The Influence of a Mobilisation of the Lesser Omentum on the Capacity of the Portal Vein, Measured with Echo-Doppler

Master Thesis zur Erlangung des Grades

Master of Science in Osteopathie

an der **Donau Universität Krems**

niedergelegt

an der **Wiener Schule für Osteopathie**

von **Hanno Halbeisen**

Dornbirn, November 2008

Tutor: *Patrick van Dun, DO*

Co-Tutor: *Mag. Katharina Musil*

Eidesstattliche Erklärung

Hiermit versichere ich, die vorgelegte Master Thesis selbständig verfasst zu haben.

Alle Stellen, die wörtlich oder sinngemäß aus veröffentlichten oder nicht veröffentlichten Arbeiten anderer übernommen wurden, wurden als solche gekennzeichnet. Sämtliche Quellen und Hilfsmittel, die ich für diese Arbeit genützt habe, sind angegeben. Die vorliegende Arbeit hat mit gleichem Inhalt noch keiner anderen Prüfungsbehörde vorgelegen.

Datum:_____ Unterschrift:_____

Preface

Hiermit möchte ich mich bei allen bedanken, die mich bei dieser Arbeit unterstützt haben:

- Herr Prim. Dr. Guntram Winder, der einen Teil der Ultraschallmessungen durchgeführt hat und mir in den entsprechenden Räumlichkeiten des Krankenhauses der Stadt Dornbirn die Untersuchungen ermöglichte.

- Herrn OA Dr. Bertram Kohler, der sich als erster spontan bereit erklärt hat Ultraschallmessungen durchzuführen und so auch ein wichtiger Wegbegleiter dieser Studie war.

- Herrn Patrick van Dun, DO der mir in einer Vorlesung an der WSO die Wichtigkeit der Grundlagenforschung näher gebracht hat. Seine Anregungen und Tipps waren während der Arbeit ständig eine große Hilfe.

- Frau Mag. Katharina Musil für die Betreuung der Master Thesis.

- Herrn Uwe Staffa, MSc, DO und Herrn Marcel Mätzler, MSc, DO welche spontan bereit waren trotz spärlicher Freizeit die Mobilisationstechniken durchzuführen.

- Herrn Dr. Gebhard Woisetschläger für die statistische Auswertung der Ergebnisse.

- Herrn Jéromê Helsmoortel, DO (1952-2007), der mir in meiner osteopathischen Grundausbildung eine oft etwas andere Denkweise hinsichtlich verschiedener Aspekte und Funktionsweisen des menschlichen Körpers beigebracht hat.

- Meinem Bruder Mario, der mir während der Untersuchungen und im organisatorischen Bereich ständiger und mehr als treuer Wegbegleiter war.

- Ganz besonderer Dank gilt meiner Frau Astrid, meinen Kindern Pius, Max und Luis, welche mich über längere Zeit entbehren mussten und für die ich jetzt wieder mehr Zeit haben werde.

Abstract

Objective: It was the aim of this study to examine physiological changes by a visceral osteopathic intervention. In special: Does a mobilisation of the lesser omentum influence the capacity of the portal vein?

Subjects and Methods: Twenty healthy male adults (age 23-40) were assigned to two equivalent groups. Groups were matched by age, body height and body weight. Inclusion criteria were a BMI ≤ 27, age 18-45 years, non-smoking and alcohol intake <25 g/day. Additional exclusion criteria were drug intake, inflammatory diseases, abdominal surgical interventions and known heart or liver pathologies.

The members of the test group (mobilisation group/MT, n=10) were treated with a mobilisation technique for the lesser omentum, the members of the sham group (non-specific-technique group/NST, n=10) with a manual abdominal compression in the height of the navel in direction of the spine. Both techniques were applied for three minutes.

Participants as well as examining medical specialists were blinded against the administered intervention.

The measurement of the portal venous diameter and mean blood flow velocity in the portal vein was performed at four moments. For each measurement, the capacity of the portal vein was calculated based on these data.

Two trials, one initial medical examination (x1) and half an hour later another comparison measurement (x2) were performed for the assessment of the baseline.

The third measurement (y) was done directly after administration of the particular intervention technique and the fourth one hour later (z).

Characteristics of the portal vein were measured in decubitus position left of the subjects by means of a 3.5 MHz transducer with colour-coded duplex sonography. The flow velocity was measured by means of a PW-Doppler ultrasound device Toshiba Xario. Statistical software used for evaluation was R 2.7.1 and SPSS® 14.0.

Results: The results of the two baseline measurements (x1, x2) of the portal vein capacity as well as the test-to-test variability in the NST group and MT group were comparable.
In contrast, heart rate, blood pressure, the diameter of the portal vein and the blood flow velocity were not stable but might indicate relaxation effects.
The results of the baseline measurement of the parameters capacity, diameter and flow velocity are assorting well with literature data.

After the interventions (measurement y) capacities increased by approximately 1.3 ml/min/kg body weight could be observed (MT group: mean (standard deviation): 11.3 (4.0) ml/min/kg, NST group: 11.5 (6.2) ml/min/kg).
The increase in capacity is not significant in either group, but slightly more distinct in the MT group, due to the lower base data (MT group: p=0.10, NST group: p=0.33).
These group differences in the change in capacity are not significant, either (ANOVA: p=0.97).
One hour after the intervention (measurement z), a mean flow of 10.0 (3.4) ml/min/kg body weight could be observed in the MT group and of 9.6 (2.6) ml/min/kg in the NST group. In both groups the base level was re-established.

Conclusion: The current data show, that there is no significant difference in the effects on the capacity of the portal vein between the specific visceral mobilisation technique for the lesser omentum and a non-specific technique with a manual abdominal compression in the height of the navel in direction of the spine.
The validity of these results above all is restricted by the low number of participants (10 per group).

Key words: Osteopathy, portal vein, capacity, visceral, mobilisation of the lesser omentum

Table of Contents

1. Introduction and Motivation

"The whole is greater than the sum of its parts" (ARISTOTELES).
The fact, that a body is more than the sum of its parts, becomes apparent in the circumstance, that by a decomposition of the human body into its parts only little or nothing can be learned about their interaction in a living organism.

While anatomy considers and describes the body parts, physiology scrutinises their function. Physiology describes the mode of changes, influencing the body exteriorly or interiorly and explains them. Furthermore, it describes the mechanisms, the body uses for living.
Thus, physiology is the teaching of life processes. Physiology teaches and researches the normal organ- and body functions [DIVISION OF PHYSIOLOGY 2008].
Without physiology, neither an effect nor an explanation is possible [VAN DEN BERG 2003]. Therefore, it should also be regarded as basis of osteopathic knowledge.

This was my motivation for this master thesis, which will be further described by the following quotation:
"If you have to judge the importance of fundamental research, you will have to retrospect. You will have to see, how things have been discovered, which are important nowadays. Often knowledge was gained that could not be put in a context at the moment of its detection. In a manner of speaking, man came across them by chance. Scientists, working in their special field, of course, did their research purposely. But in the moment of understanding they often could not anticipate, what would follow thereafter" [TRACHSEL 2008; translation by myself].

Although osteopathic work in manual diagnostics and therapeutic intervention can be administered on visceral structures, too (apart of parietal and craniosacral elements), you come across results far from satisfactory during the search for scientific explanations for the working mechanisms of certain techniques in the visceral field [VAN DUN et al. 2007].
Therefore, this thesis is intended as a complementary scientific contribution in this respect.

Further motivation is the opinion of medical specialists in my professional circle, that any pressure applied anywhere to the abdomen leads to an increase in the perfusion in the portal vein. However, up to now, I could not find appreciable scientific investigations and results by literature research that would attest this position.

Therefore, I want to find an answer to the following question: *Can osteopaths truly achieve measurable physiological changes by specific visceral techniques (mobilisation of the lesser omentum)?*

The importance of a sufficient blood supply within the visceral system was occasionally stressed during my osteopathic training. For this master thesis in particular the question arises, if a mobilisation of the lesser omentum modifies the capacity of the portal vein.

In this respect, different hypotheses can be found in osteopathic literature. For example, an author describes a compensation mechanism of the portal vein arising from mechanic stimulation as follows:

"An increase of the pressure gradient in the venous system emerges from pressure and pulling on the lesser omentum. This is a compensation mechanism via the reaction of the portal vein" [LIEM et al. 2005; translation by myself].

In contrast, another author refers to the pulling force on the lesser omentum, leading to a central venous decongestion:

"The mesos encounter pulling forces. They compress the nerves and vessels running inside of them (like a waffle iron the pastry). […] Additionally, the blood in the large veins is decongested to central by a traction" [HELSMOORTEL et al. 2002; translation by myself].

It is the aim of this experimental study to investigate potential physiological changes in the portal vein caused by a manual intervention, in special by a specific osteopathic visceral technique on the lesser omentum.

The null hypothesis underlying this study is:
A mobilisation of the lesser omentum has no influence on the blood flow in the portal vein. Additionally, no physiological alterations can be achieved.

Scientific studies for the determination of the portal blood flow were performed several times in the past by means of echo-Doppler ultrasound. They are pointing out highly reliable results [IGNEE et al. 2002, HUCK 2004, BOMBELLI et al. 2005]. Furthermore, these ultrasound examinations have no adverse effects and they are inexpensive [KUBALE / STIEGLER 2002, DEGUM 2004, DEGUM 2006].

Due to my local possibilities, the support by medical specialists in the sonographic measurements and the sanitary and economic arguments mentioned before, this experimental fundamental research study was brought into being in the sense of a scientific contribution for osteopathy.

2. Hemodynamic Fundamentals

In this section of the master thesis, I will describe the fundamental terms and contexts necessary to understand the following chapters. On the one hand, these are physical terms, as e.g. volumetric flow rate, pressure and flow resistance – on the other hand these are physical laws, as *Ohm's* or the *Hagen–Poiseuille law*.
These terms and laws serve for a better understanding of the physiologic changes that shall be investigated in this study. Thus, at first, I want to provide a first insight in the physical behaviour of blood within the human vascular system.

Generally, the flow behaviour of fluids is described by the laws of hydrodynamic. The field of hemodynamic, in special, is dealing with the flow behaviour of blood. Blood is a mixture of plasma and cellular constituents, with the erythrocytes as the largest fraction. Hemodynamic regularities can be applied to those vessels that are normally accessible for Duplex sonography, i.e. with a diameter of 0.5 mm or greater [KUBALE / STIEGLER 2007].
The portal vein has a diameter of between six and twelve millimetres [IGNEE et al. 2002] or between seven and fifteen millimetres [HUCK 2004] and thus is underlying these regularities.

Like all other fluids, blood has a certain property, called *viscosity,* that is determining the force that has to be applied in order to shear neighbouring fluid layers against each other. On the one hand, blood viscosity is dependent on the volume proportion of the haemoglobin-filled erythrocytes, on the other hand on plasma viscosity and vessel diameter [HARTEN 2007, KUBALE / STIEGLER 2007].

2.1. The Connection of Volumetric Flow Rate, Pressure and Flow Resistance

Two physical factors determine the blood flow in a blood vessel:

1. The pressure difference from the beginning to the end of the particular blood vessel as driving force.
2. The flow resistance (vascular resistance) counteracting this force.

The volumetric blood flow rate (I) defines the blood volume per time unit (ml/sec, ml/min, l/min) perfusing a vessel. It is directly proportional to the pressure difference (Δ p) from the beginning to the end of the perfused vessel and inversely proportional to the flow resistance (R).

These connections can be clearly observed in a law in the style of *Ohm's law* for electricity [HARTEN 2007].

$$I = \frac{\Delta p}{R}$$

Δ p = pressure difference
I = volumetric blood flow rate
R = flow resistance

However, this equation does not take into account pressure changes caused by pulsation. Therefore, it is valid for the temporal mean, only. That means the pressure difference is calculated using the difference of the mean arterial and mean venous blood pressure of a vascular area [KUBALE / STIEGLER 2002].

The physician and physiologist *Poiseulle (1799 – 1869)* detected, that the flow velocity of liquids is dependent on the length and diameter of the vessels, as well as on the pressure difference. He drafted the *Hagen-Poiseuille law* (which was originated by *Gotthilf Hagen* in *1939*), describing the flow velocity of liquids in narrow tubes.
This law is valid for fluids with constant viscosity and laminar flow, only. According to this law, the volumetric flow rate is determined by the pressure difference, the viscosity of the medium and the length and radius of the vessel [KAMKE 1994, HARTEN 2007].
This law implies the following: *"The flow resistance is directly proportional to viscosity and the length of the vessel and indirectly proportional to the fourth power of the vessel radius."* Consequently, small changes in radius are causing great changes of the resistance and at constant pressure difference great changes of volume flow rate, too [KAMKE 1994, SILBERNAGEL / DESPOPOULOS 2001, HARTEN 2007].

Since this law describes the perfusion velocities of fluids through tubes, the flow velocity shall be viewed more precisely.

2.2. Flow Velocity

The *Hagen-Poiseuille* law implies that pressure difference and vessel radius are factors, which influence the flow velocity [KAMKE 1994, HARTEN 2007].

$$I = \frac{\pi \cdot r^4 \cdot \Delta p}{8 \cdot \eta \cdot l}$$

I = volumetric flow rate
r = radius of tube
η = dynamic viscosity
l = length of tube
Δ p = pressure drop

According to the equation above, a halfing of the vessel radius at constant volumetric flow rate leads to a quadruplication of the flow velocity.

The principle of continuity implies that flow velocity is lower in tubes with large radius, than in tubes with small radius. That means flow velocity is indirectly proportional to the perfused profile [KAMKE 1994, HARTEN 2007].

Therefore, the accuracy of the measurement of the diameter of the portal vein is of importance for the examination of the capacity of the portal vein. The measured diameter influences the measurement result substantially.
A more precise description of the standardisation of this point can be found in chapter 8 (Methodology).

3. Hemodynamic of the Arterial Vascular System

The circulatory hemodynamic processes (they will be further described in chapter 5) show besides others, that the arterial vascular system can have an influence on the perfusion of the portal vein, either by an inter-relation with the hepatic artery or by the arteriolar resistance vessels in the splanchnic region [LAUTT / GREENWAY 1987, TAKALA 1997, WIEST et al. 2000].

Apart from the tasks of the arterial vascular system, I will explain the regulation of the flow rate by the local peripheral resistance. Apart from this local peripheral resistance, pressure regulation caused by hemodynamic reflexes can co-influence the blood flow in the portal vein, too, as will be described in this chapter.

3.1. Tasks of the Arterial Vascular System

The arterial vascular system fulfils two fundamental purposes. One is to modify the exclusively systolic pressure and volume shift into a steady systolic/diastolic pressure and volume transport. This task is accomplished by the elastic properties of the arterial walls in the trunk and is designated as “Windkessel function”.

Apart from this general task serving the overall blood circulation, another purpose of each arterial vessel is to supply the according organ with the currently necessary portion of blood as a transport vessel [HUCK 2004, BENNINGHOFF / DRENCKHAHN 2008].

3.2. Flow Rate and Peripheral Resistance

The blood volume, running through a vessel to an organ per time unit is designated as flow rate, as was described more in detail in chapter 2. Its unit is millilitre per second [ml/s] or millilitre per minute [ml/min].
The flow rate is adapted to the particular state of functioning of an organ via a change of the local peripheral resistance, that is primarily determined by the arterioles and precapillar sphincters of the organ provided for.

On high demand, these sphincters are set wide and they allow a high inflow into the corresponding capillary bed from the supplying artery. If less blood is needed, afflux from the arteries is curtailed by setting them narrow.

The relationship between local inflow and peripheral resistance is described by Ohm's law. Since blood – as each other fluid – is not compressible and since the blood stream cannot break down in an intact vessel, the same volumetric flow rate can be observed in each complete profile of the circulatory system.
This accounts not only for the local volumetric flow rate in a ramified supplying vessel but also for each single unramified vessel.

While the flow rate in a certain condition of the systemic circulation is longitudinally constant in a part of the systemic circulation with its ramifications or in the course of a single unramified vessel, the flow velocity varies in dependence of the vessel diameter. The flow velocity increases by vasoconstriction and decreases by vasodilation.

Apart from the volumetric flow rate, the hemodynamic situation of an arterial vessel can be characterised by means of the blood pressure, too. In contrast to the volumetric flow rate, the systolic and the diastolic blood pressure are not equal all over the circulatory system [HUCK 2004, HARTEN 2007, BENNINGHOFF / DRENCKHAHN 2008].

3.3. Arterial Pressure Regulation by Hemodynamic Reflexes

FINET UND WILLIAME (2002) describe the following special arterial reflex activity for the regulation of arterial blood pressure in connection with hemodynamic and visceral dynamic.

This pressure regulation of the arterial vessels is dependent on a system, consisting of two parts, which is denominated as „baroreceptor system":

- The *ramus enteroseptivus nervi vagi* (efferent parasympathetic fibres on the cardial branch of the vagus nerve also called *Cyon-Ludwig's nerve*):

The receptive fibres, which disperse in the endothelium of the arcus of the aorta run with the vagus nerve to the medullar bulb. This nerve has a moderating function, its stimulation ends in hypotension by cardial moderation and by active vasodilatation and inhibition of the vasoconstrictive tonus [FINET / WILLIAME 2000].

- The *ramus sinus carotici* (carotid branch of the glossopharyngeal nerve, also called *nerve of Hering*):
  It originates from the bifurcation of the common carotid artery, where it ramifies into the internal and external artery. The baroreceptors are located at the bifurcation. The carotid body (glomus caroticum) comprises chemoreceptors that are sensible for oxygen - and carbon dioxide concentration.

  The aggregated nerve endings of the ramus sinus carotici run to the medullar bulb with the glossopharyngeal nerve. This nerve is inhibitory active and is equal in function to the ramus enteroseptivus nervi vagi, although the ramus sinus carotici is much more sensitive.

  Summing up, pressure regularises pressure – so that even the mechanism of such a regulation seems to be possible via reflexes, only. The capacity for a vasomotoric change represents the main element of this reaction, and the reflexes are the basis [FINET / WILLIAME 2000].

4. Hemodynamic of the Venous System

On the one hand, the venous system differs from the arterial system in anatomical vessel structure, on the other hand in physiology and in the reaction to the different requirements and tasks, as e.g. the filling pressure [HUCK 2004, BENNINGHOFF / DRENCKHAHN 2008].

At first, apart from the task of the venous system, I will describe the volume- and pressure distribution in this low-pressure system, followed by the experiments by GLÉNARD (1899) and the explanations by HELSMOORTEL (2002), in which the portal vein is integrated in a compensation mechanism of the liver for reattaining its spatial position.

4.1. Tasks of the Venous System

The venous system of the systemic circulation, the right heart, the lung vessels and the left atrium together constitute a functional unit that is called “low-pressure system” because of the low mean blood pressure compared to the blood pressure in the arterial vascular system.

It is an essential task of this heterogeneous part of the systemic circulation, to grant a heart-time volume, which is adapted to the particular condition of the systemic circulation by an adequate diastolic filling of the left ventricle with oxygenated blood.
For this purpose, the deoxygenated blood flowing in from the capillaries is collected and stored in the venous sections of the systemic circulation, and then runs to the right heart according to its needs.
Hence, the crucial tasks of the venous sections of the systemic circulation are its storage- and transport function [HUCK 2004].

For storing a sufficient amount of blood, the vessels of the low-pressure system have to feature a high distensibility in comparison to the arteries.
The pressure-volume relation illustrates the relation between filling pressure and filling volume (cf. Fig.1). The unfilled venous vessel features no considerable pressure. Pressure in the vessel increases only slightly up to a certain degree of

filling. A distinct increase in pressure can be observed not before the vascular wall puts up a resistance to further distension.

Shape and size of the vessel profile change characteristically depending on the state of filling. A vessel, which is barely filled, has a profile of an eight. The vessel unfolds with increasing filling, accompanied by an only slight increase in pressure, to an oval and finally circular profile. Not before a circular profile is achieved, a distinct increase in pressure can be observed with further filling [HUCK 2004, SIEGENTHALER / BLUM 2006].

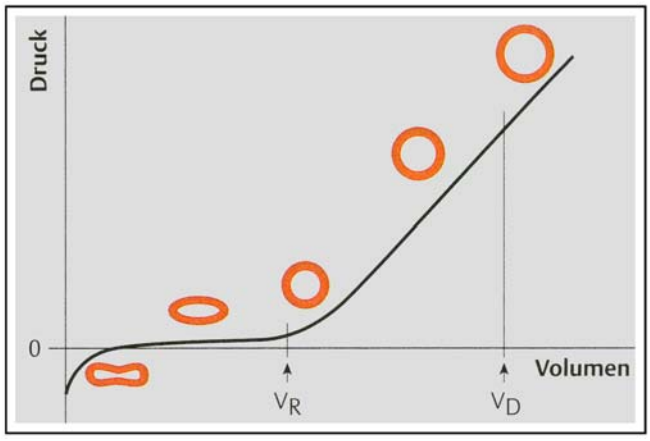


Fig. 1: With increasing filling, the profile of a venous vessel changes from oval to circular (according to Huck 2004)
VR = filling volume at rest
VD = active distension volume at rest.

4.2. Volume- and Pressure Distribution in the Low-Pressure System

The pressure difference between perivascular and intravasal pressure is called “transmural pressure“. If perivascular and intravasal pressure are equal, the transmural pressure is equal null.

In many physiological and pathophysiological situations, the perivascular pressure differs from the intravasal pressure.

Respiration, for example, modifies the perivascular pressure conditions in the rhythm of inspiration and exspiration, whereby the particular intrathoracic and extrathoracic changes in pressure are opposed.

An inspiration leads intrathoracically to perivascular decompression, while extrathoracically it causes an increase in pressure by the lowering of the

diaphragm. During exspiration conditions are the other way round [FINET / WILLIAME 2000, SIEGENTHALER / BLUM 2006].
A momentary extreme increase can be observed during diverse events accompanying the abdominal press, particularly during a Valsalva-maneuvre, but also during coughing and sneezing [HUCK 2004].

