The Liver and the Gallbladder

Luc Peeters & Grégoire Lason
The Liver and the Gallbladder

Luc Peeters & Grégoire Lason

All rights reserved. Osteo 2000 bvba © 2013. No part of this e-book may be reproduced or made public by printing, photocopying, microfilming, or by any means without the prior written permission of the publisher.

Contact: Osteo 2000, Kleindokkaai 3-5, B – 9000 Ghent, Belgium

Mail: info@osteopathie.eu

Web: http://osteopedia.iao.be and www.osteopathie.eu

Tel: +32 9 233 04 03 - Fax: +32 55 70 00 74

ISBN: 9789074400312

The International Academy of Osteopathy – I.A.O.
Content

Content ........................................................................................................................................... 3

1. Introduction ............................................................................................................................... 6

2. Anatomy .................................................................................................................................... 7

   2.1. Position and important Anatomy ...................................................................................... 7

      2.1.1. Upper or diaphragmatic Surface ............................................................................. 8
      2.1.2. The inferior Surface ................................................................................................. 9
      2.1.3. The posterior Surface ............................................................................................... 11
      2.1.4. The anterior Edge ..................................................................................................... 12

   2.2. Physiological Fixations of the Liver .................................................................................... 15

   2.3. Blood Supply of the Liver ................................................................................................. 17

3. Mobility ..................................................................................................................................... 24

4. Innervation ............................................................................................................................... 26

5. Physiology ................................................................................................................................ 29

   5.1. Functions of the Liver ...................................................................................................... 29

   5.2. Bile .................................................................................................................................... 33

   5.3. Cholesterol ....................................................................................................................... 33

6. Pathology, Patient History and Examination ........................................................................... 37

   6.1. Liver Cirrhosis .................................................................................................................. 37

   6.2. Jaundice ............................................................................................................................ 39

   6.3. Liver Cancer .................................................................................................................... 39

   6.4. Metabolic Syndrome ....................................................................................................... 41

   6.5. Fatty Liver (Steatosis-Lipidosis) ..................................................................................... 46

   6.6. Hepatitis ........................................................................................................................... 48

      6.6.1. Hepatitis A ............................................................................................................... 48

      6.6.3. Hepatitis C ............................................................................................................... 49

      6.6.4. Hepatitis D ............................................................................................................... 49

      6.6.5. Hepatitis E ............................................................................................................... 49

      6.6.6. Hepatitis G ............................................................................................................... 49

      6.6.7. Auto-Immune Hepatitis ........................................................................................... 50

   6.7. Portal Hypertension .......................................................................................................... 50

   6.8. Gallstones ........................................................................................................................ 52

   6.9. Gallbladder Inflammation ............................................................................................... 54

   6.10. Wilson’s Disease ........................................................................................................... 54

   6.11. Functional Liver Congestion .......................................................................................... 55

   6.12. Liver Ptosis ..................................................................................................................... 57

7. Clinical Examination ................................................................................................................ 58

   7.1. Palpation .......................................................................................................................... 58

      7.1.1. Palpation of the Liver and surrounding Structures ................................................... 58

      7.1.2. Palpation of the Origin of the right Phrenicocolic Ligament .................................... 60
1. Introduction

The liver is an organ that is often found to be in a state of dysfunction by the osteopath. Typical findings when palpating the liver are somatic dysfunction at the level of T₆-₉, a high position of the diaphragm and a clearly increased resistance when palpating the liver.

The liver is the port for all dietary substances into the body.

Liver dysfunctions often result in complaints that are not directly attributed to the liver but are actually associated.

Such liver dysfunctions have a negative effect upon the digestion, the energy balance, the circulation and various hormonal functions.

Osteopathic treatment of liver dysfunctions involves not only the organ itself and it’s related segment but also other organs and structures due to the essential nature of these relationships – in a mechanical, vascular, neurological and metabolic sense.

Readers who are not familiar with the osteopathic visceral approach should consult chapter 12 at the end of this e-book.
2. Anatomy

2.1. Position and important Anatomy
The shape of the liver can be compared to the upper half of an egg placed on its side with the larger, right aspect found under the diaphragm and angled to anterior.

