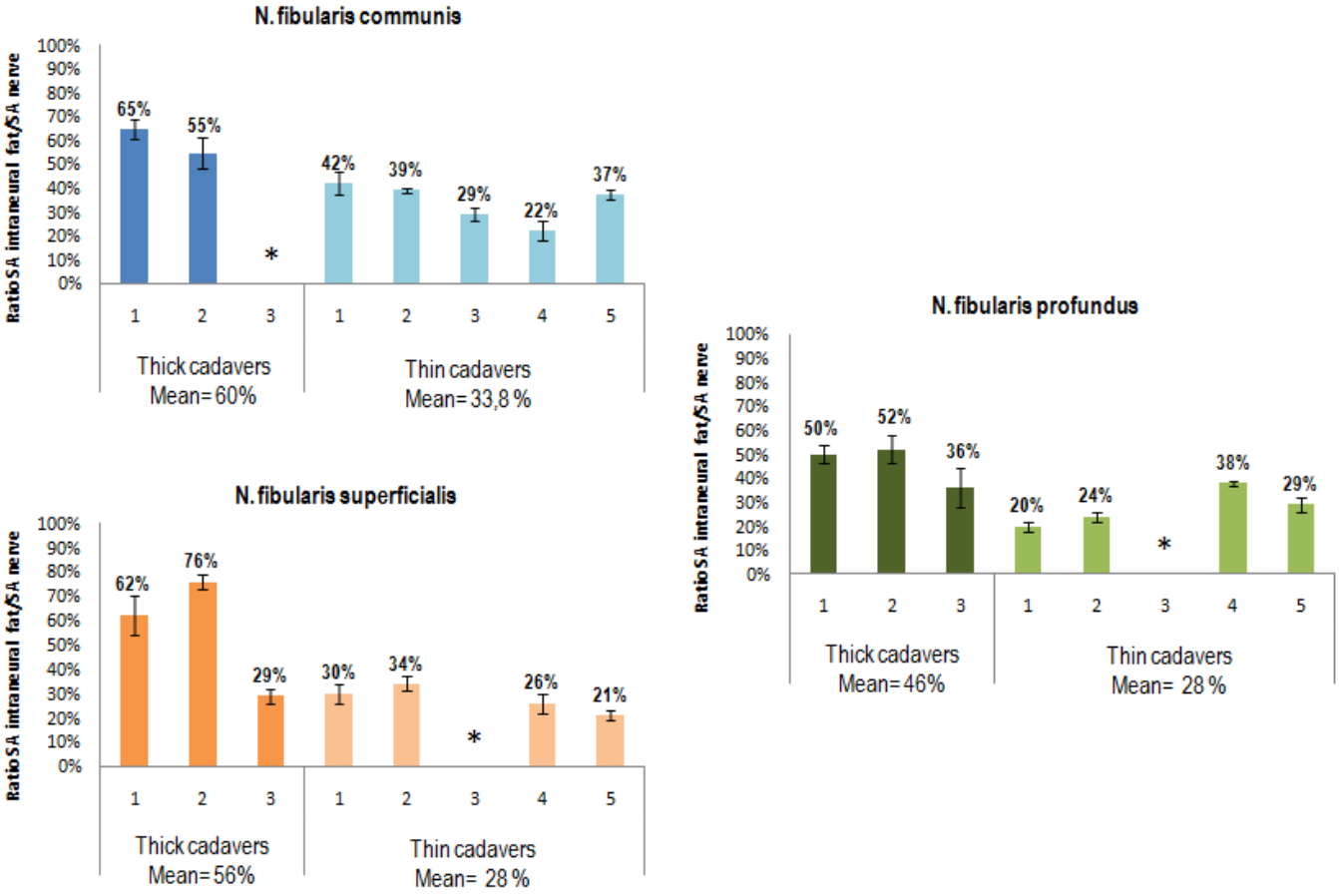


Figure 3.12 Quantification of the percentage intraneural fat present in the n. fibularis communis (blue), *n. fibularis profundus* (green) and *n. fibularis superficialis* (orange). (Missing data are indicated with an asterisk)

3.3.3 Intraneural fat percentage – subcutaneous fat correlations

The relationships between the intraneural fat ratio and adipose tissue throughout the body were assessed. Positive relationships could be observed between the intraneural fat ratio in *n. fibularis communis* and the thickness of the subcutaneous fat at these two levels. The correlation factors (R^2) were approximately 0,51 and 0,13 for the subcutaneous fat superficial to *n. fibularis communis* and at the level of the iliac crest, respectively. These positive relationships were visible at the level of the bifurcation into *n. fibularis superficialis* and *n. fibularis profundus* as well. The intraneural fat ratio obtained in *n. fibularis profundus* was

correlated with a correlation factor (R^2) of approximately 0,75 for the subcutaneous fat superficial to *n. fibularis communis* and 0,05 for subcutaneous fat at the level of the iliac crest. In *n. fibularis superficialis,* the subcutaneous fat superficial to *n. fibularis communis* and at the level of the iliac crest were correlated with the intraneural fat with a correlation factor (R^2) of approximately 0,39 and 0,01, respectively. Figure 3.13 showed that when more subcutaneous fat was present, there was a higher fat percentage in *n. fibularis communis* as well as in *n. fibularis superficialis* and in *n. fibularis profundus*. The same positive relationships were observed for the subcutaneous fat measured at the iliac crest. *(Figure 3.14)*