Apart from the respiratory system, there are further possibilities for the volume- and pressure distribution, as will be shown in the following section dealing with the liver.

GLÉNARD (1899) describes his observations and investigations concerning this matter in his 1000 - pages book *Les Ptoses viscérales*.
He compares the liver with a bunch of grapes, hanging on their stem. The stem is the area nuda, representing an adhesion field, by which the liver is connected with the diaphragm upwards at the back.
In an experiment, the liver was filled with approximately one litre of a liquid by means of an injection into the portal vein. As a result, the liver erected on its shaft forwards, upwards and to the right side by itself.
From this, *Glénard* concludes, that the liver is autonomously able to stay on its place. According to him, this fact explains, too, that an enteroptosis does not imperatively implicate a ptosis of the liver, if the liver can hold its position on its own ability.

HELSMOORTEL (2002) interprets a loss of position of the liver on the basis of *Glenard's* understanding, as follows:
A hypotense liver, losing its position by its weight, is administering pressure to the organs below and in consequence "squeezes them out" to a higher extent. The resulting increased blood volume is pressed into the portal system and necessarily passed on to the liver. This elevated filling pressure during a longer increased tonus of the „wall" leads to an increase in tension of the liver. This enables the liver to erect by itself and to acquire its normal position.

Helsmoortel uses the term "tension" for the characterisation of the quality of the elastic expression of the viscera and organs. Hence, according to him, tension arises from the pressure of the content and from the tension of the wall of the viscera or organs.

5. Hemodynamic of the Liver

The physical factors in hemodynamic fundamentals and the vascularisation of the liver are able to influence the capacity of the portal vein. As already described in chapter 2, hemodynamic describes the blood flow in dependency on the accountable forces – *flow rate, pressure and flow resistance.*

In this chapter, I will go into detail of those aspects that are in direct connection with the topic of this work. Starting with an overview about the dual vascularisation of the liver, I will describe the afferent and efferent blood vessels. The regulation of the hepatic perfusion takes place at *macroscopic level* as well as in the field of the *interaction between the hepatic artery und the hepatic portal vein* and finally also at *micro-level*. In the course of this, *intrinsic as well as extrinsic influences* play a crucial part. In this section, I will concentrate on these.

5.1. General Information about the Vascularisation of the Liver

The hepatic total flow in human beings at rest averages 800–1500 ml/min. In the point balance, the perfusion of the liver accounts for approx. 25% of the heart-time volume and thus the liver belongs to the best-perfused organs of the body [LAUTT / GREENWAY 1987, WIEST et al. 2000].

The liver is supplied with blood via the hepatic artery and parallely via the portal vein in a ratio of 20-25% to 75-80% [SCHENK et al. 1962, ROCHELEAU et al. 1999, WIEST et al. 2000].

The blood of the hepatic portal vein flows through the junction of the splenic vein, the inferior mesenteric vein and of the superior mesenteric vein. The hepatic artery proper originates from one of the terminal branches of the common hepatic artery descending from the celiac arterial trunk.

The portal vein delivers the venous and deoxygenated blood from the spleen, the stomach, the intestine, and pancreas into the liver. Its finer ramifications envelop the smallest terminal vessels of the liver, the so-called sinusoids.

The hepatic artery supplies the liver with oxygenated blood.

The blood of these two separate vascular systems mixes in the hepatic sinusoids and runs into the inferior vena cava via the hepatic veins. The portal vein receives

its afflux from the abdominal venous vessels and thus it reflects the whole perfusion of the splanchnic region [BENNINGHOFF / DRENCKHAHN 2008].

Although the portal vein contributes most to the total liver blood flow with approximately 70%, its contribution to the oxygen supply for the liver is only 50%, since the blood from the portal vein is partially de-oxygenated during the passage through the capillaries of the gastrointestinal tract.

The proportions of the dual liver blood supply of the hepatic artery and the portal vein particularly vary in dependency on ingestion [LAUTT / GREENWAY 1987, DAUZAT et al. 1994]. Apart from this, the flow in the portal vein is relevantly dependent on the perfusion of the splanchnic organs and thus of the resistance in the arterioles ahead of the gastrointestinal tract [TAKALA 1997].

Influences like anaesthetics or abdominal surgical measures can also cause changes in the hepatic circulation [ROTHE et al. 1998].

5.2. The Vascularisation of the Liver

In the initial stages of liver development, a right and left umbilical vein, a right and left vitelline vein and the bilateral cardinal veins are laid out. These vessels join the right and left sinus horn.

The both vitelline veins have a close relation to the endoderm and run directly aside of the intestinal tube. They are interconnected across the midline by three transverse anastomoses, of which two run in front of and one behind the intestinal tube.

The sprouting liver parenchyma builds up connections to the veins, and a network of further blood sinus develops, interspersing the liver parenchyma. A uniform feeding vessel stem, situated left of the duodenum develops by obliteration of cranial sections of the vitelline veins and of the transverse anastomoses. This is the later portal vein [BENNINGHOFF / DRENCKHAHN 2008].
The vascularisation of the liver is characterised by two pathway systems. The portal and arterial inflow takes place on the visceral surface of the liver in the region of the

hepatic porta. The efflux hilus with the according hepatic veins is located at the dorsal side of the liver. The individual vessels can be described with regard to perfusion as follows:

5.2.1. The Hepatic Artery

A multitude of variants concerning the pathway of the hepatic artery is described in literature [BENNINGHOFF / DRENCKHAHN 2008].
The most frequent pathway of the hepatic artery is the origin from the celiac trunk together with the splenic artery and the left gastric artery. It reaches the *hepatoduodenal ligament* dorsal of the duodenum.

BOUCHET / CUILLERET (2001) describe this part as *vascular part of the lesser omentum*. Most often the artery ramifies into a right and left branch immediately before its entry into the liver parenchyma. I will not elaborate on further single ramifications that can be observed intrahepatically [BENNINGHOFF / DRENCKHAHN 2008].

5.2.2. The Portal Vein

In the valveless efflux region of the splanchnic region, blood is transported to the liver. The structural unit develops dorsal of the head of the pancreas from the superior mesenteric vein and the lienal vein. Similar to the hepatic artery, tremendous variations are possible.
Alike the hepatic artery, the portal vein enters the liver parenchyma, where subsequent intrahepatic ramifications take place, in the hepatoduodenal ligament (vascular part of the lesser omentum).

The perfusion of the portal vein is not subject to any relevant intra-hepatic control. The maximum sphincter contraction causes an increase in pressure of the portal vein, but, however, it has no influence on its perfusion [LAUTT / GREENWAY 1987, WIEST et al. 2000].

Since the portal vein is primarily perfused by blood from the outflow region of the prehepatic organs, oxygen concentration as well as hydrostatic pressure is low

compared to the hepatic artery. The low pressure in the portal vein as well as the high flow rate, require a low vascular resistance.
The low-pressure system brings about, that the flow velocity remains almost the same when the vascular resistance changes. Therefore, the perfusion of the portal vein is dependent on the inflow from the preportal organs.

The blood in the portal vein is under higher pressure than the blood in the vena cava at the same level, and thus it can surmount the resistance of the liver circuit [TAKALA 1997, BENNINGHOFF / DRENCKHAHN 2008].

5.2.3. The Hepatic Veins

The venous outflow region of the liver begins in the sinusoidal central veins, from where the blood is drained into the hepatic veins via the sublobar veins. The hepatic veins originate inside the organ from the aforesaid sublobular veins, which are fed by the central veins.
The large branches run between the segments. They incorporate veins from vicinal segments, and thus do not abide by the supplied area of single branches of the portal vein. The three upper hepatic veins, the right and left as well as the middle hepatic vein, join the vena cava inferior directly below the diaphragm [BENNINGHOFF / DRENCKHAHN 2008].

5.3. The Regulation of Hepatic Perfusion

The hemodynamic of the liver is subject to various influences – starting from the cardiac output to the smallest hepatic vessels [BENEDIKT / PANNEN 2002]. Hence, a main factor for the regulation of hepatic perfusion is the vascular flow resistance, which underlies extrinsic as well as intrinsic regulating factors.

5.3.1. The Intrinsic Blood Flow Regulation

The intrinsic blood flow regulation is characterised by the following mechanisms:

- the classical arterial pressure - flow autoregulation
- the semiprocal relationship between the hepatic artery and the portal vein ("Hepatic Arterial Buffer Response - HABR")
- differences in connection with the portal venous and hepatic arterial blood, inducing changes of the hepatic arterial blood flow

The **arterial pressure - flow autoregulation** results from the tendency of the local blood flow to remain constant, despite changes of the systemic arterial pressure. This autoregulation tendency was attested by a certain adenosine mediated autoregulation capacity [HANSON / JOHNSON 1966, EZZAT / LAUTT 1987]. In consequence, this local adenosine release is of high importance also for intrinsic mechanisms.

These intrinsic mechanisms cause a regulation of the hepatic arterial blood flow and of the perfusion of the portal vein (and hence of the splanchnicus) in dependency on intra-hepatic regulation mechanisms. Examples are the semireciprocal change in blood flow in the hepatic artery during increase or decrease of the portal blood flow, which is denominated ***"Hepatic Arterial Buffer Response (HABR)"*** [LAUTT / GREENWAY 1987].
Changes in the portal venous blood flow produce inverse changes in flow in the hepatic artery by this mechanism, independent of oxygen demand or supply. This HABR tends to maintain the total blood flow in the liver at a constant rate which, in

turn, tends to maintain portal and intrahepatic pressures, liver blood volume and hepatic clearance of drugs and hormones steady [LAUTT et al. 1991].

The HABR is proposed to operate by the following mechanism that is based on adenosine washout. Adenosine is produced at a constant rate, independent of oxygen supply or demand. It is secreted into a very small fluid compartment that surrounds the hepatic arterial resistance vessels. If portal vein flow decreases, less adenosine is washed away into the portal blood and the accumulated adenosine leads to hepatic arterial dilatation. The second form of intrinsic hepatic arterial autoregulation is arterial autoregulation. This operates by the same mechanism, whereby a decrease in arterial pressure leads to a decrease in arterial hepatic blood flow, thus resulting in less adenosine washout into the hepatic artery blood. These intrinsic regulatory mechanisms tend to maintain total hepatic blood flow at a constant level, thus stabilizing hepatic clearance of hormones, venous return, and cardiac output. In addition, extrinsic factors such as circulating hormones, autonomic nerves, and various nutrients can interact to affect the hepatic circulation and will be described in the following chapter [LAUTT 1996].

Apart from the classical arterial pressure - flow autoregulation and the mechanism of the “Hepatic Arterial Buffer Response“, changes in the **portal venous and/or hepatic arterial blood composition** can influence hepatic blood flow additionally.

Hypercapnia, hypoxia, and the postprandial hyperosmolarity lead to an increase in the blood flow in the portal vein as well as in the hepatic artery. In contrast, changes in the pH-value cause reverse reactions in both vessels.
While a metabolic acidosis lowers the portal venous blood flow and raises the hepatic arterial blood flow, an increased perfusion of the portal vein and a flow reduction in the hepatic artery is caused by a raising pH-value [MATHIE / BLUMGART 1983].

5.3.2. The Extrinsic Blood Flow Regulation

Apart from the intrinsic influences described above, additional neuronal and humoral factors are available for the human organism for the regulation of liver perfusion. First, I will describe the neuronal factors.

5.3.2.1. Neuronal Factors

The liver is innervated by the sympathetic and parasympathetic nervous system. Branches of the nervus splanchnicus and the vagus nerve enter the liver predominantly in connection with the blood vessels or bile ducts. At that, sympathetic and parasympathetic nerves form a corresponding plexus, which ends in arterioles and venules.

The liver receives the sympathetic nerval input from the medullar level Th 6 – 9. The sympathetic nerve passes bilaterally without interconnections through the sympathetic trunk and proceeds as greater splanchnic nerves via both crura diaphragmatica to the celiac ganglion. They form the hepatic plexus, which encloses the hepatic artery and reach the hepatic porta and the parenchyma [LAUTT 1980, ROHEN 2001, BENNINGHOFF / DRENCKHAHN 2008].

The neurological mechanism that influences the hepatic perfusion, is in principle the sympathetic activity of the nervous system. Sympathetic nerve stimulation causes an increase of the arterial as well as of the portal resistance [MATHIE / BLUMGART 1983, TAKALA 1997] and thus a reduction of the blood flow and blood volume in the liver [LAUTT 1980].

The right and left vagus nerves supply the liver parenchyma parasympathetically. The right vagus nerve crosses the celiac ganglion with two thirds of its fibres and is interconnected there forming an anterior hepatic plexus for the supply of the left lobe and a posterior hepatic plexus for the supply of the right lobe [ROHEN 2007].
The capsule of the liver (Glisson's capsule), covered with peritoneal epithelium, and the falciform ligament are primarily supplied and sensory- innervated by the *rami phrenico-abdominales* of the right phrenic nerve [BENNINGHOFF / DRENCKHAHN 2008].

For the understanding of osteopathic techniques, we should know which influence a mechanic stimulation of the nerves has on the enteric nervous system. Nerves develop under pulling forces, whereby the nerve fibres grow from proximal to peripheral and establish contact with the already functioning enteric nervous system. Subsequently, they are lengthened by the growth movements of the organs. The pulling force also causes the long thin conformation of the nerves, what can be characteristically cited by the following statement:
From the biomechanical viewpoint, nerves are structures, which are pulled [BLECHSCHMID / GASSER 1978].

KUNZE et al. (1998 und 2000) could verify that a stretch on the nerve has effects on the ion channels in the nerve membrane. Neurons of the enteric nervous system have ion conducting pores on their cell bodies and cell extensions that are sensitive to pulling forces. Electric potentials are activated in the nerve by mechanic stimulation. Thus, the sensory neurons are subject to mechanic deformation.

The arterial vasoconstriction (by sympathetic stimulation) is temporal and has similar autoregulatory potentials like the intestinal vasoconstriction.

In contrast, the portal response starts slower, but the increased resistance sustains as soon as it has developed. The sympathetic nerve stimulation reduces the hepatic volume in addition to the vascular resistance, supposably by a contraction of the hepatic capacitary vessels. This reduction in volume starts slowly and persists as long as the stimulation continues [TAKALA 1997].

Apart from the maintenance of the normal vascular tonus, by this, a large part of the hepatic blood reserves can be mobilised. That way, a stimulation of the sympathetic nerve branches induces a reduction of the hepatic blood flow and hence of the blood volume, which can be quickly and precisely distributed as a response to the autonomous innervation [LAUTT 1980, ROTHE / MAASS - MORENO 1998].

The parasympathetic innervation seems to influence rather the regional hepatic blood distribution than the global hepatic flow by a stimulation of the presinusoidal sphincters [LAUTT 1980].

5.3.2.2. Humoral Factors

Under physiologic conditions, the perfusion of the liver is predominantly influenced by the vasoconstrictoric active endothelin-1 (ET-1) and the vasodilatatoric acting gaseous nitric oxide (NO) and carbon monoxide (CO) [PANNEN / BAUER 1998, BENEDIKT / PANNEN 2002].

Endothelines (ET) are peptides, whereby ET-1, which is essential for the liver, is generated in the sinusoidal endothel- and Kuppfer- cells [CLEMENS / ZHANG 1999, BENEDIKT / PANNEN 2002].

Nitric oxide plays an important part in the reduction of the basal tonus of many vessel beds, inclusively of those of the liver. Carbon monoxide is a gaseous molecule, which dilates hepatic vessels. Both monoxides contribute to the relaxation of smooth vessel muscle cells [PANNEN / BAUER 1998, BENEDIKT / PANNEN 2002].

Apart of the factors described above, gastrine and glucagon additionally favour the dilatation of the blood vessels [LAUTT / GREENWAY 1987].

5.4. The Microcirculation of the Liver

The microvascular unit of the liver is the hepatic acinus. Therefore, the acinus, a subunit of the parenchyma, is the functional unit of the liver. It consists of approximately 100.000 parenchyma cells that are arranged in a botryoid form. The end branches of the arterioles and venules unite to the parenchyma cells. The vascular stem extends to the centre of the acinus where hepatic arterial and venous blood is mixed in the *Rappaport zone* [LAUTT / GREENWAY 1987, SIEGENTHALER / BLUM 2006].

In the acinus of the liver, three functional zones can be delimited, which are shell-like arranged around the central axis of the vessel and which are reflecting the different supply of the hepatocytes of an acinus with oxygenated blood, nutrients and hormones.

Zone 1 comprises the hepatocytes that directly enclose a portal field; these hepatocytes are washed around with blood of highest oxygenation and highest content of nutrients and hormones.

Zone 3 comprises the hepatocytes that are furthest apart from blood supply of a portal field within an acinus.

Zone 2 lies in between the two zones described above [LAUTT / GREENWAY 1987, SIEGENTHALER / BLUM 2006, BENNINGHOFF / DRENCKHAHN 2008].

In the liver, the terminal capillary bed between arterial inflow and venous outflow is denominated as “microcirculation”.
In this zone, all of the blood supply, which is vital for nutrition, as well as the gaseous exchange between blood and tissue take place. Maintaining the microcirculation is essential for the appropriate supply of the organ. It is evident from the above-mentioned three metabolic zones that describe the structure of the acinus, that oxygen deficiency takes effect in the pericentral zone 3 at first.

A large part of liver perfusion is controlled by the blood volume provided by the portal vein. The vessels of the splanchnic region with their sphincters directly influence the organ perfusion. Changes of the portal circulation are compensated as far as possible by an increase of the arterial blood flow.

Since the pressure in the arterial system is approximately eight to ten times higher than the pressure in the portal venous system, the blood pressure has to be substantially reduced in an arteriole segment ahead of the liver capillaries that discharge into the sinusoids.

Sphincters in the form of smooth muscle cells are situated in the arterial vessel bed, above all at the ramifications of arteries and interlobar arterioles. The portal venous and the intrahepatic pressure are primarily controlled by the venous sphincters. Since sphincters are manifoldly supplied with nerves, this can be fulfilled via a vegetative control of the blood flow in the lobule, e.g. by vasoconstriction and –dilatation, respectively [REILLY et al. 1981, LAUTT / GREENWAY 1987].

Perfusion is considerably dependent on age [WYNNE et al. 1989, ZOLI et al. 1989 and 1999], body height, and weight [GALLIX et al. 1997], on the general hemodynamic [BENEDIKT / PANNEN 2002], as well as on the intrathoracic and intraabdominal pressure conditions [FINET / WILLIAME 2000].

Additionally, the influence of respiration and gravitational force is described with regard to their effects on the infradiaphragmal venous system [SUGANO et al. 1999, HSIA et al. 2000, FINET / WILLIAME 2000].

5.5. Summary

The hepatic blood flow is regularised on different levels. Alterations on each of these levels can cause changes in the nutrient supply via the sinusoids of the liver. It can be differentiated in intrinsic and extrinsic factors, as was described more in detail in the previous chapters 5.3.1 and 5.3.2.

The **first level** of regulation is the systemic circulation. Besides others, a declination of the heart cardiac output and a reduction of the arterial blood flow by redirection into other vascular beds can reduce hepatic perfusion [BENEDIKT / PANNEN 2002].

The **second level** of regulation concentrates directly on the regional macrocirculation. Via the hepatic artery and portal vein, blood is transported to the liver. The volume of the blood flow in the hepatic artery is determined by its vascular resistance. Since the hepatic artery has only low autoregulatory capacities, the ability to constantly maintain the arterial hepatic blood flow rate during a decline of the arterial perfusion pressure is limited.
The hepatic arterial resistance can serve as a buffer for maintaining the portal flow and in this way it can keep the whole hepatic blood flow constant. This mechanism is also known as “Hepatic Arterial Buffer Response“ [LAUTT / GREENWAY 1987].

The portal vein drains most of the venous blood from the abdominal organs. Therefore, the portal venous flow is also dependent on the arterial inflow resistance to these organs [BENEDIKT / PANNEN 2002].

The **third level** of regulation is the hepatic microcirculation. Changes on the microvascular level have been interpreted as a result of systemic and regional macrodynamic changes for a long time, and additionally as an obstruction of the sinusoidal lumen by dilated perisinusoidal cells, trapped blood cells or thrombotic material. However, there are also descriptions that the sinusoidal flow is actively regularised at the level of microcirculation [BENDIKT / PANNEN 2002].

Apart of these three levels, the orthosympathetic and the parasympathetic nervous system, as well as humoral substances as adrenaline, noradrenaline, angiotensine II, gastrin, and glucagon influence the regulation of the blood flow in the liver [LAUTT 1980, REILLY et al. 1981, MATHIE / BLUMGART 1983, LAUTT / GREENWAY 1987, ROTHE / MAASS – MORENO 1998, PANNEN / BAUER 1998, BENEDIKT / PANNEN 2002, SIEGENTHALER / BLUM 2006].

5.6. Influence of the Diaphragm on the Hemodynamic of the Portal Vein

The dynamic function of the diaphragm is described as the motor of the visceral dynamic in osteopathy. Its importance is also brought into context with a key role in circulation [FINET / WILLAME 2000, LIEM et al. 2000, HELSMOORTEL et al. 2002, BARRAL / MERCIER 2005].

In the hypothesis of FINET / WILLIAME (2000), the contraction of the diaphragm during inspiration causes a compression of the visceral mass and thus also of the veins running inside it. Therefore it plays an important part in venous blood flow. The negative intrathoracic pressure, arising in the thorax by inhalation efficiently acts at the level of the blood in the veins themselves and at the level of the right atrium of the heart [FINET / WILLIAME 2000].

In addition to this, HELSMOORTEL (2002) describes in his model, that the peritoneal content is not moved by the diaphragm during respiration at rest, but however, it is compressed by the lowering diaphragm. Simultaneously, the abdominal body wall reflexively increases its resting tonus, so that the compression finally happens uniformly from all sides.