The colour is reddish-brown, the consistency is strong and solid and a thin and fibrous capsule encloses the liver parenchyma: the tunica fibrosa hepatis or Glisson’s capsule.

**The dimensions of the liver in an adult are** *(Figure 2)*:

- Length 28 cm.
- Width 15 cm.
- Thickness 8 cm (right section).

In a living person and when filled with blood the organ weighs 2300 to 2400 grams *(Figure 2)*. The liver can contain 500 to 900 grams of blood and the internal temperature is usually higher than that of the surrounding organs - in certain hepatic veins the temperature can be elevated to 40°C.

The liver fills the entire right hypochondriac region, above the transverse mesocolon.

From under the right dome of the diaphragm, the thinner left section of the liver extends over the mid-line by passing anterior to the oesophagus, makes contact with the left dome of the diaphragm and can extend as far as the spleen.

The posterior surface attaches to the v. cava inferior, hangs over the pylo-duodenal junction and the right portion of the pancreas.

The liver is both a thoracic and abdominal organ. The superior projection gives a concave line to superior that extends between the right 5th intercostal space and the left 6th intercostal space *(Figure 9)*.

Inferiorly, the anterior border of the liver faces superior and internal along the costal margin – where it is palpable during a full inhalation – and then crosses the epigastric fossa along a line between the right 9th costal cartilage and the left 8th costal cartilage.

The vertebral relationships are posterior and extend between the spinal levels of T8-T9, superiorly, and T12, inferiorly, – on the right side *(Figure 9)*.

The topographical location of the gallbladder *(Figure 3, 4, 5, 7 and 8)* is the junction between the right mid-clavicular line and the horizontal line at navel height – or where the rectus abdominis m. crosses the 10th rib.
The ductus choledocus (cystic duct) is found posterior and medial.

2.1.1. Upper or diaphragmatic Surface

(Figure 1)

This surface is smooth and convex in an anterior posterior direction.

It is broad on the right and narrow on the left.

The liver is covered by the visceral peritoneum and, at the junction of the right 2/3 and the left 1/3, has the insertion of the falciform ligament. This ligament is a sagittal peritoneal fold that joins the convex superior surface of the liver to the diaphragm and the anterior wall. Posteriorly it extends into the coronary ligament thus forming a “T”, where the vertical section is the falciform ligament and the horizontal section is the coronary ligament.

Between both structures is the teres ligament, a remnant of the umbilical vein, that attaches the liver to the internal aspect of the navel.

Figure 1 - Upper or diaphragmatic surface of the liver
2.1.2. The inferior Surface

The inferior surface of the liver is facing obliquely inferior and anterior and is triangular in shape with the large base to the right.

The inferior surface has a group of fissures in the shape of an “H” formed by:

- **A transverse fissure**: the hepatic port (the infra-hepatic pedicle divides into the portal vein, the hepatic artery and the biliary ducts).

- **The right fissure or fossa vesicae felleae** is a wide fissure that provides the surface for the gallbladder.

- **The left fissure or fossa lig. teretis or incisura umbilicale**, is narrow and deep. The left sagittal groove contains remnant foetal vessels: anterior the teres ligament: a remnant of umbilical veins, posterior the lig. venosum: a remnant of the ductus venosus.
3. Mobility
The liver passively follows the motions of the diaphragm (Figure 16).

In the frontal plane

The motion of the diaphragm during inhalation is from superior to inferior and posterior to anterior.

The central tendon of the diaphragm descends less than the lateral parts.

The liver follows the diaphragm motion: in other words during inhalation (beginning phase) the liver will descend as a whole.

In the later phase of the motion the right lobe of the liver will descend further than the left lobe (due to tension of the central tendon). Therefore the liver makes a right sidebending around an antero-posterior axis that runs through the left triangular ligament.

The motion of the inferior ribs during inhalation is to lateral and superior. The liver will move to medial during the inhalation.

In the sagittal plane

At the end of the inhalation the liver rotates in the sagittal plane to anterior. The antero-inferior aspect of the liver therefore moves to inferior and posterior.