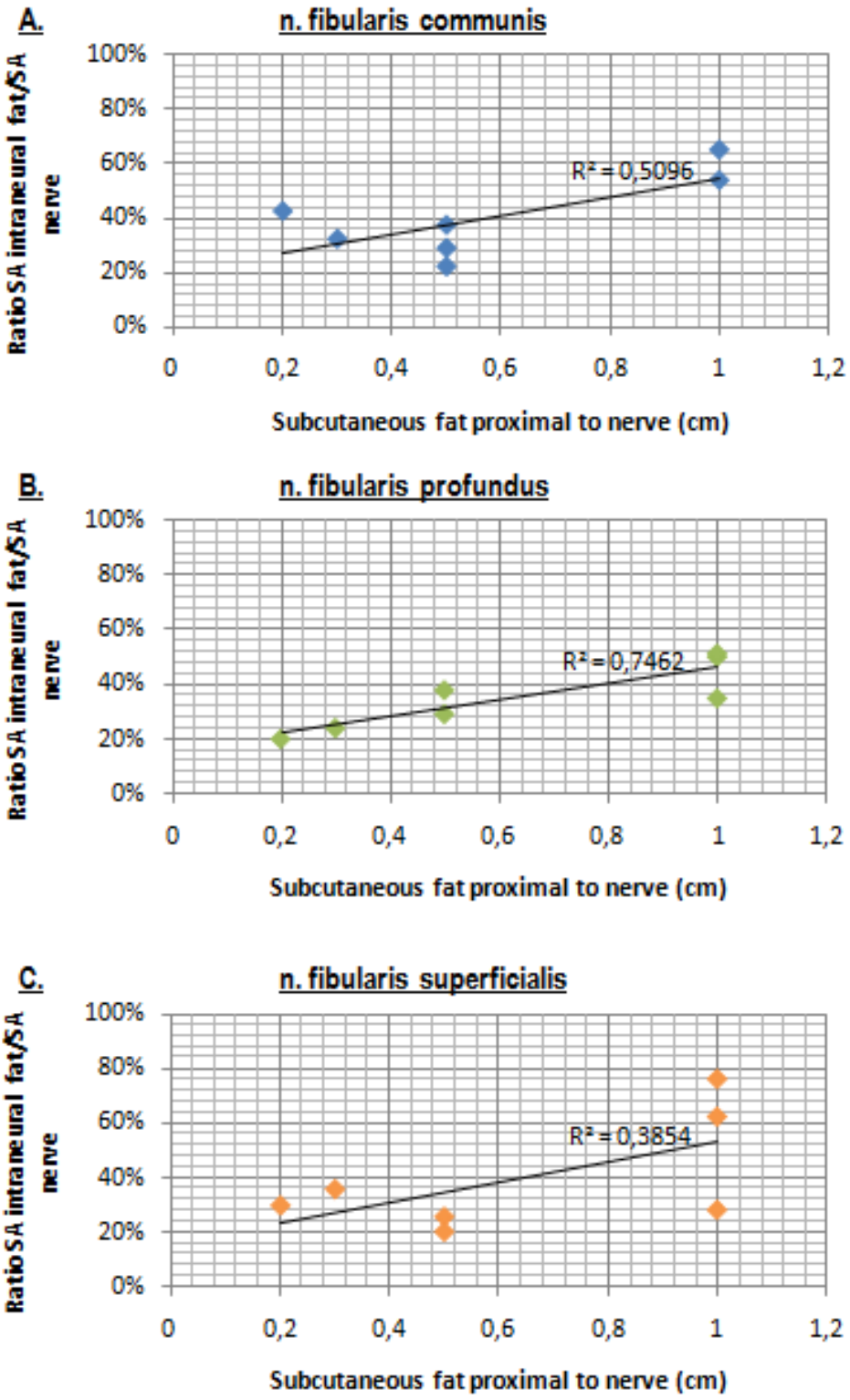


Figure 3.13 Positive relationships were observed between the intraneural fat ratio (Ai/An) of the *n. fibularis communis*, *n. fibularis superficialis* and *n. fibularis profundus* and the subcutaneous fat, measured superficial to the *n. fibularis communis* at the level of the fibular head.