During the first compression phase, the intraperitoneal pressure increases - this pressure acts from all sides and with equal force. During the second phase, the portal blood runs into the caval system, whereby the volume is decreased. In the course of this, according to the author, the organ position is not changed relatively to their particular supplying neurovascular structures [HELSMOORTEL et al. 2002].

According to the hypothesis of FINET / WILLIAME (2000), in addition to the mechanisms described above, the neck veins collapse during inspiration. The important part that respiration takes concerning the venous circulation, manifests in the subdiaphragmatic venous circulation too. The effect of the factor breathing is added to with the intra-abdominal compression during inspiration [FINET / WILLIAME 2000].

6. Mobility in Visceral Osteopathy

In osteopathy, mobility is defined as a passive motion of the viscera and organs by the extrinsic forces of respiration or of the cardiovascular system. This assumption bases on the following statement:
"In fact, mobility plays a part only as breath-induced viscero-diaphratic movement, so that osteopaths almost exclusively speak of mobility in connection with respiration" [HELSMOORTEL 2002; translation by myself].

Within the theory of HELSMOORTEL (2002), a spatial movement of the viscera is physiologically unnecessary, except for the lungs during breathing. During resting respiration, the centre of mass of the abdominal organs and viscera is not moved and the abdominal content is compressed only.
A spatial movement of mobility during resting respiration is physiologically not normal. That means, it is an indication for compensatory activity, if respiration is needed for the mobilisation of the organs. In this case, mobility stimulates external and internal functions, which support the organ to re-gain its autonomy [HELSMOORTEL et al. 2002].

In this context, FINET / WILLIAME (2000) speak of a "phrenic-mediastinal-vertebral-cranial chain"– a complex of anatomical-physiological loss at the cardiocircular, lymphatic, neurovegetative, neurophysiological, psychosomatic and somatopsychic levels. By this, they additionally stress the importance of a thoracolumbal diaphragm in reference to functionality in their hypothesis.

6.1. Movement of the Diaphragm during the Phases of Mobilisation

During enhanced inspiration, the diaphragm intensifies its motoric activity. With increasing depth of breathing it also changes its direction of motion and its shape [CLUZEL et al. 2000].

HELSMOORTEL (2002) assumes that the movement of the diaphragm as a compensatory activity can be divided into three different phases that are passed

one after another with three different directions of motion. During each phase, the author describes a stimulation activity at another vascular level.

6.1.1. Mobilisation Phase 1

In the model of HELSMOORTEL (2002), the diaphragm lowers deeper than in rest. It rotates around a frontal axis to anterior; the lateral and posterior muscle strands are subject to pre-stress. By this, the organs below the cupula rotate forwardly. That means they move in space, what can be denominated as “spatial mobility”.
In the course of this, the organ is moved relatively to its vessel trunk.
The vessels are subject to mechanical stress. In this process, the arterial structures near the organ are pulled first. If the organs are compressed a little bit more and if they are wrung, the intrahepatic pressure increases.
Hence, the portocaval pressure gradient increases and consequently an enhanced depletion of the portal blood into the caval system is the result. This effect helps to eliminate the venous congestion [HELSMOORTEL et al. 2002].

6.1.2. Mobilisation Phase 2

According to the theory of HELSMOORTEL (2002), the diaphragm lowers further and additionally rotates outwardly in this phase. By this, the mesos encounter pulling forces. In inspiration, this expands the already existing traction to the periphery of the organs. The liver encounters an exterior rotation, the lower part of the stomach is horizontalised, and its upper part rotates exteriorly, too. Therefore, tension is added to the lesser omentum from both sides.
“The mesos encounter pulling forces. They compress the nerves and vessels running inside of them (like a waffle iron the pastry). […] “ [HELSMOORTEL et al. 2002; translation by myself].

6.1.3. Mobilisation Phase 3

In the model of HELSMOORTEL (2002), a rotation of the diaphragm to posterior and a lowering to backwards is achieved in this phase, which is equivalent to the end of an inspiration.
At the same time, the superior parts of the liver and stomach rotate to posterior, too, while the inferior parts and those at the back of the organs lower further. As a consequence, a kind of vertical erection of liver and stomach is the result. The content of the radix and the trunk, as well as the vessel stems of the inferior and

superior mesenteric arteries are mechanically stressed and stimulated by the pulling on the mesos to posterior [HELSMOORTEL et al. 2002].

Echographic investigations by FINET / WILLIAME (2000) showed the following, concerning this topic:

The further apart an organ is from the diaphragm, the less it is influenced by the vertical diaphragmic pressure. Additionally, the transversal shifts in the frontal plane, described by the authors, disappear for the organs most remote to the diaphragm.

However, all niveaus in the frontal plane lower during inspiration. During inspiration, a torsion of the stomach develops by different viscero-dynamics of the parts of the stomach (fundus and corpus move opposedly in frontal and sagittal direction). More exactly, the fundus tilts left, while the corpus tilts right. As a result of the fact, that the fundus lowers three times further than the corpus, the stomach quasi hangs itself up in its longitudinal axis [FINET / WILLIAME 2000].

As mentioned above, FINET / WILLIAME (2000) show a lowering of all niveaus in the frontal plane during inspiration - in particular, the liver lowers in systematic manner top to down, on average by 23.1 mm.
Additionally, their examination showed slight transversal shifts and declinations without any obviously defined pattern.

In the sagittal plane, a systematic shift of the liver from above to below arises from the diaphragm pressure. Although additional anterior - posterior shifts and tilts were observed, no clear pattern concerning these movements could be defined. A description of the dynamics of the liver in the horizontal plane was not possible by means of echography [FINET / WILLIAME 2000].

LIEM (2000) backs on the insights of FINET / WILLIAME (2000) and assumes, that influences on the neighbours connected with the liver (e.g. the part of the stomach that is connected via the lesser omentum) necessarily arise from the visceral dynamic of the liver. The visceral mechanical deformation between the liver and the

connected neighbours is able to influence the lumen of blood vessels [LIEM et al. 2000].

HELSMOORTEL (2002) adds to this assumption and particularly brings up the relationship of liver and stomach in mobility. This relation is determined by the mechanical stress, the parts of the lesser omentum are exposed to [HELSMOORTEL et al. 2002].

6.1.4. The Lesser Omentum

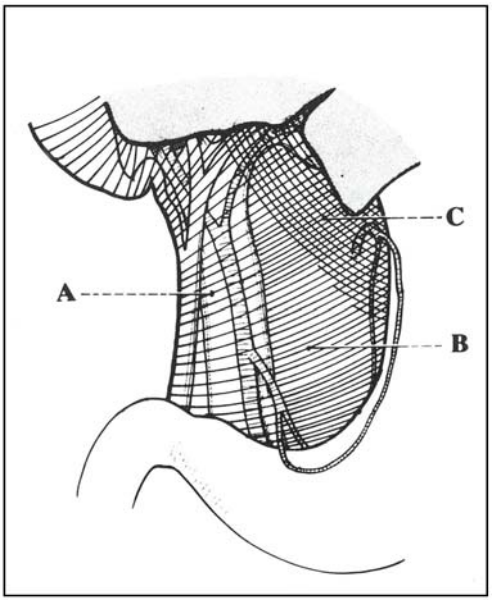
The omenta originate from the anterior and posterior mesogastrium and interconnect the organs of the peritoneum. Since they have been mesenteria, they contain neurovascular structures. The anterior mesogastrium (the later lesser omentum) develops from front to back [ROHEN / LÜTJEN - DRECOLL 2002, BENNINGHOFF / DRENCKHAHN 2008].

According to BOUCHET / CUILLERET (2001), the lesser omentum is divided into the following three parts (cf. Fig. 2) with different qualities, which, according to HELSMOORTEL et al. (2002), represent different possibilities of compensation for the organism:

- **pars vasculosa** with the portal vein, the proper hepatic artery, and the bile duct;

- **pars flaccida**, where the vessel arcade of the small curvature of the stomach is situated;

- **pars condensa** with its tight connective tissue, which continues to the esophagus.



A. pars vasculosa
B. pars flaccida
C. pars condensa

Fig. 2: Parts of the lesser omentum (according to Bouchet / Cuilleret 2001)

6.1.5. Discontinuity between Stomach and Liver

HELSMOORTEL (2002) assumes that a directed elasticity develops during a discontinuity between stomach and liver, which can hold the stomach up in direction of the liver with its force.

According to his model, each single part of the lesser omentum constitutes one possibility of compensation for the organs during each mobilisation phase of the stomach.

While, during mobilisation phase 1 of the stomach, the pars flaccida is physically stressed by the pulling force of the vessel arcade, during mobilisation phase 2, additional blood is pressed to the liver at the level of the pars vasculosa, which additionally supports its already increased tension.

During mobilisation phase 3, the pars condensa is stressed by the pulling force of the stomach, what subsequently stimulates the holding function of the esophagus.

For this reason, a torsion of the lesser omentum develops in each phase by the relative movement between stomach and liver [HELSMOORTEL et al. 2002].

7. Physical and Technical Fundamentals of Ultrasound

This section comprises an introduction into ultrasonic diagnostics and starts with the discovery of the Doppler shift. Then I will explain the calculation of the flow velocity as a directly measurable parameter, because the blood flow in the portal vein and the resulting calculated capacity of the portal vein is dependent on these values. I will also describe the technical basics, as for example, PW-Doppler sonography or duplex sonography, since they were the relevant ultrasound systems for the investigations.

In 1843, the Austrian physicist Johann Christian Doppler (1803 – 1853) noticed: “The wavelength of sound is dependent on the movement of the acoustic source and of the observer relative to each other” [HUCK 2004].

Ultrasound covers sonar waves with a frequency between 20 kHz and 10 GHz. In medicine, ultrasound is transmitted into the tissue by means of a transducer; the acoustic reflexes are received and then transformed into pixels by the sonography device.
As a form of sound, ultrasound is bound to a carrier medium. Since human tissue consists of different carrier mediums for ultrasound, there was an international agreement on the mean value of the sonic speed of 1540 m/s for soft tissue, which is used for the calculations by the ultrasound devices.

In ultrasonic diagnostics of vessels, it has to be considered as a special particularity, that flow rates of blood cells are measured by receiving sonar waves that are backscattered by these corpuscles.
That means, that moving blood cells are acting as moving receivers and then as moving transmitters, and thus the Doppler shift has to be taken into account twice [KUBALE / STIEGLER 2002, HUCK 2004, BÜCHELER et al. 2006, HARTEN 2007].

The flow velocity of the blood is quantified by the measurement of the frequency shift between the transmitted and received signal with the following Doppler equation:

$$\mathbf{v = (\Delta f \cdot c) : (2f_o \cdot \cos \theta)}$$

v = flow velocity of the blood
Δf = frequency change of the transmitted Doppler impulses (Doppler shift)
f_o = transmission frequency
c = sonic speed in human tissue (approx. 1540 m/s)
cos θ = cosine of the angle between ultrasonic beam and the direction of the blood flow (cf. Fig. 3)

It is evident from this equation, that the angle θ is not inconsiderable for the *accuracy of measurement*. With increasing angles from 0° to 90°, the measuring error increases, since the cosine of the angle is in the denominator [HUCK 2004].

Flow velocity is neither under- nor overrated, if the Doppler beam is parallel to the direction of flow. An angle of sixty degrees is considered as the outmost limit for quantitative flow measurements [IGNEE et al. 2002].

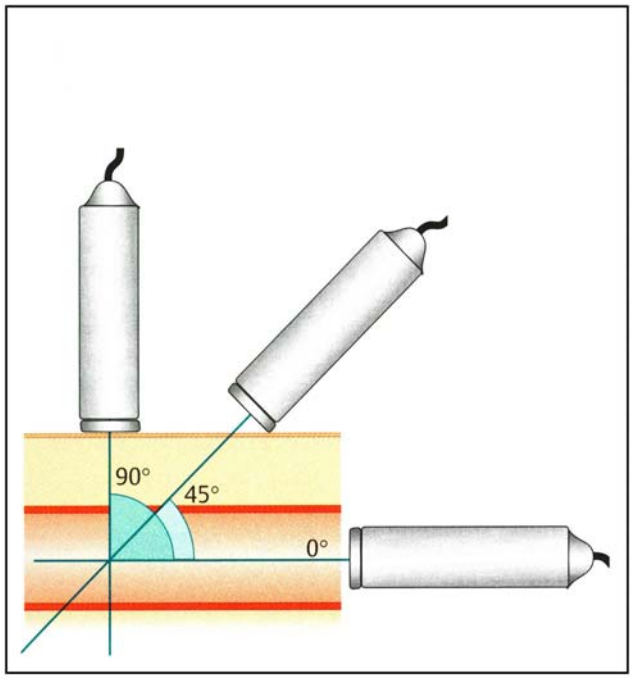


Fig. 3: The irradiation angle is defined as angle between the axis of the vessel and the acoustic beam (according to Huck 2004).

7.1. Pulsed Wave (PW) – Doppler Sonography

Pulsed wave (PW) - Doppler**:** This kind of Doppler sonography works with a single crystal that is alternately in send- and receive mode.
The depth where the information about the blood flow is gained can be calculated by the runtime of the ultrasound, equivalent to the time delay between emission of a wave packet and switching to receiving.

Before the next sonic impulse is transmitted, the arrival of the reflected echo of the first impulse has to be awaited. Otherwise a calculation of depth is not possible. If the receiving region is in high depths, an accordingly longer dead time between send- and receive mode has to be chosen.
In PW - mode, the receiving depth, as well as the spread of the region, where the stream information originates from, can be varied. The advantage of PW - Doppler is its depth selectivity [KUBALE / STIEGLER 2002, HUCK 2004].

7.2. Duplex Sonography

In colour-coded duplex sonography, different methods are combined for displaying a two-dimensional image-sonogram with overlaid stream information.
The stream information is displayed with regard to the direction and velocity or with regard to the energy of the reflected signal. That means that the flow direction in the vessels relative to the transducer is coded with different colours (with red in direction of the transducer, with blue away from it). The flow velocity is brightness coded – the faster, the brighter [BÜCHELER et al. 2006].

Interpretation of these images is, depending on the region, differently difficult.
TAI et al. (1996) describe significant dependencies on examiners.

The most favourable region for the measurement of the portal vein is in the straight course and before the ramification into the two intra-hepatic branches [IGNEE et al. 2002].

In order to minimise the measuring error, the mean value of three trials is computed for each participant and each measurement [LAFORTUNE et al. 1998]. This happens by measuring in the frozen spectral image.

In order to carry out the measurement as accurate as possible, the subjects are instructed how to reach the apnoea position (in resting midinspiration) in advance of the initial measurement and trained five times.

Since two factors, in particular the blood flow velocity and the diameter of the vessel are multiplied for the calculation of the portal blood volume, measurement data in this respect have to be as accurate as possible. Otherwise, the error might increase severely.

Ultrasonic examinations are without risk and can be used without concerns of health hazard within an appropriate, professional administration [DEGUM 2006, UNIVERSITÄTSSPITAL ZÜRICH 2008].

7.3. The Influence of Measuring Error in Ultrasound Examinations

Measuring errors of the flow velocity could arise from non-compliance of the examination conditions, as well as wrong work technique. Some relevant factors are the selection of the region, the angle of irradiation, and the right contact pressure [LAFORTUNE et al. 1998, IGNEE et al. 2002, HUCK 2004].
These factors have been considered in this study, as will be described in the following description of the method.

8. Methodology

With the topic *"Influence of a mobilisation of the lesser omentum on the capacity on the portal vein, measured with echo-Doppler"*, I wanted to find answers to the question, if actual physiological changes can be observed and measured after the administration of a specific visceral technique. Therefore, as well as for the assessment of the efficacy of these technique, I wanted to perform a prospective experimental study. This chapter describes the procedure for the experimental examination.

There is different information about the portal diameter and blood flow velocity in literature [IGNEE et al. 2002]. Hence, there is also disagreement in the capacity of the portal vein. Therefore, for standardisation, selection criteria and the planned course of the study were specified with regard to the present state of sonography and the guidelines specified for these investigations [LAFORTUNE et al. 1998, IGNEE et al. 2002, KUBALE / STIEGLER 2002, HUCK 2004].

Thus, starting with the selection criteria, classified in inclusion- and exclusion criteria, I will describe, why the particular criteria and procedures have been chosen. After predefinition of the selection criteria, I will describe the assignment to the investigational groups - a non-specific-technique group (NST group) and a mobilisation group (MT group) and afterwards I will present the examiners, on the one hand the medical specialists, on the other hand the osteopaths.

8.1. Selection Criteria

Twenty healthy adults were selected on the basis of inclusion- and exclusion criteria from a convenience sample.
In order to reach a high sensitivity of the statistical analysis, it was paid attention to form two as homogeneous groups as possible. Due to reasons specified further down (chapter 8.2), subjects were assigned to the groups match controlled by stratification by the parameters age and BMI (body mass index).

It was the aim of this classification to specify as optimal conditions as possible for the study in the sense of two homogeneous investigational groups in order to minimise possible influences, as age, body height, and weight, which might influence the perfusion in the portal vein.

In order to clarify risks and ethic concerns, the performance of this study was inhospital- approved.

8.1.1. Inclusion Criteria

- male
- healthy
- BMI (body mass index): ≤ 27
- age: 18 – 45 years
- non-smokers
- on an empty stomach (no meal after midnight)
- consumption of alcohol < 25 gr./d.
- staying in lateral decubitus position left is free of complaints
- signing of written agreement
- voluntary participation after being informed
- German speaking and reading capability

8.1.2. Exclusion Criteria

- drug-use
- inflammatory diseases
- former abdominalsurgical interventions
- BMI: > 27 (a to high BMI can make the visualisation of the portal vein difficult)
- age: ≥ 46 years
- smokers
- alcohol intake: >24 gr./d.
- known heart or liver pathologies

8.1.3. Explanation of the Inclusion- and Exclusion Criteria

Two groups, as homogeneous as possible with regard to age, sex, body weight and height should be formed for this study. In order to comply with this, beforehand, that means during selection of the subjects, additional factors had to be taken into account.

GALLIX et al. (1997) and IGNEE et al. (2002) mention in their studies, that individual physical conditions as height and weight are factors influencing the blood flow in the portal vein.

SABBA et al. (1990) describe a gender specific influence on the portal blood flow. The fact that different measurement results had to be expected for men and women is taken into account as a criterion in the actual study, where only men were included.

ZOLI et al. (1999) mention age dependent variations in the blood flow. According to them, there is a decrease of flow velocity as well of blood volume from the 45th year of age on. Again, this is taken into account in the actual study.

Smoking and consumption of alcohol change parameters as the portal blood volume, the portal blood flow velocity, the blood pressure, the heart rate, as well as the arterial wall tension [YOSHIHARA et al. 1985, MANCIA et al. 1997, AROSIO et al. 2006].
Since healthy men take part in this study, the “limit of harmlessness” of the guidelines by AKIS (2008) was used as cut-off criterion for alcohol intake.

The participants of the study must not have obvious heart- or liver pathologies, nor inflammatory diseases or abdominal surgical interventions.

This was scrutinised during anamnesis in advance of the experiments. Furthermore, other diseases (e.g. any heart- and liver pathologies), which could have negative effects on the measurement results were precluded in a short conversation with the particular general practitioners of the test persons [HOSOKI et al. 1990, BOLONDI et al. 1992, LEEN et al. 1993, NOLTE et al. 1994, KOK et al. 1999, KRÄHENBÜHL

et al. 1999, WIEST et al. 2000, YIN et al. 2001, KAYACETIN et al. 2004, SUGIMOTO et al. 2005, BOLOGNESI et al. 2007].

NOLTE et al. (1994) describe, that intake of beta-blockers, nitrates, diuretics, and serotonin receptor inhibitors lead to changes in the portal inflow and outflow.
KAMKE (1994) brings up substantial changes of the flow rate in blood vessels already after slight changes in their profiles by the application of medicinal remedies. Thus, persons taking drugs were generally excluded from the study.

Various authors, as e.g. SABBA et al. (1991), TEICHGRÄBER et al. (1997) or IGNEE et al. (2002) mention, that the moment of the last food intake is influencing the portal hemodynamic during the measurement. Therefore, all test persons had to be on an empty stomach for at least eight hours.

German speaking- and reading capability was another prerequisite for participation, since all test persons were instructed in the course of the studies in written and oral way. Additionally, they had to sign a statement of agreement and were asked about anamnestic data.

8.2. Characterisation of the Investigational Groups

Initially, I intended to assign group members (quasi-) randomised from a convenience sample after stratification by age and BMI. However, after assignment, some test persons dropped out because of occupational, familiar or other reasons.
Therefore, the remaining subjects were assigned match controlled to the particular groups. Attention was paid that as many characteristics of both groups as possible fitted as well as possible or were correspondent. At this point, I want to refer to chapter 13, the discussion.
In order to equally consider the two parameters age and BMI, these two factors were multiplied for each test person and the products were sorted in ascending order. Then, the test persons were assigned to the two groups by allocation of the single subjects with the highest and with the lowest value to one group. This procedure was repeated with the residual subjects for the allocation to the other group, until both groups were complete.

In this way, two homogeneous groups with regard to age and BMI were formed, which were called “mobilisation group“ (MT) and “non-specific technique group” (NST), depending on the technique administered to the test persons.

8.2.1. The Mobilisation Group (MT)

The mobilisation group (MT group) comprised ten test persons. In this group, the lesser omentum was mobilised with a technique according to the description by HELSMOORTEL et al. (2002). However, HELSMOORTEL did not specificate the hip- and knee position. For standardisation, joint angles described below were appointed for the MT group as well as for the NST group. Since the ultrasonic examination was performed in apnoea (in resting midinspiration position), this was trained with each single test person five times in advance of the study.

The position of the test person: The test person layed relaxed in decubitus position left, the legs in a double flexion of knee (90°) and hip (45°). Attention was paid, that no constriction by dress or belts was possible.

The position of the osteopath: The osteopath stood behind the test person and supported him with his body, so that a good contact could be established.