The axis of motion is transverse – it runs between both triangular ligaments. This axis is referred to as the bi-triangular axis.

In the horizontal plane

During inhalation a limited rotation of the liver also occurs: the external border moves from posterior to anterior and from right to left.

Motions during exhalation occur in the opposite directions.
Figure 16 - Mobility of the liver during inhalation
4. Innervation


The autonomic innervation of the liver is both parasympathetic (from occiput-atlas-axis region) via the vagus nerve and sympathetic (segments T6-9).

The liver function is determined by substrate concentrations in the blood, circulating hormonal levels, the biomatrix and the neurovegetative system.

Stimulation of the sympathetic system increases the delivery of glucose, urate and lactate. It reduces the ketogenesis, the intake of ammonia and the flow of gall. An overflow of noradrenalin in the hepatic vein will also result. Sympathetic stimulation decreases the blood flow in the liver and closes the sinusoids in the liver tissue.

Parasympathetic stimulation results in re-opening of the sinusoids in the liver.

The actions of the sympathetic nervous system are modulated by the hormones glucagon, insulin, adrenalin, noradrenalin, vasopressin and angiotensin.

For the osteopath this means that during treatment of the liver attention should be given not only to the neurological relationships but also to related hormone producing organs such as the adrenal glands, the pancreas and the kidneys as well as addressing diet. Treatment of the liver only is, at best, short-sighted.

Local stimulation (what osteopaths probably do during visceral techniques for the liver and lesser omentum region) causes an increased glucose and lactate output + a haemodynamic action. The liver is the only organ that produces glucose for energy to the muscles. Logical that liver congestion or liver conditions play an important role in muscular weakness.

Complaints in the musculoskeletal system resulting from over-use or fatigue can be significantly influenced by liver dysfunctions.

Electrical stimulation of the ventromedial hypothalamus nucleus (VMH) (sympathetic) causes glycogenolysis in the liver while an electrical stimulation of the lateral hypothalamic nucleus (LH) (vagus nerve, parasympathetic) causes glycogenesis.

This glycolysis-glycogenesis mechanism is also under modulation by blood insulin levels.

The sympathetic nervous system also functions as a regulating centre for lipolysis in fatty tissue (adipose tissue) via the sympathetic innervation of brown adipose tissue. Both generation and lysis of the fatty tissue is stimulated by sympathetic action. This occurs only in the brown adipose, not in the white adipose.

Treatment of adipose via stimulation of the sympathetic system (via osteopathic treatment and sports activity) must be combined with decreased fat intake.
The parasympathetic system plays no part in the metabolism of fatty tissue.

Stimulation of both sympathetic and parasympathetic influences will increase the rate of liver regeneration after, for example, resection of liver tissue.

The afferent supply is via the vagus nerve: receptors in the liver detect changes in the hepatocellular energy balance and communicate this information to the hunger centre in the brain via the vagus nerve (Langhans 1998).

Via baroreceptors in the portal vein and in the liver itself, information concerning the pressure gradient is sent to the central nervous system.

Even manual pressure upon the gallbladder causes an afferent flow and most likely results in a regularising effect of the blood pressure.

The contractility of the liver involves the alteration of the diameter of hepatic arteries, under sympathetic influence.

Recently, a special contractive type of endothelial cell was identified in the sinusoids of the liver. They are thought to be responsible for regulating the local microcirculation. The greater the degree of fibrotic changes to the liver, the poorer this system functions.

Referred pain from the liver and gallbladder (Figure 17) is to the right shoulder and neck.

The region between the navel, xyphoid process and the 10th rib is also a common area of referred pain.

![Referred pain liver or gallbladder](image-url)
**Visceral pain**

Visceral pain is the most frequent reason for medical consultations.

Somatic and visceral pain function via different mechanisms.

We receive little sensory experience from the visceral system: only pain and discomfort are reported. A feeling of filling is even easily associated with pain.

All forms of visceral pain also result in pain in referred structures. This is known as “referred hyperalgesia”. Sometimes even only the “referred hyperalgesia” occurs (Cervero 1995, Cervero & Laird 2009, Hobson & Aziz 2003, Mayer & Gebhart 1994).