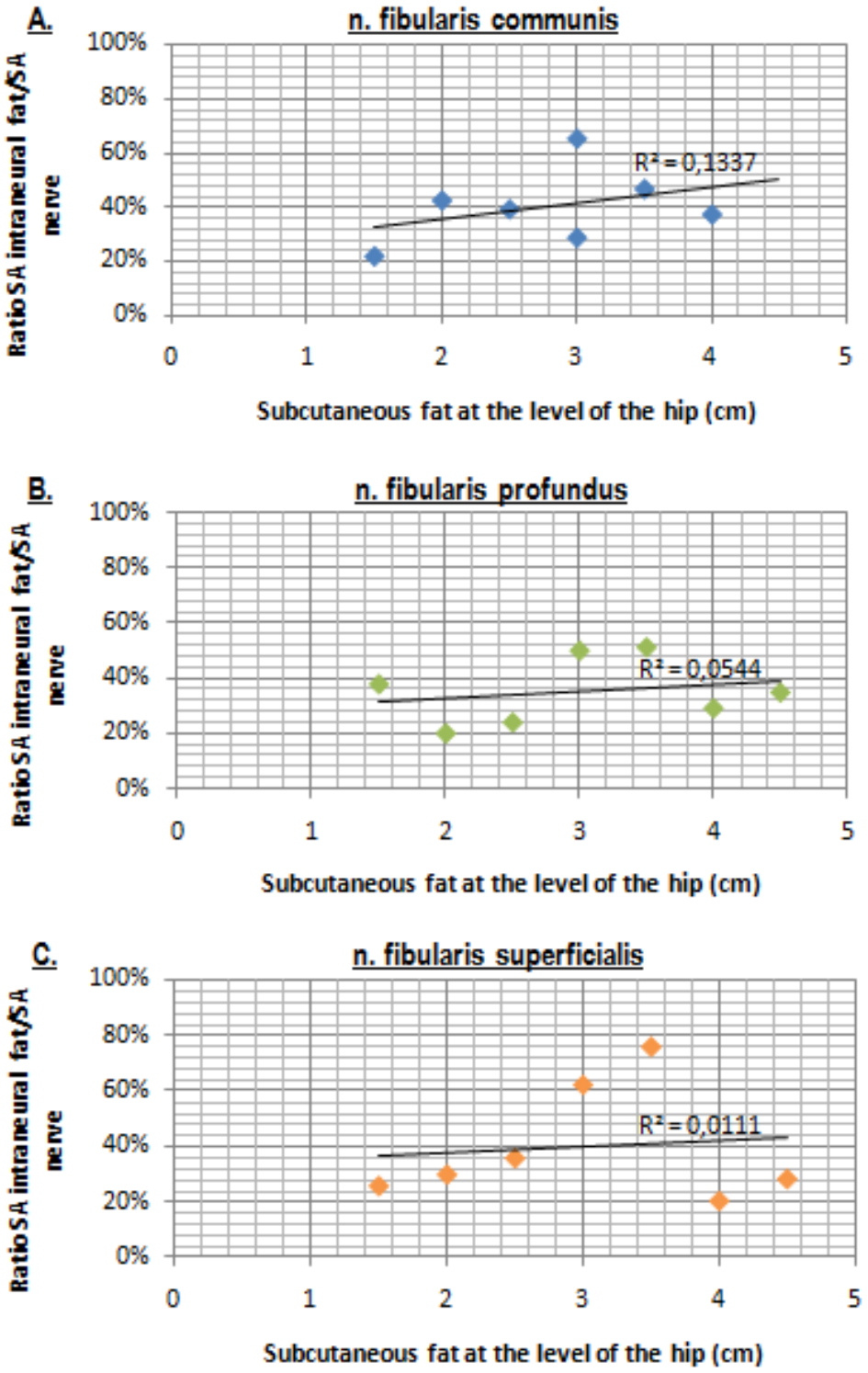


Figure 3.14 Positive relationships were observed between the intraneural fat ratio (A_i/A_n) of the *n. fibularis communis*, *n. fibularis superficialis* and *n. fibularis profundus* and the subcutaneous fat, measured at the iliac crest.

3.4 Histological characterization of *n. ulnaris*

Figure 3.15 represents *n. ulnaris* at the level of the elbow in a thick specimen, visualized with Masson's trichrome staining procedure. In comparison to what was observed in *n. fibularis communis* in thick cadavers, *n. ulnaris* contained less adipose tissue. Quantification of intraneural fat ratio of *n. ulnaris* revealed a mean value of 37%. This value was smaller in comparison to the mean values of 55%, 76% and 52% measured in *n. fibularis communis*, *n. fibularis superficialis* and *n. fibularis profundus* of the same cadavers, respectively.