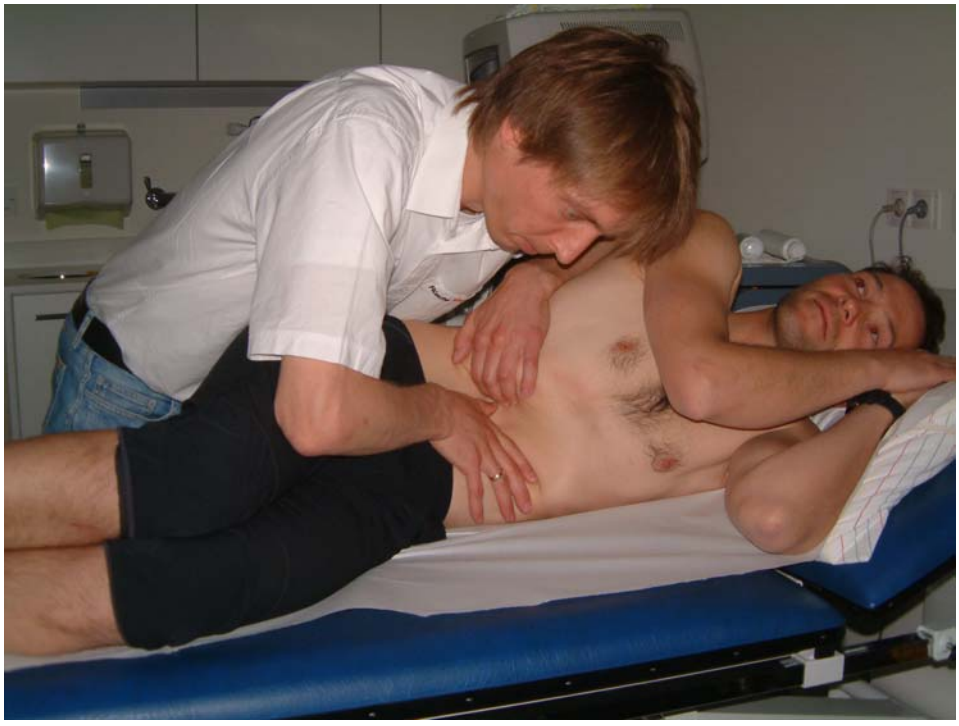


Fig. 4: Mobilisation Technique (MT group).

Hand posture (cf. Fig.4): The right hand of the osteopath got in touch with the stomach of the test person. The thumb was placed at the level of the pylorus, which is situated two centimetres to the right and two centimetres to cranial from the navel [Helsmoortel et al. 2002]. The left hand encompassed the costal arch with the fingers from top to posterior and got in contact with the liver. A slightly accentuated inspiration facilitated the contacting.

Performance: During inspiration, the osteopath fixed the liver and followed the movement of the stomach. During exspiration, the stomach was fixed and the liver was released. In the course of this, contact was not allowed to be lost. This pain free technique was administered for three minutes. The mobilisation was standardised by exactly following the above description and by staying on time schedule.

8.2.2. The Non-Specific Technique Group (NST)

The non-specific technique group (NST) consisted of ten test persons, too. During intervention, they were exposed to a constant pressure in the abdominal region. Since the ultrasonic examination happened in apnoea (in resting midinspiration position), this manoeuvre was trained with each single test person five times in advance of the study, too.

The position of the test person: The test person layed - identical to the position of the test persons in the MT group - in decubitus position left, with the legs in a double flexion of knee and hip. An impact by too narrow clothes or belts was excluded.

The position of the osteopath: The osteopath stood behind the test person and supported him with his body, so that a good contact could be established.

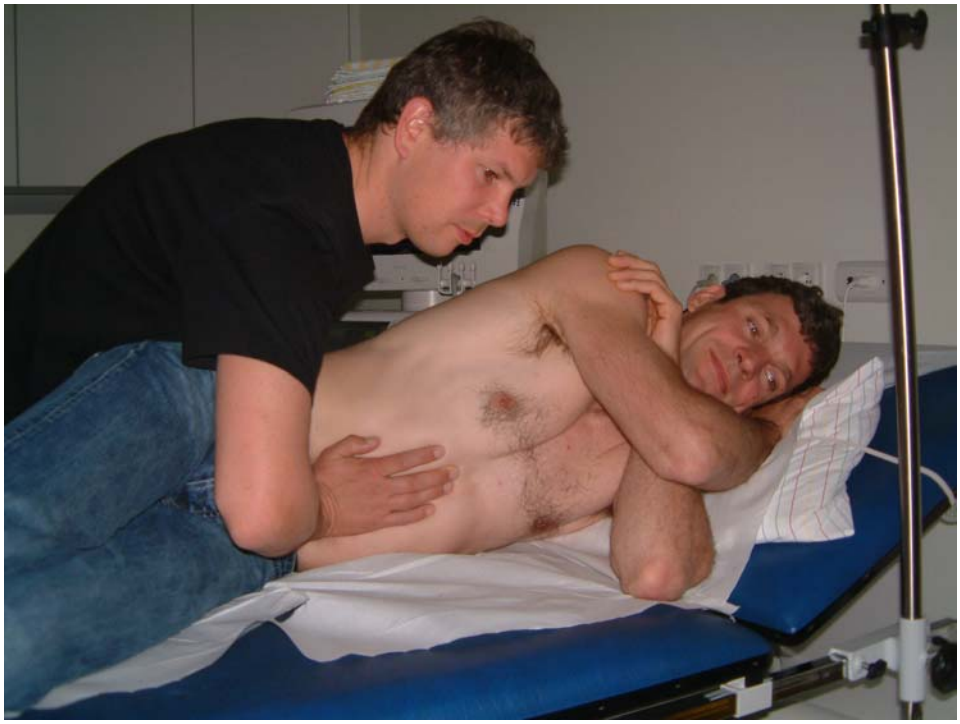


Fig. 5: Non-specific technique (NST group).

Hand posture (cf. Fig. 5): The right hand of the osteopath got in contact with the navel, which was situated between thenar and hypothenar. The finger tips pointed in the direction of the processus xyphoideus. A slightly accentuated inspiration facilitated the contacting.

Performance: During exspiration pressure was applied in direction of the spine. This pressure was sustained until a perceptible abdominal pulsation was achieved. In this moment, the osteopath reduced his pressure to a minimum of noticability of the pulsation. In the course of this, the axial pressure to posterior had to be held, that means, it was not allowed to follow any facilitation.
In that moment, time measurement was started and this pain free technique was administered for three minutes, either.

This technique was standardised, too, by exactly following the descriptions above and by staying on time schedule.

8.3. The Examiners

For an as objective course of this study as possible, the phases of intervention and measurement were performed by two osteopaths and medical specialists, each.
I myself was responsible for documentation only, and I did not perform any intervention in the test persons.

8.3.1. The Osteopaths

In order to grant an as high standard as possible for the osteopathic part of the experiment, two alumni of the Vienna School of Osteopathy (WSO), both Masters of Science in Osteopathy, were selected for the administration of the intervention techniques. The osteopaths disposed of almost the same practical experience (5 years) and both were right-handers.

In a kind of blinding, an objective approach of the osteopaths to the particular technique should be granted. The osteopaths did neither know the problem of the study, nor did they know which intervention the other osteopath performed.

In the beginning, the osteopaths received written instructions and a strict description of the technique. They were informed how to place their hands and how to apply the respective technique.
Additionally, both osteopaths took three supervisions in the particular techniques for sensibilisation and precision of the application. Each osteopath trained the particular technique on five persons supervised by me. The five persons did not take part in the study.
This should ensure an as accurate, as reproducible, and as sensitised course of the study as possible.

8.3.2. The Medical Specialists

Two medical specialists for internal medicine performed the measurements. They were employed at the same hospital, and disposed of seventeen and eight years of experience in abdominal echography, respectively. In literature, three years of experience are specified as qualification [SABBA et al. 1990, SEITZ 2006].

9. The Equipment

During the examination, always the same instruments were used. Therefore, all data in both investigational groups were gained by means of the same devices. The ultrasound device as well as the blood pressure- and the heart rate meter were available on the spot.

9.1. The Blood Pressure- and Heart Rate Meter

Since blood pressure as well as heart rate might influence the examination, both parameters were measured at pre-defined moments. Measurements were performed with the hospital owned instrument "*Mindray", type "VS 800"* (cf. Fig.6).

Fig. 6: Mindray, VS 800 (Original image by Mindray)

9.2. The Ultrasound Unit

The measurement of blood flow velocity was done with a *"Toshiba"* ultrasound device, *type "Xario"* (cf. Fig.7).

This measurement was performed with a 3.5 MHz transducer and colour-coded duplex sonography, the measurement of the blood flow velocity with PW - Doppler. All measurements in this study were done by means of the same instrument and in the same examination room.

Fig. 7: Toshiba Xario ultrasound device (Original image by Toshiba Medical Systems)

9.3. The Measurements in the Portal Vein

The liver receives the nutritious venous blood from the digestive tract via the portal vein. In the course of this, the lienal vein and the superior mesenteric vein unite behind the pancreas to form the main stem of the portal vein. The main stem crosses the upper part of the duodenum and reaches the hepatic porta in the hepatoduodenal ligament. The blood flow velocity is measured just before the intra-hepatic branching of the portal vein into the left and right branch, directly in the hepatic porta. There the averaged venous velocity (TAV $_{mean}$) is determined for the portal vein, because it is used for the calculation of the blood flow volume [IGNEE et al. 2002].

The measurements were performed on an empty stomach, because portal perfusion depends on the moment of the last food intake [WEBSTER et al. 1975, DAUZAT et al. 1994, IGNEE et al. 2002, GAIANI et al. 2005, CHUO et al. 2005].

The angle of irradiation should be lower than 60° [LAFORTUNE et al. 1998, IGNEE et al. 2002, KUBALE / STIEGLER 2002].

The sonography was performed in apnoea (in resting midinspiration position), since during enhanced inspiration and exspiration, respectively, differences in the portal blood flow velocity have to be expected [SUGANO et al. 1999].

The blood flow volume BF is calculated by means of the following formula:

$$\mathbf{BF = TAVmean \cdot A \cdot 60}$$

Legend:

BF	=	blood flow volume in ml/min.
TAVmean	=	mean flow velocity in cm/s
A	=	profile area in cm² ($A = \pi \cdot D^2/4$)
D	=	diameter in cm

(The factor 60 is used for the specification of the blood flow volume in ml/min, only.)

With the aforesaid parameters, finally the capacity (in ml/min) can be calculated by means of the following formula:

$$\mathbf{BF = D^2 \cdot TAVmean \cdot 15 \cdot \pi}$$

10. The Experiment

After informing about the course of the study, the signing of the informed consent, and the appointment of the date of examination, the test persons were directly received in the district hospital of Dornbirn. At this point, I defer to the fact, that all data were surveyed ahead of the day of examination, in order to avoid additional stress causing influences.

According to the guideline for vascular diagnostics of IGNEE et al. (2002), it was tried to create a calm, comfortable atmosphere in the waiting room. As recommended by REHRER et al. (2001), a period of rest before the measurements should counteract changes in portal blood flow caused by physical activity.

10.1. Course of the Examination

The test persons (on an empty stomach) laid down on the examination table in decubitus position left, identical with the position that was necessary for the application of the particular intervention technique (non-specific-technique and mobilisation technique).
The investigating medical specialists as well as the test persons were blinded with regard to the applied technique.
Now the first measurement (x1) of the portal vein was performed and the measurement data were directly listed.

Then, the test persons sat down in the waiting room again. Half an hour later, a second measurement (x2) was performed in decubitus position left.
This measurement was necessary in order to recognise the dispersion between the measurements (x1) and (x2), that means to assess possible physiological fluctuations. Without consideration of these natural processes, the effects of the application of the mobilisation techniques possibly could be overrated during measurement (y). By the baseline measurements (x1) and (x2), the causal relationship between the applied technique and the measured values should be ensured.

After the measurements (x1) and (x2), the specific and the non-specific technique were applied to the test persons by an osteopath. Immediately after the application of the techniques, the third measurement (y) was done by means of Doppler – ultrasound. Both, the applied techniques as well as the ultrasonic examinations were done in the standardised lateral position, described further above (decubitus position left). The medical specialist left the room, before the osteopath applied the respective technique, in order to avoid the recognition, what group the test person belonged to (NST group or MT group). A possible influencing should be avoided by this procedure.

The fourth measurement (z) was performed one hour later.
In order to detect potential alterations of the blood flow by a raised heart rate (e.g. by nervousness or by physical activity of the test persons in advance of the beginning of the test, heart rate and blood pressure were additionally measured in sitting position during the measurements (x1, x2, and z) with the hospital owned instrument Mindray, Type VS-800.
Since a positional change of the participants from decubitus position left to sitting position for the heart rate- and blood pressure measurements (y), would influence the portal blood flow, neither the heart rate nor the blood pressure was measured during measurement (y).
The ultrasonic measurements (x1, x2, y, z) of the diameter of the portal vein (in cm), as well as of the flow velocity in the portal vein (in cm/s) were performed three times. Subsequently, the mean value of these results was calculated. The capacity (BF) of the portal vein was evaluated with the mean values of these variables.

11. Statistical Evaluation

The basic question was whether influences of NST and MT intervention on the capacity of the portal vein would be different, and which physiological processes would be affected by intervention.

The null hypotheses, statistical evaluation is basing on, are:

1. The base lines of the variables blood pressure, pulse, capacity and diameter of the portal vein, as well as blood flow velocity in the portal vein are equal in the two groups.

2. The capacities and diameters of the portal vein as well as the velocity of blood flow in the portal vein during the measurements (y) and (z) after NST and MT intervention do not differ from base line.

3. There are no differences in the effects of NST and MT intervention on the capacity and the diameter of the portal vein and on the portal blood flow velocity.

Most of the evaluation was done by means of repeated measures analysis of variance (ANOVA). The dependent variables “blood pressure”, “pulse”, “capacity”, “diameter“, and “velocity” were tested tested with regard to the within-subject factor “test” and the between-groups factor “group”.
The effect of the “test”-factor describes differences within all individual measurements (without consideration of group-differences), the effect of the “group” factor describes differences between the two groups (without consideration of temporal influences), and the “test x group” interaction describes group specific differences in the changes between the different measurements.

One-way ANOVA was used for the assessment, whether baselines of age, body weight, body height, and BMI were comparable between the two groups.
In the case of significant ANOVA results, further evaluation was done by means of paired samples t-tests and independent samples t-tests.

For applicaton of these tests, tests for normal distribution (Kolmogorov Smirnov test) and homogeneity of variance (Levine's test) had to be performed in advance of these calculations.

Software used was R 2.7.1 for the repeated measures ANOVA and SPSS® 14.0. for graphics and descriptive statistics. Tests were performed two-tailed and p-values <0.05 were considered statistically significant.

12. Results

12.1. Characteristics of the Test Persons

12.1.1. General Data

The twenty test persons of the two groups had been matched by age and body mass index (BMI) in advance of the study.
Descriptive data of their characteristics (mean values and standard deviations) can be observed in Table 1.
In addition to these statistics, results of the ANOVA (p-values) performed with the dependent variables “age”, “body weight”, “height”, as well as “BMI” with the independent factor “group” are displayed in this table.

Group	NST			MT			p (Anova)
Variable	N	Mean	SD	N	Mean	SD	
body weight	10	76.6	8.7	10	75.1	5.9	0.66
body height	10	1.82	0.06	10	1.80	0.05	0.40
Age	10	34.3	4.7	10	34.3	5.8	1.00
BMI	10	23.2	1.8	10	23.3	1.6	0.86

Table 1: Characteristics of the test persons of both groups and p-values of the ANOVA performed with the factor “group”.

Due to the matching criteria, expectably, groups corresponded in age and BMI, just as in body weight and height.

Influences of the time-factor “test” and the factor “group” on the initial values (baseline) of the dependent variables “blood pressure” and “pulse”, which were measured only during the first two tests (x1 and x2) in advance of the intervention, were evaluated by repeated measures ANOVA. The results are displayed in the following chapters.

12.1.2. Systolic Blood Pressure

Mean values and standard deviations (SD) of the systolic blood pressure during both tests in advance of the intervention (tests x1 and x2) are summarised for both groups in Table 2. Results of the ANOVA are summarised in Table 3.

Group	**NST**			**MT**			**Total**		
Variable: systolic blood pressure [mm Hg]	N	Mean	SD	N	Mean	SD	N	Mean	SD
Test x1	10	124.9	10.9	10	120.2	15.4	20	122.6	13.2
Test x2	10	117.4	7.7	10	117.4	10.5	20	117.4	8.9

Table 2: Mean values and standard deviations (SD) of the systolic blood pressure during test x1 and x2 in both groups.

Variable: systolic blood pressure		
Factor	F	P
Group	0.244	0.63
Test	7.211	**0.02**
Group * Test	1.502	0.24

Table 3: Effects of the factors "group", "test" and of the "group x test" interaction on the systolic blood pressure.

According to the ANOVA results, significant differences in the systolic blood pressure can be observed between the two tests (p=0.02). Nevertheless, there is no significant influence of the factor "group" and of the "group x test" interaction. That means group differences were less distinct than test-to-test variability of the data.

Mean values and 95%-confidence intervals are displayed in Fig. 8 (The y-axis covers the actual range of data).

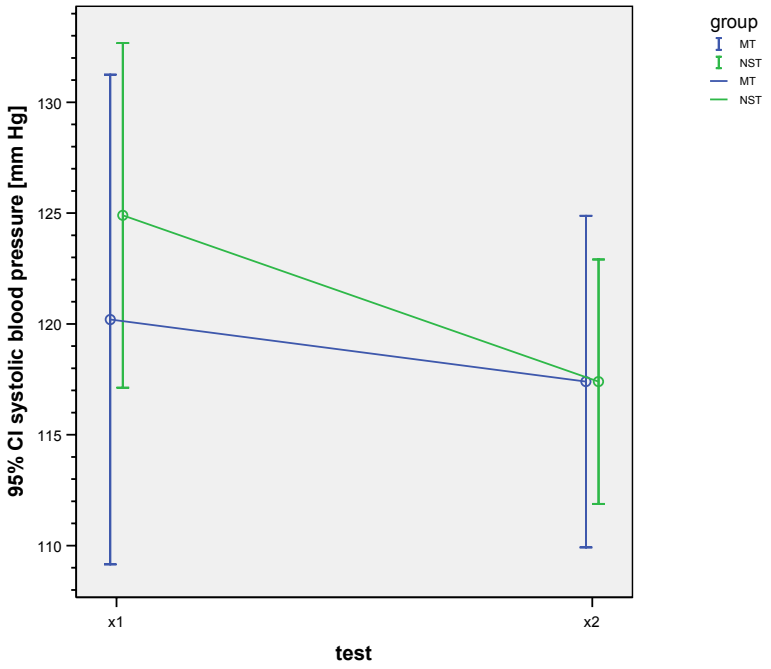


Fig. 8: Mean values and 95%-confidence intervals of the systolic blood pressure during the individual measurements and broken down by groups.

Mean group differences during test x1 were higher than during test x2, but in accord with the results of the ANOVA, group differences are not significant in either measurement (independent samples t-test: test x1: p=0.44, test x2: p=1.00).
The decrease of the systolic blood pressure was significant in the NST group (paired samples t-test: d_{x2-x1}= -7.5 mm Hg, p= 0.02), whereas in the MT group it was less distinct (paired samples t-test: d_{x2-x1}= -2.8 mm Hg, p= 0.32).

12.1.3. Diastolic Blood Pressure

Mean values and standard deviations (SD) of the diastolic blood pressure during both tests in advance of intervention (tests x1 and x2) are summarised for both groups in Table 4. Results of the ANOVA are summarised in Table 5.

Group	**NST**			**MT**			**Total**		
Variable: diastolic blood pressure [mm Hg]	N	Mean	SD	N	Mean	SD	N	Mean	SD
Test x1	10	79.8	9.2	10	77.5	8.9	20	78.7	8.9
Test x2	10	74.1	8.4	10	78.1	5.0	20	76.1	7.0

Table 4: Mean values and standard deviations (SD) of the diastolic blood pressure during test x1 and x2 in both groups.

Variable: diastolic blood pressure		
Factor	F	P
Group	0.068	0.80
Test	2.792	0.11
Group * Test	4.260	*0.05*

Table 5: Effects of the factors "group", "test" and of the "group x test" interaction on the diastolic blood pressure.

A distinct influence of the "group x test" interaction on the diastolic blood pressure can be observed in this case. That means, that diastolic blood pressure changed over time and it changed in different ways in the single groups.
Paired samples t-tests result in a slight increase of d_{x2-x1}= 0.6 mm Hg (p= 0.77) in the MT group and in a significant decrease of d_{x2-x1}=-5.7 mm Hg (p= 0.03) in the NST group.

As can be observed in Table 4, one group (MT group) increased in mean diastolic blood pressure over time and the other group (NST group) decreased significantly. Mean values of the two measurements of the two groups do not differ to a high extent (p=0.80).

Either these significant differences occurred due to unknown exterior influences or due to chance, possibly caused by the small sample (natural variability probably would not result in significant differences).