This is one of the most common reasons why osteopaths should examine and treat the organs.

It is not the visceral disease, which is treated, but the irritating factors (inflammation, ischemia, spasm, traction, overstretch).

The representation of the viscera in the central nervous system is poor. However, visceral nociception has been demonstrated for some organs, especially the hollow organs. Most of these receptors remain inactive and only become active in case of:

- Serious trauma.
- Inflammation.
- Ischemia.
- Muscular spasm.
- Traction.
- Stretch of a hollow organ.

The number of these nociceptors is small but once the impulse registers in the spinal cord more numerous secondary neurons become stimulated with divergence in the central nervous system.

Such divergent input can activate different systems: sensory, motor and autonomic. This goes some way to explaining the typical visceral pain: diffuse, referred and with maintained autonomic and motor activity.

The osteopath must be aware that the location of the pain is not always the origin or the area actually to be treated.

Pharmacological segmental blocks can play a role in managing this mechanical pain and the osteopath can contribute by way of influencing the somatic part of the segment.
5. Physiology


5.1. Functions of the Liver

Metabolic functions (Walther 2003)

Synthesis

The liver produces certain proteins and excretes these into the blood:

- **Haemostatic and fibrinolytic elements:**
  - Almost all proteins are involved in the coagulation process.
  - The inhibitors of the coagulation.
  - Fibrinolysis stimulating proteins.
  - Fibrinolysis inhibiting proteins.

- **“Carrier” proteins:**
  - Albumin, carrier of thyroid and other hormones, most importantly the fat-soluble hormones, fatty acids, bilirubin, medications and Ca^{2+} and regulator of the osmotic pressure.
  - Ceruloplasmin, carrier of copper.
  - Transcortin, carrier of cortisol, aldosterone and progesterone.
  - Haptoglobin, carrier of free haemoglobin excreted by the erythrocytes.
  - Hemopexin, carrier of haemoglobin.
  - IGF bonding protein, carrier of the insulin-like growth factor 1.
  - Retinol bonding protein, carrier of retinol.
  - Sex hormone bonding globulin, carrier of testosterone and oestradiol.
  - Thyroxine bonding globulin, carrier of the thyroid hormone thyroxine (T4) and triiodothyronin (T3).
  - Transthyretin, carrier of the thyroid hormone thyroxine (T4).
  - Transferrin, carrier of iron ions.
  - Vitamin D bonding proteins, carrier of vitamin D.

- **Hormones:**
  - Insulin-like growth factor 1, hormone that plays an important role in the growth of the child and that remains active in the adult with an anabolic effect.
  - Thrombopoietin, hormone that plays a role in the production of thrombocytes in bone marrow.
• **Prehormones:**
  o Angiotensinogen, which can remodel to become angiotensin (vasoconstrictor and aldosterone releasing): increases blood pressure.
  o Apolipoproteins: fat bonding proteins, which transport fat through the blood stream.

The liver plays an important role in the carbohydrate mechanisms:

• Gluconeogenesis (synthesis of glucose from certain amino acids, lactic acid or glycerine).

• Glycogenolysis (metabolism of glycogen into glucose) stimulated by muscle activity, hunger or fever.

• Glycogenesis (formation of glycogen from glucose) (muscle tissue can also do this).

• The primary source of dietary carbohydrates is vegetable starches. This is supplemented by a small amount of glycogen from animal sources, disaccharides such as sucrose (refined sugar) and lactose (milk). These products are digested in the intestines and become monosaccharides, which are transported to the liver. The liver turns them into glucose.

• **Glucose can undergo three possible transformations:**
  o It can be catabolised into adenosine triphosphate (ATP) – energy delivery. This occurs in the majority of body tissue (most notably brain, muscle and kidneys).

  o It can be stored as glycogen in muscle (approx. 400 g) and liver (approx. 100 g). The alteration of glycogen into glucose occurs under the influence of the hormone glucagon (produced in the pancreas, target organ: liver), the hormone adrenalin (produced in the adrenals, target organ: muscles) and the hormone insulin (produced in the pancreas, target organs: liver and muscles).