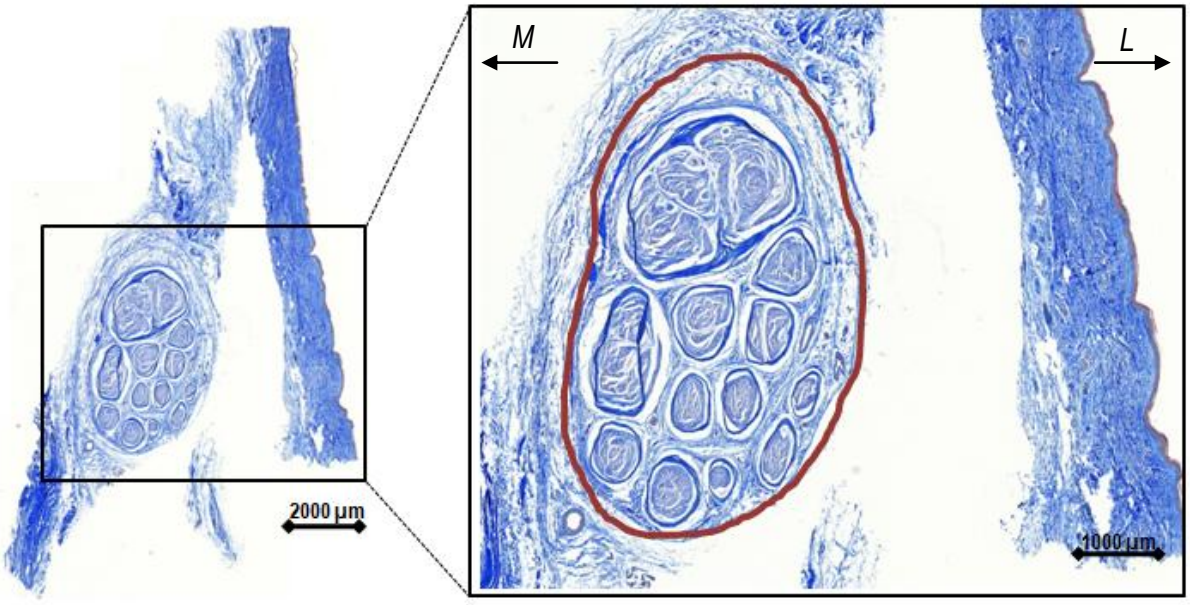


Figure 3.15 Masson's trichrome staining of the *n. ulnaris* (indicated in red) in a thick cadaver. (L= Lateral; M= Medial).

3.5 Epidemiological study

In the epidemiological study 373 patients, who underwent gastric banding, were evaluated on the prevalence of symptoms of peroneal neuropathy. Ten participants had trouble lifting their foot upwards (dorsiflexion) during walking. Two other patients suffered from loss of sensation at the dorsum of the foot and along the lateral side of the leg. From the ten patients, only one suffered from a complete fallout of the nerve, seven patients demonstrated severe motor deficits and two participants exhibited a mild form of peroneal nerve palsy. In summary, 3% of the patients had symptoms indicating a peroneal nerve entrapment. *(Figure 3.16)*

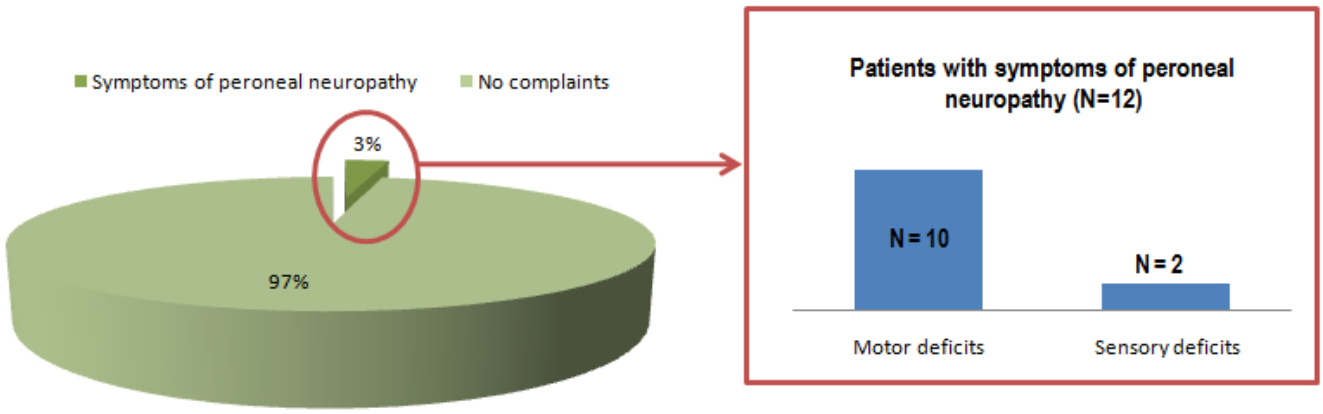


Figure 3.16 In this study 373 patients undergoing gastric banding surgery were evaluated. 3% of these patients showed symptoms of peroneal neuropathy, including 10 patients with motor deficits and 2 patients suffering from sensory deficits.

The group of patients with motor deficits had a mean weight loss of 38,7 kg during a mean period of 15 months, indicating a mean weight loss rapidity of 4 kg per month. The 38,7 kg of weight loss, represented 35% of their bodyweight. The patients suffering from sensory dysfunctions lost a mean weight of 23,8 kg, representing 22% of their bodyweight, during a mean period of 32,5 months. The mean rapidity of weight loss was 1 kg per month. In total, the mean weight loss of the patients with symptoms of peroneal neuropathy after undergoing gastric banding surgery was 35,8 kg during a mean period of 18,5 months, indicating a mean weight loss rapidity of 4 kg per month. This weight loss represented 32% of their total bodyweight. *(Table 3.5)*

Table 3.5 Characteristics of weight loss in the patient group suffering from symptoms of peroneal nerve entrapment