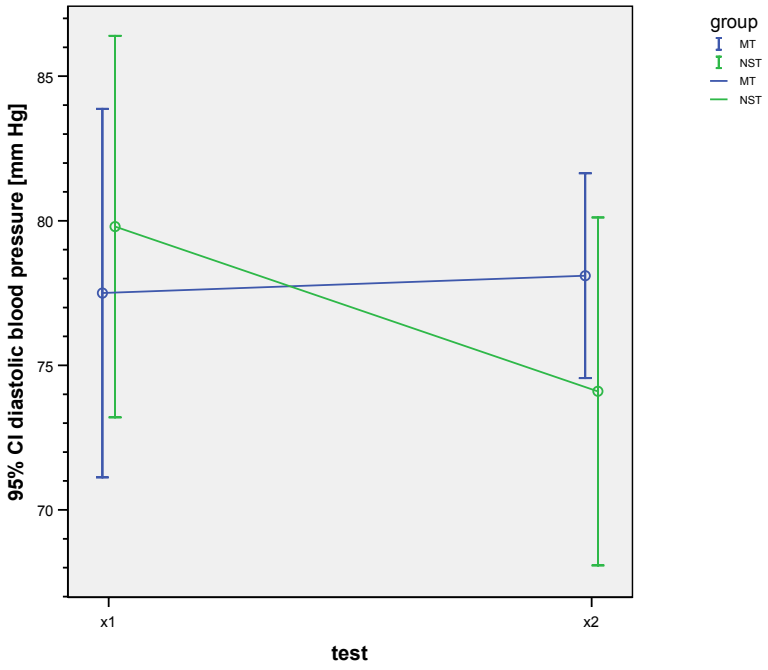


Fig. 9: Mean values and 95%-confidence intervals of the diastolic blood pressure during the individual measurements and broken down by groups.

Taking into consideration, that also systolic blood pressure was significantly reduced during the second measurement (x2) in the NST group only, it is likely, that test persons in the NST group could relax easier than subjects of the MT group.

12.1.4. Pulse Rate

Mean values and standard deviations (SD) of the pulse rate during both tests in advance of intervention (tests x1 and x2) are summarised for both groups in Table 6. Results of the ANOVA are summarised in Table 7.

Group	**NST**			**MT**			**Total**		
Variable: pulse/min	N	Mean	SD	N	Mean	SD	N	Mean	SD
Test x1	10	60.6	12.3	10	58.7	12.2	20	59.7	12.0
Test x2	10	57.7	10.1	10	57.3	13.2	20	57.5	11.4

Table 6: Mean values and standard deviations (SD) of the pulse rate during test x1 and x2 in both groups.

Variable: pulse/min		
Factor	F	P
Group	0.047	0.83
Test	5.75	**0.03**
Group * Test	0.700	0.41

Table 7: Effects of the factors "group", "test" and of the "group x test" interaction on the pulse rate.

The mean pulse rates of the test persons in the two groups are comparable (there are neither effects of the factor "group" nor of the "group x test" interaction). Nevertheless, a significant decrease of the pulse rate between the two tests can be observed in p=0.03 for the effect of the factor test, that means with the collapsed results of all test persons of both groups.

Mean values and 95%-confidence intervals are displayed in Fig. 10 (The y-axis covers the actual range of data).

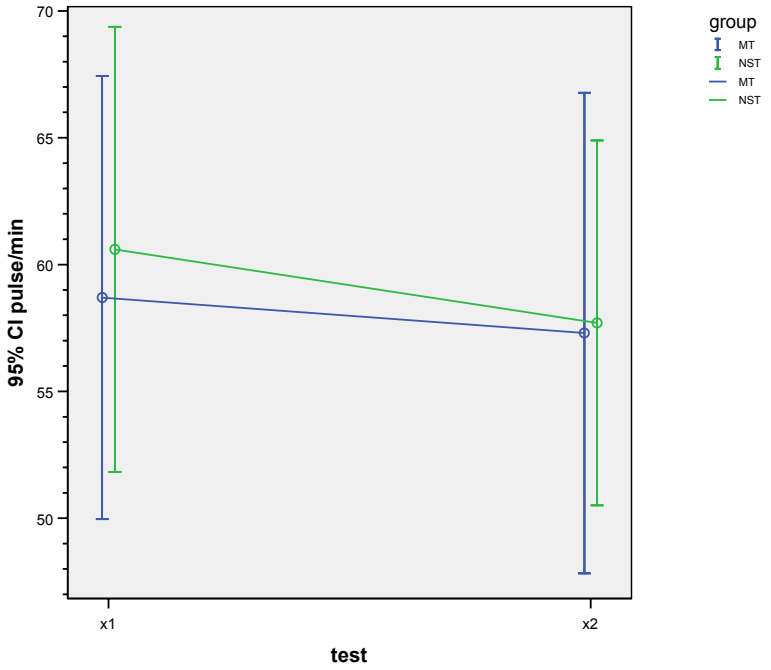


Fig. 10: Mean values and 95%-confidence intervals of the pulse rate during the individual measurements and broken down by groups.

Mean group differences during test x1 were higher than during test x2, but during neither measurement group differences were significant (independent samples t-test: test x1: p=0.73, test x2: p=0.94).

The significant effect of the factor test was mainly caused by the subjects of the NST group, where the decrease of the pulse rate was more distinct over time (paired samples t-test: d= -2.9 beats/min, p= 0.05) than in the MT group (paired samples t-test: d= -1.4 beats/min, p= 0.29). With collapsed groups, the results of the t-test are: d= -2.1 beats and p=0.03, identical with the value in Table 7.

12.2. Measurement Conditions (Irradiation Angle)

For measurement of the diameter and velocity of blood flow in the portal vein, theoretically, measurement with an irradiation angle of 0° is favoured. Typically, irradiation angles are between 55 and 60° and results are corrected by the device software.

Mean values and standard deviations (SD) of the irradiation angles during the individual tests are summarised for both groups in Table 8.

Group	**NST**			**MT**			**Total**		
Variable: mean angle [°]	N	Mean	SD	N	Mean	SD	N	Mean	SD
Test x1	10	58.8	2.1	10	55.4	10.6	20	57.1	7.6
Test x2	10	58.5	1.8	10	54.9	10.7	20	56.7	7.7
Test y	10	59.3	1.3	10	55.6	7.8	20	57.4	5.8
Test z	10	58.9	1.2	10	55.9	10.9	20	57.4	7.7

Table 8: Mean values and standard deviations (SD) of the mean angle during the individual tests in both groups.

In Table 9 results of repeated measures analysis are displayed.

Variable: angle		
Factor	F	P
Group	1.497	0.24
Test	1.707	0.21
Group * Test	0.027	0.87

Table 9: Effects of the factors "group", "test" and of the "group x test" interaction on the measurement angles.

Generally, lower values can be observed in the MT group during all measurements, but according to the results of repeated measures ANOVA, angles did not differ significantly between the groups and individual tests.

12.3. Main Outcome Variables

12.3.1. Capacity of the Portal Vein (mean flow)

Mean values and standard deviations (SD) of the unnormalised capacity of the portal vein (mean flow) during all tests are summarised for both groups in Table 10. Results of the ANOVA are summarised in Table 11.

Group	**NST**			**MT**			**Total**		
Variable: mean flow [ml/min]	N	Mean	SD	N	Mean	SD	N	Mean	SD
Test x1	10	756.7	361.7	10	766.7	258.6	20	761.7	306.1
Test x2	10	782.1	333.9	10	744.7	318.4	20	763.4	318.1
Test y	10	886.2	501.9	10	843.6	288.6	20	864.9	399.1
Test z	10	730.1	211.7	10	751.6	250.2	20	740.8	225.8

Table 10: Mean values and standard deviations (SD) of the capacity of the portal vein during the individual tests in both groups.

Variable: mean flow		
Factor	F	P
Group	0.001	0.92
Test	1.668	0.18
Group * Test	0.143	0.93

Table 11: Effects of the factors "group", "test" and of the "group x test" interaction on the capacity of the portal vein.

The most distinct effect on the capacity of the portal vein can be described by the factor "test". The results of paired samples t-tests performed with the results of two consecutive tests, each, are displayed in Table 12.

Mean values and 95%-confidence intervals are displayed in Fig. 11 (The y-axis covers the actual range of data).

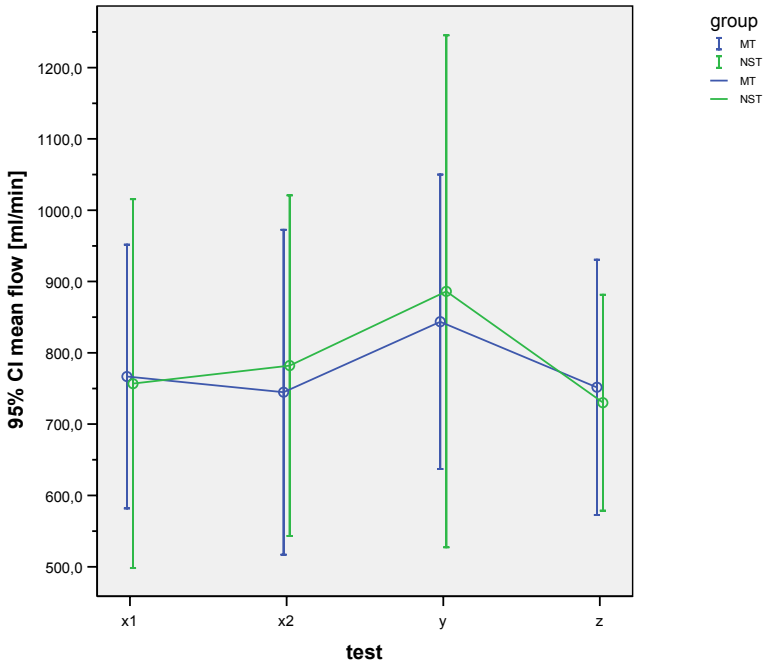


Fig. 11: Mean values and 95%-confidence intervals of the capacity of the portal vein during the individual measurements and broken down by groups.

As can be observed in this figure, the mean capacity of the portal vein was almost equal during the first baseline measurement in advance of the intervention (test x1). During measurement (y) that was performed directly after intervention, a higher capacity could be observed in both groups, and during measurement (z), the mean capacities were comparable to the baselines again. Nevertheless, differences in the capacity of the portal vein between the two groups as well as between the pre- and post-tests are not significant, as can be observed in the ANOVA results, too (cf. Table 11).

Tests	Group	Mean difference	SD (difference)	P
x2-x1	MT	22.1	204.2	0.74
	NST	-25.3	186.6	0.68
	Total	-1.6	191.9	0.97
y-x2	MT	-98.9	170.4	*0.10*
	NST	-104.1	316.1	0.32
	Total	-101.5	247.2	*0.08*
z-y	MT	92.0	316.6	0.38
	NST	156.1	362.7	0.21
	Total	124.1	333.0	0.11

Table 12: Results of the paired samples t-tests with the capacities of two consecutive tests.

Test- to-test data variability of the capacity of the portal vein was comparable in the two groups during the two baseline measurements. After intervention, the increase of the capacity was more distinct in the MT group (p=0.10) than in the NST group (p= 0.32), but differences were not significant.

12.3.2. Capacity of the Portal Vein (mean flow/kg body weight)

Mean values and standard deviations (SD) of the capacity of the portal vein (mean flow/kg body weight) of all tests are summarised for both groups in Table 13. Results of the ANOVA are summarised in Table 14.

Group	**NST**			**MT**			**Total**		
Variable: mean flow/kg body weight [ml/min]	N	Mean	SD	N	Mean	SD	N	Mean	SD
Test x1	10	10.0	4.8	10	10.2	3.6	20	10.1	4.1
Test x2	10	10.3	4.3	10	10.0	4.4	20	10.1	4.2
Test y	10	11.5	6.2	10	11.3	4.0	20	11.4	5.1
Test z	10	9.6	2.6	10	10.0	3.4	20	9.8	2.9

Table 13: Mean values and standard deviations (SD) of the capacity of the portal vein during the individual tests in both groups.

Mean values and 95%-confidence intervals are displayed in Fig. 12 (The y-axis covers the actual range of data).

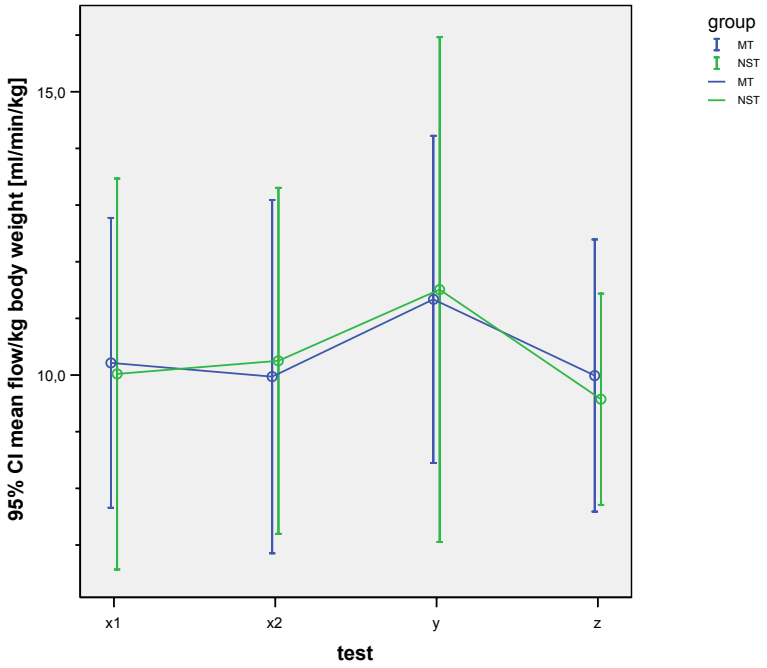


Fig. 12: Mean values and 95%-confidence intervals of the capacity of the portal vein during the individual measurements and broken down by groups.

As can be observed in this figure, the mean capacity of the portal vein was almost equal during the two baseline measurements in advance of the intervention (test x1 and x2). During measurement (y), which was performed directly after intervention, a higher capacity could be observed in both groups, and during measurement (z), the mean capacities were comparable to the baselines again. Nevertheless, differences in the capacity of the portal vein between the two groups as well as between the pre- and post-tests are not significant, as can be observed in the ANOVA results, too (cf. Table 14).

Variable: mean flow/kg body weight		
Factor	F	P
Group	0.0003	0.99
Test	1.674	0.18
Group * Test	0.079	0.97

Table 14: Effects of the factors "group", "test" and of the "group x test" interaction on the capacity of the portal vein.

The most distinct effect on the capacity of the portal vein can be described by the factor "test". According to the results of the ANOVA, no significant effects on the capacity of the portal vein could be expected, but since capacity was the main

outcome variable, a further look was taken on this factor. For this, the results of paired samples t-tests (performed with the results of two consecutive tests, each) are displayed in Table 15.

Tests	Group	Mean	SD	P
x2-x1	MT	-0.24	2.76	0.79
	NST	0.23	2.39	0.76
	Total	-0.00	2.52	0.99
y-x2	MT	1.36	2.34	*0.10*
	NST	1.26	3.89	0.33
	Total	1.31	3.13	*0.08*
z-y	MT	-1.35	4.00	0.32
	NST	-1.93	4.79	0.23
	Total	-1.64	4.30	0.10

Table 15: Results of the paired samples t-tests with the capacities of two consecutive tests.

Test- to-test data variability of the capacity of the portal vein was comparable in the two groups during the two baseline measurements. After intervention, the increase of the capacity was more distinct in the MT group (p=0.10) than in the NST group (p= 0.33), but differences are not significant.

Generally, the results of the variable “mean flow/kg body weight” do not differ distinctly from the results of the variable “mean flow” described in the chapter before.

12.4. Physiological Alterations

The capacity of the portal vein is calculated by the diameter and velocity of the blood flow in the portal vein. Therefore, it might be possible, that each of the two techniques affect these two variables in a different way. For that reason, influences of the interventions on these variables were assessed in the same way as the variables discussed before.

12.4.1. Diameter of the Portal Vein

Mean values and standard deviations (SD) of the mean diameter of the portal vein during all tests are summarised for both groups in Table 16.

Group	**NST**			**MT**			**Total**		
Variable: mean diameter [cm]	N	Mean	SD	N	Mean	SD	N	Mean	SD
Test x1	10	1.015	0.194	10	1.072	0.178	20	1.044	0.184
Test x2	10	1.081	0.235	10	1.005	0.181	20	1.043	0.207
Test y	10	1.145	0.250	10	1.083	0.220	20	1.114	0.231
Test z	10	1.042	0.143	10	1.029	0.181	20	1.036	0.159

Table 16: Mean values and standard deviations (SD) of the mean diameter of the portal vein during the individual tests in both groups.

Mean values and 95%-confidence intervals are displayed in Fig. 13 (The y-axis covers the actual range of data).

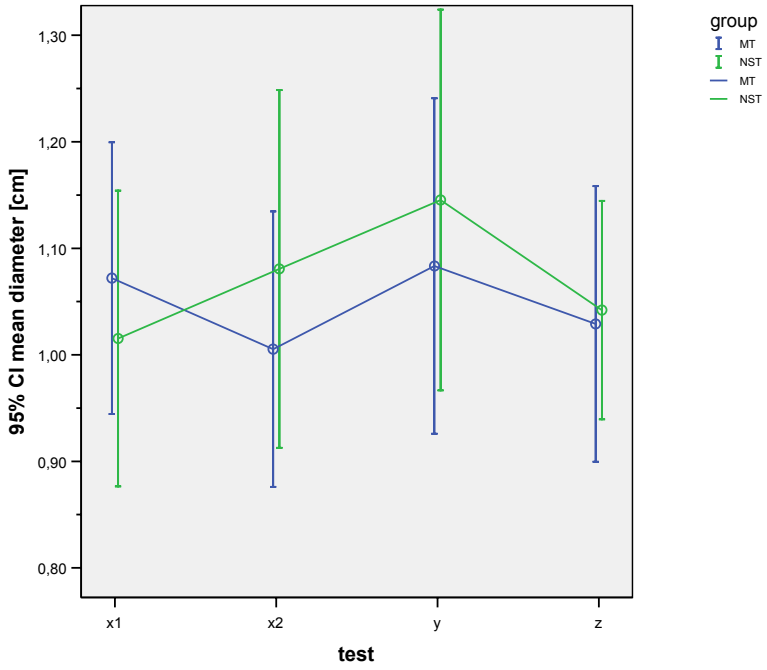


Fig. 13: Mean values and 95%-confidence intervals of the mean diameter of the portal vein during the individual measurements and broken down by groups.

Test-to test variability during the first two measurements (tests x1 and x2) was similar in both groups, but the mean values of the two measurements were diametrically opposed in the two groups. After intervention (between test x2 and test y) an almost parallel increase of the mean diameters could be observed in both groups. During measurement (z) the diameter of the portal vein was decreased

again to a value, almost corresponding to the mean of the measurements (x1) and (x2).

Results of repeated measures ANOVA (Table 17) indicate, that there was a significant difference between the individual tests, but not between the groups.

Variable: mean diameter		
Factor	F	P
Group	0.082	0.78
Test	3.144	**0.03**
Group * Test	2.008	0.12

Table 17: Effects of the factors "group", "test" and of the "group x test" interaction on the mean diameter of the portal vein.

In order to work out, how the diameter of the portal vein changed within the individual groups, results of paired samples t-tests (performed with the results of two consecutive tests, each) are displayed in Table 18.

Tests	Group	Mean	SD	P
x2-x1	MT	-0.067	0.102	*0.07*
	NST	0.065	0.076	**0.02**
	Total	-0.001	0.111	0.98
y-x2	MT	0.078	0.100	**0.04**
	NST	0.065	0.143	0.19
	Total	0.071	0.121	**0.02**
z-y	MT	-0.054	0.186	0.38
	NST	-0.103	0.148	*0.05*
	Total	-0.079	0.166	**0.05**

Table 18: Results of the paired samples t-tests with the mean diameters measured during two consecutive tests.

During the baseline measurements, there was a distinct difference between the results of measurement (x1) and (x2) in the MT group and even a significant one in the NST group. The mean diameter was decreasing with time in the MT group and increasing to a similar extent in the NST group. Therefore, after collapsing the two groups, there was almost no difference between the mean values of the two measurements at all (cf. "Total").

After intervention (at measurement y), a similar increase of the diameter could be observed in both groups, which was significant in the MT group and in total. Nevertheless, the increase in the MT group was only slightly higher than natural variability, evaluated by the baseline measurements (x1) and (x2). Additionally, according to Fig. 13, only the approximate initial mean value of measurement (x1)

was reached again. In contrary, an additional increase of the diameter could be observed in the NST group. In order to test, whether this intervention has significantly affected the diameter of the portal vein, mean values of the results of the baseline measurements were calculated and subsequently compared by means of paired samples t-tests.

Mean values and standard deviations are summarised in Table 19.

Group	**NST**			**MT**			**Total**		
Variable: mean diameter [cm]	N	Mean	SD	N	Mean	SD	N	Mean	SD
Test x (mean value of x1 and x2)	10	1.048	0.212	10	1.039	0.172	20	1.043	0.188
Test y	10	1.145	0.250	10	1.083	0.220	20	1.114	0.231

Table 19: Mean values and Standard deviations of the variable "mean diameter" during baseline measurements (test x…mean values of the results of test x1 and x2) and test y (after intervention).

Paired samples t-tests result in p= 0.06 in the NST group and in p= 0.20 in the MT group. That means, the increase of the diameter after NST intervention was distinct, but not significant, whereas in the MT group, means of the diameter did not differ to such a high extent.

12.4.2. Velocity of the Blood Flow in the Portal Vein

Mean values and standard deviations (SD) of the mean velocity of the blood flow in the portal vein during all tests are summarised for both groups in Table 20.

Group	**NST**			**MT**			**Total**		
Variable: mean velocity [cm/s]	N	Mean	SD	N	Mean	SD	N	Mean	SD
Test x1	10	15.29	3.71	10	13.74	2.64	20	14.52	3.24
Test x2	10	13.78	1.53	10	15.21	4.24	20	14.49	3.19
Test y	10	13.39	2.88	10	14.98	3.48	20	14.19	3.21
Test z	10	14.16	3.26	10	14.65	3.36	20	14.41	3.23

Table 20: Mean values and standard deviations (SD) of the mean velocity of blood flow in the portal vein during the individual tests in both groups.

Mean values and 95%-confidence intervals are displayed in Fig. 14 (The y-axis covers the actual range of data).