  o It can be turned into fatty acids and stored as triglycerides in the fatty tissue.
The regulation of the carbohydrate mechanism in the body is of exceptional importance:

- A 50% decrease in the blood sugar levels leads to dizziness, nausea and possible lost consciousness.
- The brain, spinal cord and retina require a constant supply of glucose. These tissues cannot stock glycogen. A shortage of glucose rapidly results in damage.
- The blood-brain barrier (gliacells) turns glucose into lactic acid, which the brain uses for energy delivery. Starvation damages the brain and nervous system.
6. Pathology, Patient History and Examination

6.1. Liver Cirrhosis
Liver cirrhosis (Figure 18) is a fibrosis of the liver tissue where the degeneration of the cells outpaces the regeneration.

The liver becomes lumpy and then a shrivelled mass. When palpated the cirrhotic liver is hard, irregular and, during the end stadium, shrunken.

In cases of liver cirrhosis a congestion of the portacaval anastomoses will be present due to the venous blood flowing via the path of least resistance.

The portacaval anastomoses are found:

- At the cardia (Figure 19).
- At the rectum (haemorrhoids).
- At the umbilical veins (caput medusae) (Figure 19).
- At the spleen (splenic swelling).

Due to altered osmotic pressures and reduced hormonal production in the liver, ascites and oedema of the feet and ankles develop during a later stadium.

Due to these hormonal dysfunctions in the liver, spider naevi (spider web-like red skin colourisation) can be seen on the face and body.

The metabolism of bilirubin also functions poorly resulting in jaundice from the collection of unmetabolised red blood cells.

Increased levels of ammonia lead to encephalopathy and disruption of brain function with drowsiness, confusion and even aggression.

Male breast development is also a sign of liver degeneration.

Liver cirrhosis can be inactive and present little evolution and then at once begin progressing to an end stadium where the liver function fails totally leading to a life-threatening situation.

The cause of liver cirrhosis is multi-factorial. Alcohol misuse is a frequently associated factor. However, viral hepatitis is the leading cause of liver cirrhosis worldwide. In 10% of the patients suffering from liver cirrhosis the origin remains undiagnosed.
Figure 18 - Liver cirrhosis

The storage capacity for carbohydrates is altered so that the blood glucose level is abnormal. Fainting is a common result of this alteration.

Blood clotting is also altered leading to frequent and easy bruising and episodes of bleeding, most commonly from the nose.

Due to damage of the Kupfer cells (usually from alcohol) there will also be a decrease in immune function often with unexpected autoimmune reactions.

Treatment must be directed at the cause and the hepatitis or the alcohol misuse must be addressed effectively.

Liver cirrhosis never fully repairs.

Figure 19 - Caput medusa and cardio-oesophageal varices
7. Clinical Examination

7.1. Palpation

7.1.1. Palpation of the Liver and surrounding Structures

The easiest way to palpate the liver and to test for mobility is with the patient sitting.

The osteopath stands behind the patient and places the hands under the right dome of the diaphragm.

The right liver is palpated:

- Normal: the liver tissue is not painful with palpation and feels soft and smooth.
- Pathological: small and hard + ascites = liver cirrhosis (+ associated symptoms).
- Pathological: painful = inflammation.
- Pathological: not painful but irregular: possible tumours or other trophic changes.
- Pathological: swollen in the posterior section = venous congestion.
- Pathological: completely swollen, “fatty” consistency together with a positive palpation of the gallbladder = “dietary” liver congestion (fatty deposits).

Palpation of the left liver (left of the mid-line and anterior):

- Normal: the liver tissue feels harder than the right side but is not painful nor swollen.
- Pathological: painful and sometimes swollen: toxicity.

Palpation of the anterior edge of the liver:

- Normal: relatively sharp edge and not painful.
- Pathological: the sharp edge is less defined in cases of liver congestion.

Palpation of the lesser omentum (right of the mid-line and posterior):

- Normal: clear resistance to palpation but not painful.
- Pathological: too much resistance but not painful: retraction.
- Pathological: very painful: inflammation or spasm of the biliary ducts.