	Weight loss (kg)	Period of weight loss (months)	Weight loss(kg)/month	% bodyweight that was lost	% bodyweight reduction/ month
Motor deficits (N=10)	38,7 kg	15 months	4 kg/month	35%	4%/month
Sensory deficits (N=2)	23,8 kg	32,5 months	1 kg/month	22%	1%/month
Total (N=12)	35,8 kg	18,5 months	4 kg/month	32%	3%/month

In this patient group, the treatment and treatment outcome of these patients were evaluated. Nine participants were not treated for their symptoms, including the two patients with sensory deficits. Only one patient underwent surgical decompression. Post operatively, this person showed a complete recovery from peroneal neuropathy. Two participants received a conservative treatment. This conservative treatment was mainly comprised of physiotherapy. The majority of these patients still showed their symptoms after being treated. *(Figure 3.17)*

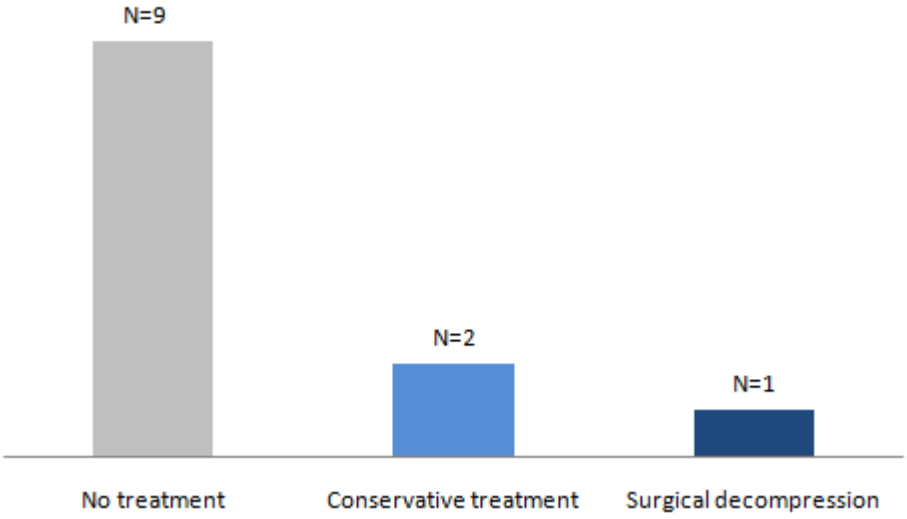


Figure 3.17 Of the 12 patients, showing symptoms of peroneal neuropathy, only one patient was treated with surgical decompression and two patients were treated with conservative treatment, mainly physiotherapy. The majority, nine patients, were not treated.

4. Discussion

Peroneal neuropathy is a condition in which *n. fibularis communis* is compressed. Several studies have shown that a significant percentage of patients developed peroneal neuropathy after weight loss. [1, 22, 28, 43] Because it is not yet known how weight loss induces peroneal neuropathy, the best treatment option has yet to be elucidated. It is thought that a combination of mechanical causes and metabolic disturbances will lead to the development of peroneal nerve entrapment. Basic knowledge of the microanatomy is an important feature in understanding nerve compression injuries. For this reason, we examined the microanatomy of the peroneal nerve. Herein, *n. fibularis communis* was visualized and examined using in vivo ultrasonographic and histological techniques. Because weight loss results in a decrease of adipose tissue, we focussed on the adipose tissue in and surrounding the nerve. Relevant literature concerning this subject is absent.

Because *n. fibularis communis* has a superficial course at the level of the fibular head, it can be easily visualized with ultrasonographic techniques. Since peroneal neuropathy occurs mainly at the level of the fibular head and neck, *n. fibularis communis* and its surrounding structures were examined at this location. Different morphometrical characteristics were measured on the ultrasound images. Our techniques were standardized by taking the image at the position where the nerve has the closest proximity to the fibular head. The characteristics were measured by one observer, to overcome bias between the different measurements.

The mean value measured for the subcutaneous adipose tissue, superficial to *n. fibularis communis*, was 0,44 ± 0,20 cm. The great standard deviation is due to the wide variety in BMI present in the sample, visualized in figure 3.2. The distance measured between the fibular head and *n. fibularis communis* could also vary in the sample. This value ranged from 0,08 cm, indicating that *n. fibularis communis* is in close proximity with the fibular head, to 0,36 cm. The transverse cross-sectional area of *n. fibularis communis* and of the fibular tunnel had a mean value of respectively 0,18 ± 0,5 cm^2 and 0,44 ± 0,16 cm^2. Literature could not provide any reference values for these measurements.

Using correlation studies, we found that BMI was significantly correlated to different characteristics, measured on the ultrasound image. According to the National Institutes of Health BMI can be used to define overweight and obesity in patients. BMI correlates well with total body fat for the majority of the people and is simple to calculate. We found that an increase in BMI was correlated to an increase in the subcutaneous fat superficial to the nerve, indicating that obese persons have more subcutaneous fat that covers *n. fibularis communis*.