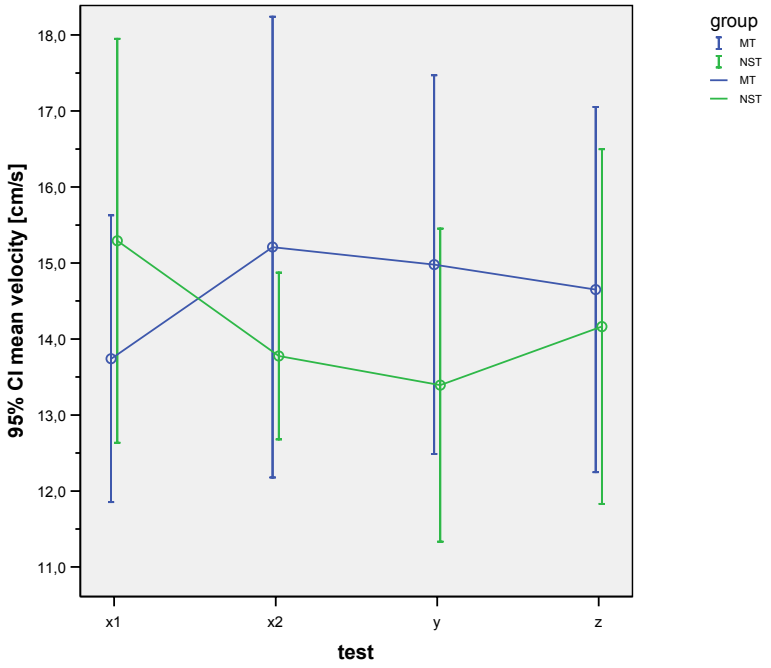


Fig. 14: Mean values and 95%-confidence intervals of the mean velocity of blood flow in the portal vein during the individual measurements and broken down by groups.

The baselines of the two groups (x1, x2) showed a high test-to-test variability of the mean velocity.

After intervention (test y), a slight, almost parallel decrease of the mean velocity could be observed in both groups. The extent of this decrease was less than the variability before intervention.

ANOVA outcomes (Table 21) show a similar result. There are no significant influences of the factors “group” and “test” and only tendential influences of the “group x test” interaction.

Variable: mean velocity		
Factor	F	P
Group	0.159	0.69
Test	0.111	0.95
Group * Test	2.65	*0.06*

Table 21: Effects of the factors “group”, “test” and of the “group x test” interaction on the mean velocity of the blood flow in the portal vein.

The effect of the “group x test” interaction is highest during the baseline measurements: The p-value for the group comparison of the differences in the

velocity between test x2 and test x1 is p= 0.05. The according p-values for the group comparisons of the differences between test y versus test x2 and test z vs. test y are p=0.88 and p=0.38, respectively. That means, that the two different methods did not influence velocity in a significantly different way.

12.5. Summary of the Results

12.5.1. The Test Persons

Groups corresponded in age and BMI, body weight and height.
Additionally, there were no group differences in systolic and diastolic blood pressure and neither in pulse rate. Group differences were less distinct than test-to-test variability of the data.
There are hints (a significant reduction of systolic and diastolic blood pressure and of pulse with time), that group members of the NST group could relax easier than test persons of the MT group.

12.5.2. Measurement Conditions

Measurement conditions (irradiation angles) were comparable in both groups.

12.5.3. Capacity of the Portal Vein

Test- to-test data variability of the capacity of the portal vein was comparable in the two groups during the two baseline measurements. After intervention, the increase of the capacity was more distinct in the MT group (p=0.10) than in the NST group (p= 0.33), but differences are not significant.

12.5.4. Diameter of the Portal Vein

Test-to test variability during the first two baseline measurements (tests x1 and x2) was similar in both groups. Nevertheless, significant “group x test” interactions could be observed in baseline analysis. Taking into consideration the mean values of the two baseline measurements, NST intervention turned out to affect the diameter more distinctly (p= 0.06) than the mobilisation technique (MT) (p=0.20).

12.5.5. Velocity of the Blood Flow in the Portal Vein

Analogical to the results of the diameter of the portal vein, test-to test variability during the first two baseline measurements (tests x1 and x2) was similar in both groups and again a significant “group x test” interaction could be observed in baseline analysis.

The two different methods did not influence velocity in a significantly different way.

13. Discussion

13.1. Ensuring the Causal Relationship between the Mobilisation Technique and the Results of the Measurements

In order to ensure the causal relationship between the administration of the two intervention techniques and the measurement results, two baseline measurements (x1) and (x2) were performed in advance of the application of the entire techniques. By a comparison of the results of the baseline measurements with the results at the moment y (after intervention) in the two single groups by repeated measures ANOVA, physiological changes in the portal venous blood flow by the application of the technique could be uncovered *under consideration of its natural range of variation.*

The control variables heart rate and blood pressure were used as indicators, if test persons had achieved a resting state before application of the osteopathic techniques.
These parameters were measured only twice in an interval of half an hour (x1 and x2) in advance of the administration of the mobilisation techniques. Admittedly, at least one additional measurement in advance of the intervention would have been necessary in order to document the stability of the resting state, but the longer waiting time might have induced reverse effects, that means, a re-increase in heart rate and blood pressure.
Since mean heart rate and systolic blood pressure were almost equal in both groups at measurement (x2), at least an equivalent initial situation in both groups can be assumed. Additionally, a significant reduction in the results of these measurements indicate, that the test persons calmed down during the waiting period in advance of the intervention.

13.2. Physiological Changes

13.2.1. Blood Pressure

The mean systolic value decreased in both groups between the measurements (x1) and (x2). The reduction was significant in the non-specific technique group (NST, p=0.02). Since lower pressures were measured in the MT already during (x1), the

decrease was less distinct in this group (p=0.32). Nevertheless, group means of the systolic blood pressure were identical during measurement (x2).

Since the test persons were blinded to which technique was applied, and thus did not know, what group they belonged to, these differences can be lead back to the individual blood pressure readings and the group assignment.
Probably, a larger sample would result in more comparable initial values.

The extent of the declination of the systolic blood pressure was comparable in both groups.
The latter does not apply to the diastolic values. While diastolic blood pressure decreased in the NST group between measurement (x1) and (x2), in contrast, a slight increase could be observed in the MT group. Individual unrest and nervousness in advance of the intervention and during the initial measurement might have had a crucial influence in the small sample. These influences were more distinct in the NST group than in the MT group, where the lower differences between the two baseline measurements in systolic as well as diastolic blood pressure can be explained by individual physiological fluctuations.

Ahead of the examination and between the measurements, it was tried to offer the test persons an as comfortable atmosphere as possible in the sense of time and calmness. Additionally, all participants were being informed about the purpose and course of the examination. However, it is probable, that each of the test persons reacted individually to the experimental setting.

13.2.2. Heart Rate

A parallel declination between the measurements (x1) and (x2) could be observed comparing the mean heart rate in the two groups. Also from this, an influence of increasing ease between the measurements (x1) and (x2) might be deduced.

Similar to the variable blood pressure, the decrease of heart rate was more distinct in the NST group (p= 0.05), than in the MT group (p=0.29).

13.2.3. Capacity of the Portal Vein

The capacity of the portal vein was calculated in two ways in this study. On the one hand, it was normalised by division by the body weight (variable *mean flow/min/kg body weight)*, on the other hand it was used without normalisation (variable *mean flow/min).* Thus, results of this study can be compared to literature data which are alternately presented normalised and non-normalised (cf. Table 22).

Reference	n	mean velocity (cm/s)		mean flow (ml/min)		mean flow (ml/min/kg BW)	
		$\bar{x}$	SD	$\bar{x}$	SD	$\bar{x}$	SD
Touche et al. 1982**	15	17		740			
Dauzat et al. 1984**	18			819	298	14.25	4.5
Gill et al. 1985**	16			820		12.7	2.9
Chou et al. 1985**	21	18.23	4.61	973	290		
Ohnishi et al. 1985**	41	16.5	4.9	648	186		
Ackroyd et al. 1986**	10			952	273	14.1	2.7
Moriyasu et al. 1986**	88	15.3	4	899	284	16.3	5
Moriyasu et al. 1986**	110	15.1	3.7	846	287		
Ohnishi et al 1986**	21			632	203		
Smith et al. 1986**	72	17		900			
Zoli et al. 1986**	50	16	0.5	694	23		
Okazaki et al. 1986*	40	21	5	966	344	14.7	3.9
Mostbeck et al. 1987**	15	15.37	4.48	755	276		
Ozaki et al. 1988**	22	18.99	0.86	874	44		
Brown et al. 1989**	35	12.32	5.90	864	188	13.45	3.2
Sabba et al. 1991*	12	13	0.4	1066	38		
Bolondi et al. 1992*	45	19	2				
Dauzat et al. 1994*	30	16	10	940	408		
Arienti et al. 1996*	9	22	1				
Iwao et al. 1996*	10	15	1	733	46		
Maconi et al. 1998*	40	18	3	1011	193		
Ludwig 1999*	30	17	2				
Haag, et al. 1999*	100	27	6	810	180		
Guadagni et al. 2000*	18			503	153		
Van Dun et al. 2007** before intervention(x)	30	11.25	3.82	760	268	10.5	4.3
Our examination Total, (x1)	20	14.52	3.24	761.7	306.1	10.1	4.1
Our examination MT, (x1)	10	13.74	2.64	766.7	258.6	10.2	3.6
Our examination MT, (x2)	10	15.21	4.24	744.7	318.4	10.0	4.4
Our examination MT, (y)	10	14.98	3.48	843.6	288.6	11.3	4.0

Table 22: Mean velocity and mean flow in the portal vein in several studies (… from Ignee et al. 2002, **… from van Dun et al. 2007) and the according results of the actual study.*

In this study, all measuring processes were performed under conditions that were standardised as much as possible, as was described in the chapters 8.1.3. and 9.3. At that, individual physical conditions as height, weight, and age were taken into account.
A healthy general condition of the test persons was predefined by non-smoking, alcohol intake within the defined “quantity of harmlessness”, as well as by the exclusion of heart- and liver pathologies.
Persons with inflammatory diseases, abdominal surgical interventions, and drug use were excluded in advance of the study.
Furthermore, all participants had to be on an empty stomach for at least eight hours before the start of the examinations. During the sonographic measurements it was paid attention, that the irradiation angle was smaller than 60°. For standardisation, ultrasound measurements were performed in apnoea (in resting midinspiration position).

***Mean flow/min/kg body weight*:** In scientific literature (cf. Table 22), a broad range of the capacity of the portal vein can be observed. For example, a mean value of 13.45 ml/kg body weight was described by BROWN et al. (1989) and a capacity of 10.5 ml/kg body weight by VAN DUN et al. (2007). In this connection, it has to be stressed, that some measurements (e.g. by *BROWN et al. (1989))* were performed in supine position, whereas *VAN DUN et al. (2007)* measured in lateral position (decubitus position left).

OHNISHI et al. (1985) describe a significantly reduced portal flow velocity as well as a decrease of the blood flow volume in healthy subjects, if they change from supine into a seated position.

Also HSIA et al. (2000) could verify the influence of gravity on the portal venous blood flow.
GALLIX et al. (1987) describe, that the pulsation behaviour of the portal vein changes significantly by moving from the supine into a seated position, what has to be considered in standardisation of the measurements.

Since changes in the body position change the portal blood flow decisively [OHNISHI et al. 1985, GALLIX et al. 1987, HSIA et al. 2000], all measurements

were performed in decubitus position left. Therefore, results are comparable to studies in supine position with reservations only.
Mean values (standard deviations) in the NST group were equal 10.0 (4.8) ml/min/kg body weight (BW) during baseline measurement (x1), and 10.3 (4.3) ml/min/kg BW during baseline measurement (x2). The according values in the MT group were 10.2 (3.6) ml/min/kg BW and 10.0 (4.2) ml/min/kg BW. That means, that the initial values were almost equal in both groups, and were excellently consistent with the results of *van Dun's* study.

Furthermore, baseline data of the portal capacity were very stable in both groups in contrast to blood pressure and heart rate, and they did not display dependencies on these parameters.

Although physiological changes could be observed during measurement (y), that means immediately after the particular intervention, they were not significant (NST group: p=0.33, MT group: p=0.10).
Tendency for an increased capacity was more distinct in the MT group (measurement y: 11.5 ml/min/kg BW), than in the NST group (measurement y: 11.4 ml/min/kg BW).

However, this has to be lead back to the lower initial values in the MT group during measurement (x2) and the lower variance of the results during measurement (y). There was no significant group difference in the change in portal capacity.

Values were lower again during measurement (z). They were comparable to the results of the baseline measurements (x1) and (x2). That means, that the effect of the interventions - as far as the changes were not basing on random variation - did last shorter than one hour.
To sum up, there were neither significant differences in the capacity behaviour between the NST group and the MT group, nor between the results of the baseline measurements (x1) and (x2) and measurement (y) within the individual groups.

Mean flow: Since body weight was considered for group assignment of the test persons, the variable "mean flow" did not differ relevantly in the changes during the four measurements from the variable „mean flow per kg body weight".

There are no significant differences - neither between the two groups, nor between the two baseline measurements (x1 and x2) and the post-test (y). Compared to the study by *VAN DUN et al. (2007),* where mean initial flow volumes of 760 ml/min were measured in the mobilisation group (MT), the mean initial flow volume of the test persons of the MT group was consistent (measurement x1: 766.7 ml/min, x2: 744.7 ml/min).

However, after the intervention in the study of *VAN DUN* (2007) values increased to 1079 ml/min, whereas in this study the mean flow reached only 843.6 ml/min in the measurement (y). Furthermore, no continuing effect of the intervention could be observed in the actual study in the results of measurement (z).

13.3. Examination of the Causes for the Increase in Capacity of the Portal Vein

13.3.1. Diameter of the Portal Vein

In literature, the diameter of the portal vein is specified with 6 – 12 mm [IGNEE et al. 2002] or 7 – 15 mm [HUCK 2004].

In this examination, mean values of 10.2 mm (x1) and 10.8 mm (x2) during the baseline measurements were observed in the NST group. The according values in the MT group were 10.7 mm and 10.1 mm, respectively. That means, there is a good agreement with literature data. Both (blinded) medical specialists measured test persons of each group at the portal confluence. Thus, different experience of the physicians in measurement could not influence the outcomes.

After the intervention, increased diameters could be observed in both groups. In measurement (y) a mean diameter of 11.5 mm was calculated for the NST group and of 10.8 mm for the MT group. The non-specific technique yielded in higher influences on the diameter ($p= 0.06$) than the specific mobilisation technique ($p= 0.20$). However, also this change can not be regarded as significant.
According to the results of measurement (z), the increased diameter could not be observed anymore in both groups one hour after the intervention, as can be

deduced from the almost equal values compared to the baseline values of the measurements (x1) and (x2).

13.3.2. Blood Flow Velocity in the Portal Vein

Several authors (e.g. IGNEE et al. 2002, HUCK 2004) stress the importance of the spot of the measurement at the portal confluence for the comparability of the results. Therefore, both medical specialists performed the measurements at this location. Both physicians were blinded against the technique and examined test persons (on an empty stomach) of both groups.

IGNEE et al. (2002) specify the averaged mean velocity (TAV_{mean}) on an empty stomach with 13 – 22 cm/s, *HUCK (2004)* with 15 – 20 cm/s. The group means are comparable with these data, although they are tendentially in the lower velocity range.

The values in the NST group were 15.3 cm/s during measurement (x1) and 13.8 cm/s during measurement (x2); the according values in the MT group were 13.7 cm/s and 15.2 cm/s. A high test-to-test variability could be observed in the results of the baseline measurements. After the particular intervention, a slight parallel lessening of the flow velocity could be observed in both groups. During measurement (y), the mean flow velocity was equal 13.4 cm/s in the NST group and equal 15.0 cm/s in the MT group. Neither the non-specific technique, nor the specific mobilisation technique had significant effects on the flow velocity in the portal vein.

VAN DUN (2007) describes an increased capacity of the portal vein after a mobilisation of the mesenterium, primarily due to an enlargement of the portal diameter. Consistent with the actual results, blood flow velocity played only a minor role.

13.4. Measurement Conditions (Irradiation Angle)

The mean irradiation angles, measured by the two medical specialists were lower than 60° in both groups, which is a directive in vascular diagnostics [LAFORTUNE et al. 1998, IGNEE et al. 2002, HUCK 2004]. Each medical specialist was blinded towards the intervention and performed sonographic measurements in either group. Generally, irradiation angles were lower in the MT group than in the NST group.

Nevertheless, differences between the two investigational groups and between the individual examinations were not significant.

13.5. Restrictions

As already mentioned several times, sample size (with ten test persons per group) is too low for save predications.
For example, this could be observed in the different progression of the blood pressure in the two groups during the baseline measurements.

Nevertheless, results of the baseline measurements of the main outcome variables “mean flow/kg body weight” and “mean flow” were consistent between the individual tests as well as between the two groups. Therefore, test-to-test variability was assumed to be the normal, that means independent variation of these parameters. By evaluation with repeated measures ANOVA, results of both baseline measurements and thus this variance were considered.

In order to ensure, that test persons reached a period of rest before intervention, eventually further baseline measurements of blood pressure and heart rate would be advisable, whereby a re-increase of blood pressure and heart rate might arise from to the longer lasting procedure.

14. Conclusions

Starting point of this study was the idea, to accomplish a basis for osteopathic activity in respect of measurable physiological changes.
The following null hypotheses were formulated:

- No changes on the physiological level are achieved by administration of a specific visceral technique (mobilisation of the lesser omentum).

- A mobilisation of the lesser omentum has no influence on the portal blood flow.

After performance of the study and evaluation of the results, both null hypotheses can be confirmed:

- **No changes at the physiological level are achieved by administration of a specific visceral technique.** The mobilisation technique for the lesser omentum, administered in this study under the chosen test conditions, does not cause significant physiological changes in the portal blood flow. The tendencies found in the examination have to be classified as statistically insignificant. A reason for the lack of significance might be the low sample size, with the resulting overvaluation of the influence of individual fluctuations.

- **A mobilisation of the lesser omentum has no influence on the portal blood flow.** It is not possible to clearly distinguish between true and random influences under the present test conditions and their restrictions (e.g. group size). The results are not significant, because values after intervention do not differ distinctly enough from the initial values. Nevertheless, results should not be taken as definite, since tendencies of elevated portal blood flow can be observed.

On the basis of the results of this investigation, only trends or tendencies can be observed, that possibly could confirm an influence of a mobilisation of the lesser omentum on the blood flow in the portal vein. Significant changes, as well as

persistent effects by a specific osteopathic mobilisation of the lesser omentum could not be established.

In this respect, a higher number of participants might achieve a higher significance.

15. Bibliography

AKIS - Alkohol Koordinations- und Informationsstelle des Anton Proksch Instituts (2007): Die „Harmlosigkeitsgrenze“ und die „Gefährdungsgrenze“. http://www.api.or.at/akis/texte/002/harmgef.htm [23.02.2008].

AROSIO, E.; DE MARCHI, S.; RIGONI, A.; PRIOR, M. and LECHI, A. (2006): Effects of smoking on cardiopulmonary Baroreceptor activation and peripheral vascular resistance. Eur. J. Clin. Invest. 36, 320-325.

BARRAL, J.P. and MERCIER, P. (2005): Lehrbuch der viszeralen Osteopathie, 2. Aufl. Band 1. München: Urban & Fischer.

BENEDIKT, H. and PANNEN, J. (2002): New Insights into the Regulation of Hepatic Blood Flow after Ischemia and Reperfusion. Anesth. Analg. 94, 1448-1457.

BENNINGHOFF, A. and DRENCKHAHN, D. (2008): Anatomie. 17. Aufl. Band 1. München-Jena: Urban & Fischer.

BLECHSCHMID, E. and GASSER R.F. (1978): Biokinetics and Biodynamics of Human Differentiation. Springfield: Thomas.

BOLOGNESI, M.; SACERDOTI, D.; MESCOLI, C.; BOMBONATO, G.; CILLO, U.; MERENDA, R.; GIACOMELLI, L.; MERKEL, C.; RUGGE; M. and GATTA, A. (2007): Different hemodynamic patterns of alcohol and viral endstage cirrhosis: analysis of explanted liver weight, degree of fibrosis and splanchnic Doppler parameters. Scand. J. Gastroenterol. 42, 256-262.

BOLONDI, L.; GAIANI, S.; BRIGNOLA, C.; CAMPIERI, M.; RIGAMONTI, A.; ZIRONI, G.; GINOCHETTI, P.; BELLOLI, C.; MIGLIOLI, M. and BARBARA, L. (1992): Changes in Splanchnic Hemodynamics in Inflammatory Bowel Disease Non-Invasive Assessment by Doppler Ultrasound Flowmetry. Scand. J. Gastroenterol. 27, 501-507.

BOMBELLI, L.; GENITONI, V.; BIASI, S.; MATERAZZO, C. and BONFANTI, G. (2005): Liver hemodynamic flow balance by image-directed Doppler ultrasound evaluation in normal subjects. Journal of Clinical Ultrasound. 19, 257-262.

BOUCHET, A. and CUILLERET, J. (2001): Anatomie – topographique descriptive et fonctionelle. 2^{e} édition. Tome 4. Paris: Simep.

BROWN H.S.; HALLIWELL, M.; QAMAR, M.; READ, A.E.; EVANS, J.M. and WELLS, P.N. (1989): Measurement of normal portal venous blood flow by Doppler ultrasound. Gut. 30, 503-509.

BÜCHELER, E.; LACKNER, K.J. and THELEN, M. (2006): Einführung in die Radiologie. 11. Aufl. Stuttgart-New York: Thieme.

CHUO, L. S.; MAHMUD, R. and SALIH, Q.A. (2005): Colour Doppler Ultrasound examination of the main portal vein and inferior cava in normal malaysian adult population: A fasting and postprandial evaluation. The Internet Journal of Cardiovascular Research. Vol 2. Number 2.

CLAPP, J.F.; STEPANCHAK, W.; TOMASELLI, J.; KORTAN, M. and FANESLOW, S. (2000): Portal vein blood flow: Effects of pregnancy, gravity, and exercise. Am. J. obstet. and gynecol.183, 167-172.