Palpation of the gallbladder (found against the inferior surface of the right liver):

- Normal: lightly sensitive especially before meals.
- Pathological: very painful and distended (together with painful palpation of the sphincter of Oddi and biliary ducts): congestion of the gallbladder and the biliary ducts.
• Pathological: very painful and swollen: possible emotional problem.

**Palpation in the direction of the right triangular ligament:** very difficult.

• Normal not palpable.
• Pathological: painful region but without the structure itself being palpable.
• Usually a consequence of liver congestion.

**Palpation in the direction of the left triangular ligament:** found at the left tip of the left liver.

• Normal: no pain and difficult to palpate.
• Pathological: painful, usually a consequence of liver congestion or ptosis.

*Video 1 - Palpation of the liver and surrounding structures*
8. Osteopathic Techniques

8.1. General Remarks related to Mobilisation of the Ribs

A thorax lacking elasticity will decrease the cardiac outflow. Not only in rest but also during exertion. The heart volume decreases and the lungs demonstrate decreased vital capacity. During exertion the heart rhythm increases but this is not sufficient to compensate for the decreased heart volume. The blood pressure quickly rises. The gastric pressure also increases significantly (Miller et al. 2002).

Therefore good mobility of the thorax is essential for an optimal condition of the heart and general circulation.

Loss of elasticity in the thorax will also reduce the effect of the diaphragm pump function.

Via the inferior ribs a mechanical effect is also achieved for the segments T6-9.

8.2. Mobilisation of the Ribs in the frontal Plane

The patient is sitting with the thorax erect.

Using both hands the osteopath contacts the inferior ribs laterally and lifts them to cranial during an inhalation from the patient and follows to caudal during the exhalation. While inhaling, the thoracic spine is helped into extension and while exhaling into flexion.

The restrictive components are mobilised.

Mobilisation to cranial is most effective during inhalation and mobilisation to caudal is most effective during exhalation.

Video 10 - Mobilisation of the ribs in the frontal plane
8.3. Mobilisation of the Ribs in the sagittal Plane
The patient is lying on the side and the osteopath places one hand posterior and one hand anterior upon the inferior ribs. The ribs are mobilised to superior (inhalation) and inferior (exhalation).

Care must be taken that the patient lays in the sagittal plane with the upper thorax in neutral (no rotation).

Mobilisation to cranial is most effective during inhalation and mobilisation to caudal is most effective during exhalation.

**Video 11 - Mobilisation of the ribs in the sagittal plane**

8.4. Mobilisation of the Ribs in the horizontal Plane
The patient is lying on the side with the lower leg straightened on the table and the upper leg flexed so that the patient is stable.

The osteopath mobilises the ribs to anterior and to posterior rotation.

Mobilisation to cranial is most effective during inhalation and mobilisation to caudal is most effective during exhalation.

**Video 12 - Mobilisation of the ribs in the horizontal plane**
10. About the Authors

Grégoire Lason
Gent (B), 21.11.54

Luc Peeters
Terhagen (B), 18.07.55

Both authors are holders of university degrees, namely the Master of Science in Osteopathy – University of Applied Sciences, and are very active with the promotion and academic structuring of osteopathy in Europe. In 1987 they began The International Academy of Osteopathy (IAO) and are, to this day, the joint-principals of this academy. The IAO is since several years the largest teaching institute for osteopathy in Europe. Both osteopaths are members of diverse professional organisations, including the American Academy of Osteopathy (AAO), the International Osteopathic Alliance (IOA), the World Osteopathic Health Organisation (WOHO), as part of their mission to improve osteopathic development.

This osteopathic encyclopaedia aims to demonstrate the concept that a proper osteopathic examination and treatment is based upon the integration of three systems: the musculoskeletal, visceral and craniosacral systems.
This e-book is a product of Osteo 2000 bvba.

If you are interested in publishing an e-book or if you have questions or suggestions, please contact us:

**Mail:** ebooks@osteopathie.eu

**Fax:** +32 55 70 00 74

**Tel:** +32 9 233 04 03

**Web Osteopedia:** [http://osteopedia.iao.be](http://osteopedia.iao.be)

**Web The International Academy of Osteopathy – IAO:** [http://www.osteopathie.eu](http://www.osteopathie.eu)