A second positive correlation was found between the BMI and the transverse cross-sectional area of the fibular tunnel. On US images, the controls with a high BMI presented next to a thicker nerve, a larger fibular tunnel. This can suggest that the fibular tunnel is filled with more adipose tissue, surrounding the nerve. A significant correlation was found between the BMI and the transverse cross-sectional area of *n. fibularis communis*. This correlation implies that the nerve in an obese person is significantly thicker than in a thin person, estimating that more intraneural fat could be present in the nerve. On the US image of an obese person, we observed that the nerves appeared hyperechoic. Because adipose tissue has a higher echogenicity, this suggests that more adipose tissue could be present.

US was performed in four patients suffering from peroneal neuropathy. The first patient presented atrophy of *n. fibularis communis*, after a prolonged coma. The atrophy was detected on the ultrasound through a thinner appearance of the nerve compared to the non-affected side. Because the last case presented with bilateral peroneal nerve palsy, the ultrasound did not show many differences between the left and right side. However, the US images of the other two patients, developing peroneal neuropathy after losing more than 10% of their bodyweight, showed a thickening of the nerve on the pathological side. In these patients the transverse cross-sectional area of the nerve was approximately two times ticker on the affected side compared contralaterally. In addition, the nerve appeared more hypoechoic or darker on the pathological side. Although literature focussing on the role of ultrasound imaging in peroneal neuropathy is limited, these findings are in agreement with findings of ultrasound imaging in other neuropathies, such as carpal tunnel syndrome. This common compression neuropathy is caused by the entrapment of *n. medianus* within the carpal tunnel in the wrist. On US images, the compressed nerve of the carpal tunnel syndrome appeared thicker in comparison to normal nerves. The cross-sectional area of the nerve was larger in the pathological arm than in the normal arm. This nerve enlargement was due to nerve swelling and underlying oedema formation. Therefore, the transverse cross-sectional area of the nerve was found to be a viable tool in the diagnosis of carpal tunnel syndromes. Furthermore, the nerve appeared in most cases hypoechoic in comparison to the normal nerves. [47] In our study we found that differences in nerve thickness and echogenicity between the affected and non-affected side were detectable on the US images. Consequently, high-resolution sonography may be used as an adjunct in the visualization of *n. fibularis communis* next to MRI.

In the future, it is interesting to analyze the morphometrical characteristics of *n. fibularis communis* in a larger and homogenous cohort of patients suffering from peroneal neuropathy, to determine whether nerve enlargement and hypoechoism are viable tools in the diagnosis of peroneal neuropathy. These measurements could also be compared between the controls and the patients suffering from peroneal neuropathy.

N. fibularis communis of eight specimens, prelevated from human cadavers, were visualized using Masson's trichrome staining. The histological exploration allowed us to examine the microstructure and architecture of the nerve. We were mainly focussed on the amount of the interfascicular adipose tissue in thick and thin cadavers. Although the amount of intraneural fat changes during the course of the nerve, we examined *n. fibularis communis* at the level of the fibular head and after its bifurcation into *n. fibularis superficialis* and *n. fibularis profundus*. By using high-field MRI, we tried to obtain the same level in each specimen. In a first observation we distinguished differences in the amount of interfascicular fat in *n. fibularis communis* between the thick and the thin specimens. When the cadavers were more obese, more interfascicular fat was visible. On the histological sections, we saw that more adipose tissue was present in *n. fibularis superficialis* as well as in *n. fibularis profundus*.
An estimation of the intraneural fat ratio was made on these histological sections using a low magnification. To overcome bias, the histological measurements were carried out by one observer, after discussion in group. At each level in the specimen, the mean value of the intraneural fat percentage was calculated from three consecutive sections. In addition, an average was made from the obtained fat percentages of the thick (N=3) and thin (N=5) specimens. The number of specimens was too small to apply statistical analysis.
In the thick cadavers (N=3) the mean intraneural fat ratio was 60%. This value was by average 33,8% in the thin cadavers (N=5). Quantification of the intraneural fat ratio confirmed more interfascicular fat in *n. fibularis communis* of the thick cadavers. The thick cadavers (N=3) contained a mean intraneural fat ratio of 46% and 56% for *n. fibularis profundus* and *n. fibularis superficialis*, respectively. In the thin cadavers (N=5), *n. fibularis superficialis* and *n. fibularis profundus* contained both a mean intraneural fat ratio of 28%. In general, the thick cadavers demonstrated more interfascicular fat in thin cadaver.
Due to the replacement of blood by balm-fluid, the exact weight of the cadaver couldn't be assessed. Consequently, it was not possible to calculate the BMI of the cadavers and the subcutaneous fat was measured at two different locations: superficial to *n. fibularis communis* at the region of the fibular head and at the iliac crest. In this study, positive relationships were

observed between the subcutaneous fat at the two different locations and the ratio of intraneural fat present in the nerve. Using US, a significant positive correlation was found between the BMI and the subcutaneous fat superficial to *n. fibularis communis*, shown in figure 3.3A. This confirmed that when more subcutaneous fat was present, the nerve could have a higher intraneural fat percentage in *n. fibularis communis*, as well as in its two final branches.