CLEMENS, M.G. and ZHANG J.X. (1999): Regulation of sinusoidal perfusion: in vivo methodology and control by endothelins. Semin. Liver Dis. 19, 383-396.

CLUZEL, PH.; SIMILOWSKI, TH.; CHARTRAND-LEFEBVRE, C.; ZELTER, M.; DERENNE, J. PH. and GRENIER, PH. A. (2000): Diaphragm and chest wall: assessment of the inspiratory pump with MR imaging-preliminary observations. Radiology. 215, 574-583.

DAUZAT, M.; LAFORTUNE, M.; PATRIQUIN, H. and POMIER- LAYRAGUES, G. (1994): Meal induced changes in hepatic and splanchnic circulation: a noninvasive Doppler study in normal humans. European Journal of Physiology. 68, 373-380.

DEGUM - Deutsche Gesellschaft für Ultraschall in der Medizin (2004): Ultraschall unverzichtbar in der Medizin. Presse-Info Ultraschall in der Medizin. www.sonoweb.de/downloads/PresseInfoSept2004.pdf [15.03.2008]

DEGUM - Deutsche Gesellschaft für Ultraschall in der Medizin (2006): Ultraschall bei Schwangeren ungefährlich. Informationsdienst Wissenschaft. Pressemitteilung. http://idw-online.de/pages/de/news176783 [15.03.2008]

DIVISION OF PHYSIOLOGY, Departement of Physiology and Medical Physics, Innsbruck Medical University, Austria (2008). http://physiologie.i-med.ac.at/index.php [20.08.2008]

EZZAT, W.R. and LAUTT, W.W. (1987): Hepatic arterial pressure- flow autoregulation is adenosine mediated. Am. J. Physiol. 252, 836-845.

FINET, G. and WILLIAME, CH. (2000): Treating Visceral Dysfunction – An Osteopathic Approach to Understanding and Treating the Abdominal Organs. Portland: Stillness Press, LLC.

GAIANI, S.; BOLONDI, L.; LI BASSI, S.; SANTI, V.; ZIRONI, G. and BARBARA, L. (2005): Effect of meal on portal hemodynamics in healthy humans and in patients with chronic liver disease. Hepathology. 9, 815-819.

GALLIX, B.P.; TAOUREL, P.; DAUZAT, M.; BRUEL, J.M. and LAFORTUNE, M. (1997): Flow Pulsatility in the Portal Venous System: A Study of Doppler Sonography in Healthy Adults. AJR. 169, 141-144.

GLÉNARD, F. (1899): Les Ptoses viscèrales. Paris: F.Alcan.

HANSON K.M. and JOHNSON, P.C. (1966): Local control of hepatic arterial and portal venous flow in the dog. Am. J. Physiol. 211, 712-720.

HARTEN, U. (2007): Physik für Mediziner. 12. Aufl. Heidelberg: Springer.

HELSMOORTEL, J.; HIRTH, TH. and WÜHRL, P. (2002): Lehrbuch der viszeralen Osteopathie. Stuttgart-New York: Thieme.

HOSOKI, T.; ARISAWA, J.; MARUKAWE, T.; TOKUNAGA, K.; KURODA, CH.; KOZUKA, T. and NAKANO, S. (1990): Portal Blood Flow in congestive Heart Failure: Pulsed Duplex Sonographic Findings. Radiology. 174, 733-736.

HSIA, T.Y.; KHAMBADKONE, S.; REDINGTON, A.N.; MIGLIAVACCA, F.; DEANFIELD, J.E. and DE LEVAL, M.R. (2000): Effects of Respiration and Gravity on Infradiaphragmatic Venous Flow in Normal and Fontan Patients. Circulation. 102, 148-153.

HUCK, K. (2004): Kursbuch Doppler - und Duplexsonographie. Stuttgart: Thieme.

IGNEE, A.; GEBEL, M.; CASPARY, W.F. and DIETRICH, CH.F. (2002): Duplexsonographie der Lebergefäße – eine Übersicht. Z Gastroenterol. 40, 21- 32.

KAMKE, D. and WALCHER, W. (1994): Physik für Mediziner. 2. Aufl. Wiesbaden: Vieweg-Teubner.

KAYACETIN, E.; EFE, D. and DOGAN, C. (2004): Portal and splenic hemodynamics in cirrhotic patients: relationship between esophageal variceal bleeding and severity of hepatic failure. J. Gastroenterol. 39, 661-667.

KOK, T.; VAN DER JAGT, E.T.; HAAGSMA, E.B.; BIJLEVELD, C.M.; JANSEN, P.L. and BOEVE, W.J. (1999): The value of Doppler ultrasound in cirrhosis and portal hypertension. Scand. J. Gastroenterol. Suppl. 230, 82-88.

KRÄHENBÜHL, L.; SEILER, CH.A. and BÜCHLER, W. (1999) : Ösophagusvarizenblutung bei portaler Hypertonie. Schweiz. Med. Wochenschr. 129, 631-638.

KUBALE, R. and STIEGLER, H. (2002): Farbkodierte Duplexsonographie - Interdisziplinärer vaskulärer Ultraschall. Stuttgart: Thieme.

KUNZE, W.A.A.; FURNESS, J.B.; BERTRAND, P.P. and BORNSTEIN, J.C. (1998): Intracellular recording from myenteric neurons of the guinea-pig ileum that respond to stretch. J. Physiol. 506, 827-842.

KUNZE, W.A.A.; CLERC, N.; FURNESS, J.B. and GOLA, M. (2000): The soma and neurites of primary afferent neurons in the guinea-pig intestine respond differentially to deformation. J. Physiol. 526, 375-385.

LAFORTUNE, M.; PATRIQUIN, H.; BURNS, P.N. and DAUZAT, M. (1998): Doppler Sonographie measurement of Portal Venous Flow: What went wrong? Radiology. 206, 844-846.

LAUTT, W.W. (1980): Hepatic nerves: a review of their functions and effects. Can. J. Pharmacol. 58, 105-123.

LAUTT, W. W. and GREENWAY C. V. (1987): Conceptual Review of the Hepatic Vascular Bed. Hepatology. 7, 952-963.

LAUTT, W.W.; LEGARE, D.J. and EZZAT, W.R. (1990): Quantitation of hepatic arterial buffer response to graded changes in portal blood flow. Gastroenterology. 98, 1024-1028.

LAUTT, W.W. (1996): The 1995 Ciba-Geigy Award Lecture. Intrinsic regulation of hepatic blood flow. Can. J. Physiol. Pharmacol. 74, 223-233.

LEEN, E.; GOLDBERG, J.A.; ANDERSON, J.R.; ROBERTSON, J.; MOULE, B.; COOKE, T.G. and MC ARDLE, C.S. (1993): Hepatic perfusion changes in patients with liver metastases: comparison with those patients in cirrhosis. Gut. 34, 554-557.

LIEM T.; DOBLER K. and PUYLAERT M. (2005): Leitfaden - Viszerale Osteopathie. München: Urban & Fischer.

MANCIA, G.; GROPPELLI, A.; DI RIENZO, M.; CASTIGLIONI, P. and PARATI, G. (1997): Smoking impairs baroreflex sensitivity in humans. Am. J. Physiol. 273, 1555-1560.

MATHIE, RT. and BLUMGART, L.H. (1983): Effect of denervation on the hepatic haemodynamic responses to hypercapnia and hypoxia in the dog. Pflügers Arch. European Journal of Physiology. 397, 152-157.

NOLTE, W.; HARTMANN, H. and RAMADORI, G. (1994): Portale Hypertension-Pathophysiologie und Therapieansätze. Z Gastroenterol. 32, 447-459.

OHNISHI, K.; SAITO, M.; NAKAYAMA, T.; IIDA, S.; NOMURA, F.; KOEN, H. and OKUDA, K. (1985): Portal venous hemodynamics in chronic liver disease: effects of posture change and exercise. Radiology. 155, 757-761.

PANNEN, B.H.J. and BAUER, M. (1998): Differential regulation of hepatic arterial and portal venous vascular resistance by nitricoxide and carbon monoxide in rats. Life Sciences. 62, 2025-2033.

REHRER, N.J.; SMETS, A.; REYNAERT, H.; GOES, E. and DE MEIRLEIR, K. (2001): Effect of exercise on portal vein blood flow in man. Med. Sci. Sports Exerc. 33, 1533-1537.

REILLY, F.D.; MC CUSKEY, R.S. and CILENTO, E.V. (1981): Hepatic microvascular regulatory mechanisms. Microvasc. Res. 21, 103-116.

ROCHELEAU, B.; ÉTHIER, C.; HOULE, R.; HUET, P.M. and BILODEAU, M. (1999): Hepatic artery buffer response following left portal vein ligation: its role in liver tissue homeostasis. Am. J. Physiol. Gastrointest. Liver Physiol. 277, 1000-1007.

ROHEN, J.W. (2001): Funktionelle Neuroanatomie. 6. Aufl. Stuttgart: Schattauer.

ROHEN, J.W. and LÜTJEN - DRECOLL, E. (2002): Funktionelle Embryologie. Stuttgart. Schattauer.

ROHEN, J.W. (2007): Topographische Anatomie. 10. Aufl. Stuttgart - New York: Schattauer.

ROTHE, CF. and MAASS - MORENO, R. (1998): Hepatic venular resistance responses to norepinephrine, isoproterenol, adenosine, histamine, and ACh in rabbits. Am. J. Physiol. 274, 777-785.

SABBA, C.; FERRAIOLI, G.; SARIN, K.; LERNER, E.; GROSZMANN, R.J. and TAYLOR, K.J. (1990): Feasibility spectrum for Doppler flowmetry of splanchnic vessels. In normal and cirrhotic populations. Journal of Ultrasound in Medicine. 9, 705-710.

SABBA, C.; WELTIN, G.G.; CICCHETTI, D.V.; FERRAIOLI, G.; TAYLOR, K.J.; NAKAMURA, T.; MORIYASU, F. and GROSZMAN R.J. (1990): Observer variability in echo- Doppler measurements of portal flow in cirrhotic patients and normal volunteers. Gastroenterology. 98, 1603-1611.

SABBA, C.; FERRAIOLI, G.; GENECIN, P.; COLOMBATO, L.; BUONAMICO, P.; LERNER, E.; TYLOR, K.J.W. and GROSZMANN, R.J. (1991): Evaluation of postprandial hyperemia in superior mesenteric artery and portal vein in healthy and cirrhotic humans: An operator- blind echo-Doppler study. Hepatology. 13, 714–718.

SCHENK, W.G.; MC DONALD, J.C.; MC DONALD, K. and DRAPANAS, TH. (1962): Direct measurement of hepatic blood flow in surgical patients. Ann. Surg. 156, 463-469.

SEITZ, K. (2006): Zur Qualität der Oberbauchsonographie. Ultraschall in Med. 27, 217-219.

SIEGENTHALER, W. and BLUM, H.E. (2006): Klinische Pathophysiologie. 9. Aufl. Stuttgart: Thieme.

SILBERNAGEL, S. and DESPOPOULOS A. (2001): Taschenatlas der Physiologie. 5. Aufl. Stuttgart-New York: Thieme.

SUGANO, S.; YAMAMOTO; K.; SASAO, K.I and WATANABE, M. (1999): Portal venous blood flow while breath holding after inspiration or expiration and during normal respiration in controls and cirrhotics. Journal of Gastroenterology. 34, 613-618.

SUGIMOTO, H.; KANEKO, T.; HIROTA, M.; INOUE, S.; TAKEDA, S. and NAKAO, A. (2005): Physical hemodynamic interaction between portal venous and hepatic arterial blood flow in humans. Liver Int. 25, 282-287.

TAI, D.I.; CHUAH, S.K.; CHEN, CH.L.; LO, S.K. CHANGCHIEN, CH.S. and LI; I.T. (1996): Inter- observer variability of portal hemodynamics measured by Doppler ultrasound on three different locations of portal vein. Journal of Clinical Ultrasound. 24, 61-66.

TAKALA, J. (1997): Determinants of splanchnic blood flow. British Journal of Anaesthesia. 77, 50-58.

TEICHGRÄBER, U.K.; GEBEL, M.; BENTER, T. and MANNS, M.P. (1997): Effect of respiration, exercise, and food intake on hepatic vein circulation. Ultrasound Med. 16, 549-554.

TRACHSEL, H. (2008): „Die Antwort auf jede Frage führt zur nächsten Frage“. Zentrum für Biosicherheit und Nachhaltigkeit. http://www.bats.ch/bats/biosicherheit/blickpunkt/00-12_Grundlagenforschung.php [25.04.2008]

UNIVERSITÄTSSPITAL ZÜRICH (2008): Institut für Diagnostik und Radiologie. Sicherheit Ultraschall. http://www.radiologie.usz.ch/german/PatientenUndBesucher/SicherheitStrahlenschutz/Ultraschall.htm [15.03.2008]

VAN DEN BERG, F. (2003): Angewandte Physiologie. 2. Aufl. Stuttgart-New York: Thieme.

VAN DUN, P.L.S.; DOBBELAERE, E.; DILLIES, P.; INGHELBRECHT, F.; VAN EEGHEM, P.; STEYAERT, L.; DANSE, E. and ROSSEEL, Y. (2007): Invloed van een mobilisatie van het mesenterium ophet hepatisch portale debiet, geobjectiveerd door echo – Doppler.*[Influence of a mobilisation of the mesentery upon the capacity of the portal vein, reported by echo -Doppler].* De Osteopaat. 8, 22-30.

WEBSTER, A.J.F.; OSUJP, O.; WHITE, F. and INGRAM, J.F. (1975): The influence of food intake on portal blood flow and heat production in the digestive tract of sheep. British Journal of Nutrition. 34, 125-139.

WIEST, R.; LOCK, G. and SCHÖLMERICH, J. (2000): Portale Hypertension. Der Internist. 41, 1077-1095.

WYNNE, H.A.; COPE, L.H.; MUTCH, E.; RAWLINS, M.D.; WOODHOUSE, K.W. and JAMES, O.F. (1989): The effect of age upon liver volume and apparent liver blood flow in healthy man. Hepathology. 9, 297-301.

Yin, X.Y.; LU, M.D.; HUANG, J.F.; XIE, X.Y. and LIANG, L.J. (2001): Color Doppler velocity profile assessment of portal hemodynamics in cirrhotic patients with portal hypertension: correlation with esophagal variceal bleeding. J. Clin. Ultrasound. 29, 7-13.

YOSHIHARA, H.; SATO, N.; SASAKI, Y.; UCHIMA, E.; INOUE, A.; MATSUMURA, T.; HAYASHI, N.; KAWANO, S.; KAMADA, T. and ABE, H. (1985): Effect of alcohol ingestion on portal venous blood flow in healthy volunteers: comparison between the subjects with and without ALDH I isozyme. Alcohol. 2, 463-468.

ZOLI, M.; IERVESE, T.; ABBATI, S.; BIANCHI, G.P.; MARCHESINI, G. and PISI, E. (1989): Portal blood velocitx and flow in aging man. Gerontology. 35, 61-65.

ZOLI, M.; MAGALOTTI, D.; BIANCHI, G.P.; GUELI, C.; ORLANDINI, C.; GRIMALDI, M. and MARCHESINI, G. (1999): Total and functional hepatic blood flow decrease in parallel with ageing. Age and Ageing. 28, 29-33.

16. Table of Illustrations

17. Appendix

1. Match controlled (All)

MATCH CONTOLLED WITH THE PARAMETERS AGE AND BMI

Name / ID	Age	Weight	Tallness	BMI	BMI * Age	Question with BMI * Age	Average BMI of Group NST	Average BMI of Group MT	Average Age of Group NST	Average Age of Group MT
							23.17	23.14	34.30	34.30
RP	23	65	1.79	20.29	466.6	MT		20.29		23.00
RT	26	77	1.86	22.26	578.7	NST	22.26		26.00	
KR	26	79	1.82	23.85	620.1	MT		23.85		26.00
PE	29	85	1.82	25.66	744.2	NST	25.66		29.00	
PT	30	78	1.77	24.90	746.9	NST	24.90		30.00	
LG	32	84	1.83	25.08	802.7	MT		25.08		32.00
AD	34	63	1.75	20.57	699.4	NST	20.57		34.00	
ALJ	34	70	1.72	23.66	804.5	MT		23.66		34.00
RA	35	70	1.73	23.39	818.6	MT		23.39		35.00
PH	36	73	1.78	23.04	829.4	MT		23.04		36.00
GCH	37	85	1.83	25.38	939.1	NST	25.38		37.00	
ALJÜ	38	75	1.84	22.15	841.8	NST	22.15		38.00	
FRAU	38	88	1.90	24.38	926.3	NST	24.38		38.00	
SCHHE	39	70	1.82	21.13	824.2	MT		21.13		39.00
SCHH	39	78	1.88	22.07	860.7	NST	22.07		39.00	
FRI	39	75	1.81	22.89	892.8	NST	22.89		39.00	
SJ	39	74	1.78	23.36	910.9	MT		23.36		39.00
HC	39	82	1.87	23.45	914.5	MT		23.45		39.00
MJ	40	79	1.81	24.11	964.6	MT		24.11		40.00
HM	33	62	1.70	21.45	708.0	NST	21.45		33.00	

2. Match controlled (NST)

MATCH CONTOLLED WITH THE PARAMETERS AGE AND BMI (NST)

Name / ID	Age	Weight	Tallness	BMI	BMI * Age	Question with BMI * Age	Average BMI of Group NST	Average BMI of Group MT	Average Age of Group NST	Average Age of Group MT
							23.17		34.30	
RT	26	77	1.86	22.26	578.7	NST	22.26		26.00	
PE	29	85	1.82	25.66	744.2	NST	25.66		29.00	
PT	30	78	1.77	24.90	746.9	NST	24.90		30.00	
AD	34	63	1.75	20.57	699.4	NST	20.57		34.00	
GCH	37	85	1.83	25.38	939.1	NST	25.38		37.00	
ALJÜ	38	75	1.84	22.15	841.8	NST	22.15		38.00	
FRAU	38	88	1.90	24.38	926.3	NST	24.38		38.00	
SCHH	39	78	1.88	22.07	860.7	NST	22.07		39.00	
FRI	39	75	1.81	22.89	892.8	NST	22.89		39.00	
HM	33	62	1.70	21.45	708.0	NST	21.45		33.00	

3. Match controlled (MT)

MATCH CONTOLLED WITH THE PARAMETERS AGE AND BMI (MT)

Name / ID	Age	Weight	Tallness	BMI	BMI * Age	Question with BMI * Age	Average BMI of Group NST	Average BMI of Group MT	Average Age of Group NST	Average Age of Group MT
								23.14		34.30
RP	23	65	1.79	20.29	466.6	MT		20.29		23.00
KR	26	79	1.82	23.85	620.1	MT		23.85		26.00
LG	32	84	1.83	25.08	802.7	MT		25.08		32.00
ALJ	34	70	1.72	23.66	804.5	MT		23.66		34.00
RA	35	70	1.73	23.39	818.6	MT		23.39		35.00
PH	36	73	1.78	23.04	829.4	MT		23.04		36.00
SCHHE	39	70	1.82	21.13	824.2	MT		21.13		39.00
SJ	39	74	1.78	23.36	910.9	MT		23.36		39.00
HC	39	82	1.87	23.45	914.5	MT		23.45		39.00
MJ	40	79	1.81	24.11	964.6	MT		24.11		40.00

4. Examination Form

Date:	

Time	ID/Name	Condition	Doctor	Question	Weight/kg	Tallness/m	Age/years

Time/min	HR X1 Puls/min	Velocity 1 cm /s	Velocity 2 cm /s	Velocity 3 cm/s	Angle ° 1	Angle ° 2	Angle ° 3
	RR mmHg						

Diameter X1	Diameter 1 cm	Diameter 2 cm	Diameter 3 cm

Time/min	HR X2 Puls/min	Velocity 1 cm /s	Velocity 2 cm /s	Velocity 3 cm/s	Angle ° 1	Angle ° 2	Angle ° 3
	RR mmHg						

Diameter X2	Diameter 1 cm	Diameter 2 cm	Diameter 3 cm

Time/min	HR Y Puls/min	Velocity 1 cm/s	Velocity 2 cm/s	Velocity 3 cm/s	Angle ° 1	Angle ° 2	Angle ° 3
	RR mmHg						

Diameter Y	Diameter 1 cm	Diameter 2 cm	Diameter 3 cm

Time/min	HR Z Puls/min	Velocity 1 cm/s	Velocity 2 cm/s	Velocity 3 cm/s	Angle ° 1	Angle ° 2	Angle ° 3
	RR mmHg						

Diameter Z	Diameter 1 cm	Diameter 2 cm	Diameter 3 cm

5. Schedule of the Examination

8:00 AM	Echo 1						
8:05 AM							
8:10 AM							
8:15 AM		Echo 1					
8:20 AM							
8:25 AM			Echo 1				
8:30 AM	Echo 2						
8:35 AM	Mobil						
8:40 AM	Echo 3			Echo 1			
8:45 AM		Echo 2					
8:50 AM		Mobil					
8:55 AM		Echo 3	Echo 2				
9:00 AM			Mobil				
9:05 AM			Echo 3				
9:10 AM				Echo 2			
9:15 AM				Mobil			
9:20 AM				Echo 3			
9:25 AM					Echo 1		
9:30 AM							
9:35 AM						Echo 1	
9:40 AM	Echo 4						
9:45 AM							Echo 1
9:50 AM							
9:55 AM		Echo 4			Echo 2		
10:00 AM					Mobil		
10:05 AM			Echo 4		Echo 3	Echo 2	
10:10 AM						Mobil	
10:15 AM						Echo 3	Echo 2
10:20 AM				Echo 4			Mobil
10:25 AM							Echo 3
10:30 AM							
10:35 AM							
10:40 AM							
10:45 AM							
10:50 AM							
10:55 AM							
11:00 AM							
11:05 AM					Echo 4		
11:10 AM							
11:15 AM						Echo 4	
11:20 AM							
11:25 AM							Echo 4