In conclusion, the ultrasonographic and histological exploration of *n. fibularis communis* has shown that the gross appearances and the amount of intraneural fat is different between the thin, people with a low BMI and obese, people with a higher BMI, persons. Through correlation studies we have positively correlate these difference to the presence of adipose tissue on other locations, e.g. BMI, subcutaneous fat superficial to *n. fibularis communis* and at the location of the iliac crest.

It has been described that rapid weight loss is associated with peroneal neuropathy.[1, 4, 22, 43] In literature, next to *n. fibularis communis*, no other nerve is has been reported to be complicated after weight loss. No explanations are available for this peculiar observation. For this reason it is important to evaluate other nerves that are frequently involved in a compression neuropathy but not associated with weight loss. Herein, we prelevated *n. ulnaris* of the left elbow of a thick cadaver. In cubital tunnel syndromes, *n. ulnaris* can be compressed in the cubital tunnel of the elbow. [5] This nerve was chosen for its anatomical similarities with *n. fibularis communis*. Both nerves have a superficial course before entering the osteofibrous tunnel and are located in an extremity. After visualization with Masson's trichrome staining, the amount of intraneural fat of the nerve could be evaluated. Quantification of intraneural fat ratio of *n. ulnaris* revealed a mean value of 37%. This value was lower in comparison to the other mean values measured in *n. fibularis communis* (55%), *n. fibularis profundus* (52%) and *n. fibularis superficialis* (76%) of the same thick cadavers. Due to the fact that only one nerve was evaluated, no conclusions can be made. In the future, a higher number of *n. ulnaris* in specimens, prelevated from thin and thick cadavers must be evaluated and the study must also extend to other peripheral nerves

In literature, the prevalence of peroneal neuropathy has not been defined. In an epidemiological study, we try to evaluate the extent of this problem in patients, who underwent gastric banding. The patients were used for this study because they undergo rapid

and marked weight loss, indicating a good model for assessing peroneal neuropathy after weight loss. Approximately 3% of the patients showed symptoms of peroneal nerve entrapment. On average, these 3% had lost 35,8 kg (representing 32% of their total bodyweight) during a period of 18,5 months. In addition, the treatments of the patients with peroneal neuropathy symptoms were evaluated. Although the good outcomes, only one patient was treated with surgical decompression. Two patients received conservative treatment, but remained showing signs of peroneal neuropathy. The rest were untreated, mainly due to unawareness of their compression neuropathy. As a future direction, the amount and rapidity of weight loss must be assessed in the controls.

It has yet to be investigated how a decrease in intraneural fat could lead to peroneal neuropathy. In literature this subject is to our knowledge unexplored. We postulate that weight reduction will deplete fat stores, subsequently resulting in the disappearance of subcutaneous fat and intraneural adipose tissue. During this study we have observed that in thin cadavers the intraneural fat ratio was lower than in thick cadavers. Hence, we hypothesize that upon weight loss, this intraneural fat could disappear. Adipocytes have been classically viewed as a storage depot for lipids. It is present throughout the entire body and serves as a protective layer against mechanical insults. When weight loss occurs in a short period, the body is not adapted to the loss of the intraneural adipose tissue and the nerve will become more prone to mechanical injury, resulting in an impairment of *n. fibularis communis*. It should be noted that fat functions as an endocrine organ, secreting adipocytokines, e.g. leptin, adiponectin, resistin, interleukin 6 (IL-6) but also insulin growth factor 1 (IGF-1), adenosine, plasminogen activator inhibitor, and tumor necrosis factor alpha (TNF-a). [48] Therefore the fast catabolism of adipose tissue could have metabolic consequences. Hence, it is important to look for the metabolic changes that occur during weight loss. In addition, adipose tissue has a high blood supply. Upon weight loss this blood supply could be lost when adipose tissue disappears leading to a decrease in vascularization and subsequently ischemia. This also could provide a possible explanation why the nerve is more prone to ischemia during severe weight loss.

In conclusion, it is important to investigate the role of adipose tissue in the development of peroneal neuropathy after weight loss. Because of its high blood supply, great metabolic and protective function, the disappearance of intraneural adipose tissue could be an important factor in the development of peroneal neuropathy after weight loss. It must be noted that

peroneal neuropathy is more likely to be caused by more than one factor. Other factors could be involved in the pathophysiology of this condition. It is important to consider that by releasing the nerve from compression, using surgical decompression, peroneal neuropathy is resolved. In the future, investigations at the anatomical, histological and molecular levels are necessary to confirm these hypothesis. In order to unravel peroneal neuropathy after weight loss, it is necessary to focus on the exploration of the micro- and macrostructure of *n. fibularis communis* in pathological legs.

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Supplement I

Table 1 *Overview of the nerves that are innervated by the* n. fibularis superficialis *and* n. fibularis profundus.

n. fibularis superficialis	
Muscle	***Function***
m. peroneus longus	*Evert the foot*
m. peroneus brevis	*Evert the foot*
n. fibularis profundus	
Muscle	***Function***
m. tibialis anterior	*Dorsiflexes ankle and inverts the foot*
m. extensor digitorum longus	*Dorsiflexes the ankle and extends lateral four digits*
m. extensor digitorum brevis	*Extends the lateral four digits*
m. extensor hallucis longus	*Dorsiflexes the ankle and extends the great toe*
m. extensor hallucis brevis	*Extends the great toe*
m. peroneus tertius	*Dorsiflexes the ankle and aids in eversion of the foot*

Vragenlijst

Naam: ……

Geboortedatum: ……/……/19…..