	Echo measurement
	Mobilisation technique
	Rest

6. Raw Data (x1) and (x2)

MEASUREMENT 1 (X1)

Time X1 min	Initial	Condition	Dr	Question	Weight kg	Height m	BMI	Age years	RR mm Hg Syst.	RR mm Hg Diast.	HR puls/min	Flow 1 ml/min	Flow 2 ml/min	Flow 3 ml/min	Average ml/min	ml/ min/ Kg	Diameter 1 cm	Diameter 2 cm	Diameter 3 cm	Average cm	Velocity cm/s	Velocity cm/s	Velocity cm/s	Average cm/s	Angle 1 °	Angle 2 °	Angle 3 °	Average °
0	MJ	1X1-1	2	MT	79	1,81	24,11	40	119	76	52	529	928	793	749,84	9,49	1,17	1,35	1,26	1,26	8,20	10,80	10,60	9,87	56	56	56	56,00
0	RA	1X1-2	2	MT	70	1,73	23,39	35	132	89	68	1041	1020	871	977,21	13,96	1,27	1,23	1,17	1,22	13,70	14,30	13,50	13,83	53	53	55	53,67
0	HC	1X1-3	2	MT	82	1,87	23,45	39	111	66	45	471	689	707	622,04	7,59	0,87	1,09	1,01	0,99	13,20	12,30	14,70	13,40	60	60	60	60,00
0	SCHH	1X1-4	2	NST	78	1,88	22,07	39	117	72	46	481	857	1031	789,67	10,12	0,86	1,31	1,25	1,14	13,80	10,60	14,00	12,80	60	60	60	60,00
0	PT	1X1-5	2	NST	78	1,77	24,90	30	150	97	88	754	927	661	780,58	10,01	1,04	1,02	0,98	1,01	14,80	18,90	14,60	16,10	60	60	60	60,00
0	GCH	1X1-6	2	NST	85	1,83	25,38	37	135	88	65	337	579	412	309,54	5,99	1,09	1,12	0,98	1,06	9,60	9,80	9,10	9,50	60	60	60	60,00
0	LG	1X1-7	2	MT	84	1,83	25,08	32	140	84	60	828	1234	716	925,73	11,02	1,12	1,18	1,13	1,14	14,00	18,80	11,90	14,90	26	26	26	26,00
0	SJ	1X1-8	2	MT	74	1,78	23,36	39	103	67	63	1321	1167	1097	1194,96	16,15	1,19	1,16	1,15	1,17	19,80	18,40	17,60	18,60	61	63	62	62,00
0	KR	1X1-9	2	MT	79	1,82	23,85	26	146	89	55	286	719	800	601,67	7,62	0,61	0,88	1,13	0,87	16,30	19,70	13,30	16,43	60	62	56	59,33
0	RP	1X1-10	1	MT	65	1,79	20,29	23	122	83	43	245	290	286	273,36	4,21	0,70	0,80	0,76	0,75	10,60	9,60	10,50	10,23	60	60	60	60,00
0	ALJ	1X1-11	1	MT	75	1,72	25,35	34	102	72	49	578	819	359	585,30	7,80	0,92	1,02	0,79	0,91	14,50	16,70	12,20	14,47	60	57	57	58,00
0	SCHHE	1X1-12	1	MT	70	1,82	21,13	39	105	68	81	897	910	926	910,79	13,01	1,27	1,29	1,22	1,26	11,80	11,60	13,20	12,20	60	60	60	60,00
0	PH	1X1-13	1	MT	73	1,78	23,04	36	122	81	71	734	787	958	826,24	11,32	1,07	1,17	1,18	1,14	13,60	12,20	14,60	13,47	57	60	60	59,00
0	PE	1X1-14	2	NST	85	1,82	25,66	29	125	83	51	402	502	432	445,43	5,24	0,84	0,98	0,87	0,90	12,10	11,10	12,10	11,77	55	55	60	56,67
0	ALJÜ	1X1-15	1	NST	75	1,84	22,15	38	121	79	68	574	477	614	555,18	7,40	0,95	0,90	0,99	0,95	13,50	12,50	13,30	13,10	60	60	60	60,00
0	HM	1X1-16	1	NST	62	1,70	21,45	33	112	84	66	834	701	657	730,64	11,78	0,92	0,85	0,89	0,89	20,90	20,60	17,60	19,70	60	60	60	60,00
0	FRU	1X1-17	1	NST	88	1,90	24,38	38	125	80	58	705	842	833	793,37	9,02	0,84	0,97	0,94	0,92	21,20	19,00	20,00	20,07	56	56	58	56,67
0	FRI	1X1-18	1	NST	75	1,81	22,89	39	119	63	52	451	607	627	561,84	7,49	0,68	0,83	0,82	0,78	20,70	18,70	19,80	19,73	60	60	60	60,00
0	AD	1X1-19	1	NST	63	1,75	20,57	34	118	77	50	717	756	566	679,39	10,78	0,96	1,09	1,04	1,03	16,50	13,50	11,10	13,70	53	55	55	54,33
0	RT	1X1-20	1	NST	77	1,86	22,26	26	127	75	62	1575	1651	1938	1721,62	22,36	1,45	1,44	1,36	1,48	15,90	16,90	16,90	16,57	60	60	60	60,00

MEASUREMENT 2 (X2)

Time X1 → X2 min	Initial	Condition	Dr	Question	Weight kg	Height m	BMI	Age years	RR mm Hg Syst.	RR mm Hg Diast.	HR puls/min	Flow 1 ml/min	Flow 2 ml/min	Flow 3 ml/min	Average ml/min	ml/ min/ Kg	Diameter 1 cm	Diameter 2 cm	Diameter 3 cm	Average cm	Velocity cm/s	Velocity cm/s	Velocity cm/s	Average cm/s	Angle 1 °	Angle 2 °	Angle 3 °	Average °
32	MJ	1X2-1	2	MT	79	1,81	24,11	40	119	81	46	491	609	1097	732,06	9,27	0,92	1,12	1,37	1,14	12,30	10,30	12,40	11,67	54	58	53	55,00
30	RA	1X2-2	2	MT	70	1,73	23,39	35	118	81	60	665	540	775	660,07	9,43	1,05	1,11	1,33	1,16	12,80	9,30	9,30	10,47	60	60	60	60,00
28	HC	1X2-3	2	MT	82	1,87	23,45	39	108	76	44	347	307	310	321,31	3,92	0,78	0,69	0,74	0,74	12,10	13,70	12,00	12,60	60	60	60	60,00
30	SCHH	1X2-4	2	NST	78	1,88	22,07	39	109	61	44	944	1106	1171	1073,51	13,76	1,32	1,37	1,30	1,33	11,50	12,50	14,70	12,90	60	60	60	60,00
30	PT	1X2-5	2	NST	78	1,77	24,90	30	132	83	75	912	876	1077	955,12	12,25	1,10	1,14	1,17	1,14	16,00	14,30	16,70	15,67	54	60	60	58,00
32	GCH	1X2-6	2	NST	85	1,83	25,38	37	116	78	61	805	904	787	832,04	9,79	1,04	1,21	1,17	1,14	15,80	13,10	12,20	13,70	60	60	60	60,00
30	LG	1X2-7	2	MT	84	1,83	25,08	32	128	82	59	784	1165	540	829,76	9,88	1,01	1,15	1,05	1,07	16,30	18,70	10,40	15,13	26	26	26	26,00
28	SJ	1X2-8	2	MT	74	1,78	23,36	39	117	72	60	1381	1167	1670	1406,04	19,00	1,19	1,26	1,21	1,22	20,70	15,60	24,20	20,17	61	61	64	62,00
30	KR	1X2-9	2	MT	79	1,82	23,85	26	140	87	53	727	937	828	830,47	10,51	0,81	0,89	0,84	0,85	23,50	25,10	24,90	24,50	57	57	57	57,00
33	RP	1X2-10	1	MT	65	1,79	20,29	23	119	74	42	278	443	606	442,41	6,81	0,75	0,86	0,92	0,84	10,50	12,70	15,20	12,80	60	64	60	61,33
33	ALJ	1X2-11	1	MT	75	1,72	25,35	34	107	79	53	449	506	432	462,68	6,17	0,85	0,83	0,86	0,85	13,20	15,60	12,40	13,73	51	51	51	51,00
30	SCHHE	1X2-12	1	MT	70	1,82	21,13	39	106	71	86	1059	984	1145	1062,37	15,18	1,20	1,23	1,26	1,23	15,60	13,80	15,30	14,90	60	60	60	60,00
30	PH	1X2-13	1	MT	73	1,78	23,04	36	112	78	70	702	647	749	699,45	9,58	0,92	0,96	1,00	0,96	17,60	14,90	15,90	16,13	57	57	57	57,00
29	PE	1X2-14	2	NST	85	1,82	25,66	29	122	82	52	346	602	475	474,40	5,58	0,74	0,98	0,84	0,85	13,40	13,30	14,30	13,67	60	60	60	60,00
33	ALJÜ	1X2-15	1	NST	75	1,84	22,15	38	124	84	67	613	742	628	660,94	8,81	1,05	1,15	0,99	1,06	11,80	11,90	13,60	12,43	60	55	57	57,33
35	HM	1X2-16	1	NST	62	1,70	21,45	33	121	80	67	647	626	645	642,61	10,36	0,99	0,93	0,94	0,95	14,00	15,60	15,50	15,03	60	60	60	60,00
30	FRU	1X2-17	1	NST	88	1,90	24,38	38	113	71	57	739	583	634	652,06	7,41	0,89	0,94	0,92	0,92	19,80	14,00	15,90	16,57	56	56	56	56,00
33	FRI	1X2-18	1	NST	75	1,81	22,89	39	107	70	51	279	429	647	451,49	6,02	0,66	0,86	0,99	0,84	13,60	12,30	14,00	13,30	60	60	57	59,00
27	AD	1X2-19	1	NST	63	1,75	20,57	34	111	69	45	608	639	372	539,73	8,57	1,02	1,05	0,88	0,98	12,40	12,30	10,20	11,63	55	55	55	55,00
30	RT	1X2-20	1	NST	77	1,86	22,26	26	119	63	58	1765	1625	1227	1538,76	19,98	1,58	1,61	1,59	1,59	15,00	13,30	10,30	12,87	60	60	58	59,33

7. Raw Data (y) and (z)

MEASUREMENT 3 (Y)

Time X1 ↔ Y min	Initial	Condition	Dr	Question	Weight kg	Height m	BMI	Age years	RR mm Hg Syst.	RR mm Hg Diast.	HR puls/min	Flow 1 ml/min	Flow 2 ml/min	Flow 3 ml/min	Average ml/min	ml/ min/ Kg	Diameter 1 cm	Diameter 2 cm	Diameter 3 cm	Average cm	Velocity cm/s	Velocity cm/s	Velocity cm/s	Average cm/s	Angle 1 °	Angle 2 °	Angle 3 °	Average °
44	MJ	1Y1	2	MT	79	1,81	24,11	40	---	---	---	1267	520	915	900,65	11,40	1,33	1,05	1,38	1,25	15,20	10,00	10,20	11,80	55	55	55	55,00
44	RA	1Y2	2	MT	70	1,73	23,39	35	---	---	---	859	1083	1108	1016,61	14,52	1,27	1,34	1,35	1,32	11,30	12,80	12,90	12,33	56	56	56	56,00
42	HC	1Y3	2	MT	82	1,87	23,45	39	---	---	---	230	272	255	252,49	3,08	0,62	0,70	0,68	0,67	12,70	11,80	11,70	12,07	60	60	60	60,00
43	SCHH	1Y4	2	NST	78	1,88	22,07	39	---	---	---	1896	1603	1760	1753,83	22,47	1,66	1,44	1,50	1,53	14,60	16,40	16,60	15,87	60	60	60	60,00
44	PT	1Y5	2	NST	78	1,77	24,90	30	---	---	---	696	639	772	702,11	9,00	1,05	1,10	1,07	1,07	13,40	11,20	14,30	12,97	60	60	60	60,00
45	GCH	1Y6	2	NST	85	1,83	25,38	37	---	---	---	755	504	718	658,89	7,75	1,07	0,96	1,07	1,03	14,00	11,60	13,30	12,97	60	60	60	60,00
42	LG	1Y7	2	MT	84	1,83	25,08	32	---	---	---	848	1134	920	967,49	11,52	1,13	1,33	1,25	1,24	14,10	13,60	12,50	13,40	35	35	35	35,00
42	SJ	1Y8	2	MT	74	1,78	23,36	39	---	---	---	1047	1307	827	1127,28	15,23	1,14	1,24	1,19	1,19	17,10	20,80	12,40	16,77	61	61	61	61,00
43	KR	1Y9	2	MT	79	1,82	23,85	26	---	---	---	843	739	1096	892,50	11,30	0,82	0,85	1,04	0,90	26,60	21,70	21,50	23,27	59	59	59	59,00
44	RP	1Y10	1	MT	65	1,79	20,29	23	---	---	---	539	588	507	544,42	8,38	0,82	0,88	0,81	0,84	17,00	16,10	16,40	16,50	60	60	60	60,00
46	ALJ	1Y11	1	MT	75	1,72	25,35	34	---	---	---	652	688	558	632,82	8,44	1,04	1,00	0,91	0,98	12,80	14,60	14,30	13,90	51	51	51	51,00
40	SCHHE	1Y12	1	MT	70	1,82	21,13	39	---	---	---	1277	1203	1143	1207,66	17,25	1,27	1,24	1,22	1,24	16,80	16,60	16,30	16,57	60	60	60	60,00
42	PH	1Y13	1	MT	73	1,78	23,04	36	---	---	---	903	888	891	893,99	12,25	1,20	1,17	1,23	1,20	13,30	13,77	12,50	13,19	59	59	58	58,67
44	PE	1Y14	2	NST	85	1,82	25,66	29	---	---	---	440	405	528	457,86	5,39	1,03	1,00	1,11	1,05	8,80	8,60	9,10	8,83	60	60	60	60,00
40	ALJÜ	1Y15	1	NST	75	1,84	22,15	38	---	---	---	467	630	681	592,74	7,90	0,92	1,06	1,02	1,00	11,70	11,90	13,90	12,50	57	57	57	57,00
41	HM	1Y16	1	NST	62	1,70	21,45	33	---	---	---	679	734	638	683,54	11,02	0,94	0,93	0,89	0,92	16,30	18,00	17,10	17,13	60	60	60	60,00
43	FRU	1Y17	1	NST	88	1,90	24,38	38	---	---	---	1336	1199	1303	1279,32	14,54	1,28	1,22	1,20	1,23	17,30	17,10	19,20	17,87	60	60	60	60,00
44	FRI	1Y18	1	NST	75	1,81	22,89	39	---	---	---	550	538	615	574,27	7,66	0,97	0,94	1,07	0,99	12,40	13,40	11,40	12,40	57	57	57	57,00
40	AD	1Y19	1	NST	63	1,75	20,57	34	---	---	---	534	324	483	450,41	7,15	1,02	0,85	1,05	0,97	10,90	9,80	9,30	10,00	58	58	60	58,67
41	RT	1Y20	1	NST	77	1,86	22,26	26	---	---	---	1783	1736	1610	1709,75	22,20	1,61	1,69	1,64	1,65	14,60	12,90	12,70	13,40	60	60	60	60,00

MEASUREMENT 4 (Z)

Time X1 ↔ Z min	Initial	Condition	Dr	Question	Weight kg	Height m	BMI	Age years	RR mm Hg Syst.	RR mm Hg Diast.	HR puls/min	Flow 1 ml/min	Flow 2 ml/min	Flow 3 ml/min	Average ml/min	ml/ min/ Kg	Diameter 1 cm	Diameter 2 cm	Diameter 3 cm	Average cm	Velocity cm/s	Velocity cm/s	Velocity cm/s	Average cm/s	Angle 1 °	Angle 2 °	Angle 3 °	Average °
105	MJ	1Z1	2	MT	79	1,81	24,11	40	125	81	46	674	1170	661	835,44	10,58	1,07	1,27	1,14	1,16	12,50	15,40	10,80	12,90	60	59	59	59,33
104	RA	1Z2	2	MT	70	1,73	23,39	35	113	80	63	863	1089	996	982,42	14,03	1,22	1,28	1,29	1,26	12,30	14,10	12,70	13,03	60	60	60	60,00
101	HC	1Z3	2	MT	82	1,87	23,45	39	118	50	45	688	879	1363	976,83	11,91	0,99	1,09	1,16	1,08	14,90	15,70	21,50	17,37	55	60	60	58,33
102	SCHH	1Z4	2	NST	78	1,88	22,07	39	111	68	44	490	1221	884	864,73	11,09	1,03	1,38	1,17	1,19	9,80	13,60	13,70	12,37	60	60	60	60,00
103	PT	1Z5	2	NST	78	1,77	24,90	30	123	90	77	516	1125	946	862,44	11,06	0,94	1,21	1,11	1,09	12,40	16,30	16,30	15,00	60	60	60	60,00
106	GCH	1Z6	2	NST	85	1,83	25,38	37	121	78	61	499	505	729	577,92	6,80	0,96	0,97	1,04	0,99	11,50	11,40	14,30	12,40	60	60	60	60,00
99	LG	1Z7	2	MT	84	1,83	25,08	32	139	82	57	580	610	1536	908,48	10,82	1,00	1,08	1,45	1,18	12,30	11,10	15,50	12,97	25	25	25	25,00
101	SJ	1Z8	2	MT	74	1,78	23,36	39	103	71	58	785	975	589	782,98	10,58	1,03	1,13	0,91	1,02	15,70	16,20	15,10	15,67	62	62	62	62,00
104	KR	1Z9	2	MT	79	1,82	23,85	26	124	86	54	226	588	715	509,27	6,45	0,51	0,73	0,79	0,68	18,40	23,40	24,30	22,03	60	59	59	59,33
106	RP	1Z10	1	MT	65	1,79	20,29	23	125	79	47	323	300	417	346,67	5,33	0,82	0,81	0,96	0,86	10,20	9,70	9,60	9,83	58	58	58	58,00
106	ALJ	1Z11	1	MT	75	1,72	25,35	34	107	69	45	428	647	562	545,71	7,28	0,87	0,98	0,91	0,92	12,00	14,30	14,40	13,57	60	60	60	60,00
101	SCHHE	1Z12	1	MT	70	1,82	21,13	39	107	58	81	1235	879	1160	1091,57	15,59	1,28	1,09	1,20	1,19	16,00	15,70	17,10	16,27	60	60	60	60,00
103	PH	1Z13	1	MT	73	1,78	23,04	36	114	76	62	508	442	660	536,38	7,35	0,90	0,88	1,03	0,94	13,30	12,10	13,20	12,87	57	57	57	57,00
104	PE	1Z14	2	NST	85	1,82	25,66	29	120	85	50	395	425	598	472,89	5,56	0,96	0,98	1,11	1,02	9,10	9,40	10,30	9,60	59	60	60	59,67
99	ALJÜ	1Z15	1	NST	75	1,84	22,15	38	118	84	62	504	585	639	575,78	7,68	0,96	1,03	1,10	1,03	11,60	11,70	11,20	11,50	57	57	57	57,00
101	HM	1Z16	1	NST	62	1,70	21,45	33	106	79	65	584	519	510	537,73	8,67	0,83	0,84	0,81	0,83	18,00	15,60	16,50	16,70	58	58	60	58,67
105	FRU	1Z17	1	NST	88	1,90	24,38	38	117	74	54	735	1098	1311	1048,17	11,91	1,01	1,00	1,07	1,03	15,30	23,30	24,30	20,97	60	60	60	60,00
100	FRI	1Z18	1	NST	75	1,81	22,89	39	110	65	47	617	682	459	585,86	7,81	0,88	0,92	0,79	0,86	16,90	17,10	15,60	16,53	57	57	57	57,00
102	AD	1Z19	1	NST	63	1,75	20,57	34	103	64	51	674	708	826	736,04	11,68	0,92	1,11	1,17	1,07	16,90	12,20	12,80	13,97	58	58	58	58,00
100	RT	1Z20	1	NST	77	1,86	22,26	26	107	65	52	1216	920	981	1039,00	13,49	1,42	1,26	1,28	1,32	12,80	12,30	12,70	12,60	55	60	60	58,33

8. Declaration of Consent

Hanno Halbeisen
Osteopath
Unterer Kirchweg 4
A - 6850 Dornbirn

0043-5572-25535
hanno.halbeisen@cable.vol.at

Declaration of Consent

Name:

First Name:

Date of Birth:

- I declare my consent to take part in the osteopathic study „Mobilisation of the lesser omentum“
- I was informed about the background, purpose and possible adverse reactions.
- I agree, that relevant examination data of this research are passed on to Hanno Halbeisen for his master thesis.

All data are treated as confidental according to the data protection act.
Before evaluation data are being anonymised.

Place, Date:_____Signature:_____

9. Explanation for the Participants

Dear participants of the study,

in the course of my master study in osteopathy at the Donau Universität in Krems, I perform an osteopathic study, or more specifically a fundamental research.

Therefore, first I want to inform you about the procedure of the study:

During these examinations, an ultrasound examination of your portal vein is performed by a medical specialist while you are lying on your left side. This examination is absolutely pain free and lasts only a short time.
After half an hour, during which you can pause in a resting room, that is reserved for you, another ultrasound examination will be performed.
Then, you will be treated with a manual technique by a trained and experienced osteopath. The administered technique will neither be invasive nor painful.
After the intervention, a third ultrasound examination will be conducted by the medical specialist.
In order to find out, if the effect of the technique continues for a longer time, another examination will be performed one hour later. During this hour, you should wait sitting in the resting room.
By a comparison of the initial data and the data after the osteopathic intervention, we can evaluate changes in the portal blood flow.

Although you don’t have to expect any adverse reactions, you might experience side effects e.g. due to nervousness or tension during the examinations. In this case, you can claim the termination of the examination at any time.

Thank you for your co-operation

Hanno Halbeisen