Telefoonnummer: ……..

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1. Heeft u na het plaatsen van een maagring ooit een verminderde kracht in de voet ervaren of zijn er ooit klachten van een dropvoet geweest?

Ja
Nee

Zo ja:

1.1 Wanneer zijn deze klachten ontstaan (periode na het plaatsen van de maagring)?

1.2 Hoe ernstig was de krachtsvermindering in de voet?

Duid het vakje aan dat het best de toestand beschrijft.

Volledig normale kracht.
Nauwelijks verminderde kracht, niet storend in het dagelijks leven.
Verminderde kracht, valt op in dagelijks leven.
Verminderde kracht, storend bij de activiteiten van het dagelijks leven.
Sterk verminderde kracht, duidelijke weerslag op het gebruik van het been.
Kracht zeer slecht, nauwelijks bruikbare spieren.

Hoe ernstig was de krachtsvermindering volgens u? Trek een verticaal streepje op onderstaande schaal tussen 0 en 10, waarbij 0 betekent dat u een zeer sterk verminderde kracht had en 10 betekent dat u een volledig normale kracht had:

0 ⊢——————————⊣ 10

Sterk verminderde kracht — Normale kracht

1.3 Zijn deze klachten spontaan verdwenen?

Ja
Nee

Zo ja:

1.3.1 Hoe lang na het ontstaan van de klachten?

1.4 Heeft er een behandeling plaats gevonden?

Ja
Nee

Zo ja:

1.4.1 Welke behandeling ?

Chirurgie
Kinesitherapie

2. Heeft u na het plaatsen van een maagring ooit tintelingen in de voet of het been ervaren?

Ja
Nee

Zo ja:

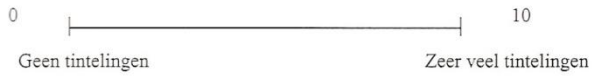
2.1 Wanneer zijn deze klachten ontstaan (periode na het plaatsen van de maagring)?

2.2 Hoe ernstig waren deze tintelingen in de voet of het been?

Ondervond u een abnormaal gevoel in het been of de voet, zoals tintelingen, prikkelingen, … (gevoel of "men met tientallen fijne naaldjes in de huid prikt")? Duid het vakje aan dat het best de toestand beschrijft:

Geen tintelingen.
Nauwelijks tintelingen.
Af en toe tintelingen, niet storend.
Regelmatig tintelingen, wel storend.
Veel tintelingen, quasi continu aanwezig.
Zeer veel tintelingen, erg storend en onhoudbaar.

Hoe storend waren deze tintelingen voor u? Trek een verticaal streepje op onderstaande schaal tussen 0 en 10, waarbij 0 betekent dat u helemaal geen tintelingen ondervond en 10 betekent dat u zeer veel en extreem storende tintelingen ondervond:



2.3 Zijn deze klachten spontaan verdwenen?

Ja
Nee

Zo ja:
2.3.1 Hoe lang na het ontstaan van de klachten?

2.4 Heeft er een behandeling plaats gevonden?

Ja
Nee

Zo ja:

2.4.1 Welke behandeling ?

Chirurgie
Kinesitherapie

3. Heeft u na het plaatsen van een maagring ooit een verminderd gevoel in de voet of het been ervaren?

Ja
Nee

Zo ja:

3.1 Wanneer zijn deze klachten ontstaan (periode na het plaatsen van de maagring)?

3.2 Hoe ernstig was deze gevoelsvermindering in de voet of het been?

Ondervond u een verminderd gevoel, alsof er een dof gevoel was in het been of voet, alsof ze leken te slapen? Duid het vakje aan dat het best de toestand beschrijft:

Geen dof gevoel.
Nauwelijks een dof gevoel.
Een beetje een dof gevoel, niet storend.
Lichtjes een dof gevoel, wel storend.
Duidelijk verminderd gevoel, storend.
Ernstig verminderd gevoel, erg storend.

Hoe storend was dit verminderd gevoel voor u? Trek een verticaal streepje op onderstaande schaal tussen 0 en 10, waarbij 0 betekent dat u een normaal gevoel had in het been en/of voet en 10 betekent dat u een zeer sterk verminderd en dof gevoel ondervond:

Normaal gevoel Ernstig verminderd gevoel

3.3 Zijn deze klachten spontaan verdwenen?

Ja
Nee

Zo ja:

3.3.1 Hoe lang na het ontstaan van de klachten?

3.4 Heeft er een behandeling plaats gevonden?

Ja
Nee

Zo ja:

3.4.1 Welke behandeling ?

Chirurgie
Kinesitherapie

4. Heeft u deze krachtsvermindering, tintelingen of gevoelsvermindering ook elders in het lichaam ervaren ?

Ja
Nee

Zo ja:

4.1 Waar situeerde deze klachten zich precies ?

4.2 Zijn deze klachten spontaan verdwenen ?