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Does osteopathy influence diabetes mellitus type II ?

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Motion is not life. Motion is a manifestation of life. The miracle of life expresses itself in motion and movement.

(Becker, 1997, 13)

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1. INTRODUCTION

In this work I will deal with the disease “diabetes mellitus“. This disease was only briefly mentioned in all my training, so I want to look closer into the field of physiology, pathophysiology and anatomy now. I chose the anatomy of the pancreas as an example. The entire anatomy that should be discussed with regard to diabetes mellitus would be too much – in fact endless.

The pancreas as such was not dealt with in my practical osteopathic training, so some questions arose.

How can I influence diabetes mellitus in a respectful way, and can osteopathy influence this disease at all? Can we change metabolism by improving the range of motion in the individual tissues and planes?

Ten diabetics (only type II) will be treated. Further ten diabetics will serve as a control group. The patients will be treated six times with intervals of three to four weeks. The measurement – HbA1c (long-term –blood-sugar control) is very exact and objective.

“Science, if anything, has taught us an increased respect for our body by deepening a sense of the wonder and mystery of its workings.”

Lin Yutang (Mc Catty, 1988,203)

Taking this as a motto I will commence my work.

Some introducing comments to the question: „ Does osteopathy influence diabetes mellitus type 2?“

Diabetes mellitus is a very complex disease. Several organs and systems are involved in metabolism. Liver, stomach, duodenum, pancreas, the whole gastrointestinal tract are responsible for good metabolism. The hormone system, blood flow, the unrestricted flow in lymph vessels, the powerful flow of liquor and the interchange of all these systems cause a functional metabolism.

If barriers, such as tensed ligaments of organs exist, or tension in tissue exists, for example in the Treitz'sche fascia which accompanies the whole intestinal tract in the dorsal part of the abdomen, quite a remarkable lack of motion would be the result. Lack of motion shows the consequence in the flow of blood, lymph, liquor. Assimilation is reduced if the flexura duodenojejunalis is tensed.

The thoracic diaphragm, the pump between thorax and abdomen, conditions a lot of motion. The free tension of the diaphragm muscles is necessary to unfold completely.

The chemical work at liver, duodenum and pancreas is restricted if the diaphragm is not relaxed. The diaphragm cannot move harmonically. The completely unfolding is needed for a perfect pump work.

These thoughts let me suppose that improvement of tension in tissues leads to more motion. A good motion in tissue results in a good metabolism. Therefore I suppose that diabetes mellitus, as a disease of metabolism, lets influence by osteopathic treatment.

To understand the mechanism of diabetes mellitus and the work of osteopathy, we have to go through anatomy, physiology and pathophysiology first.

2. BASIS

2.1. Anatomy

2.1.1. Topography:

Benninghoff describes the anatomy of the pancreas as follows:

“The pancreas is an organ weighing 70 to 90g. It is 14 to 20 cm long, 3 to 5 cm wide and 2 to 3 cm thick. It lies in the retroperitoneum at the level of the first and the second lumbar vertebrae, ascending diagonally from the right to the left towards the spleen.”
(Benninghoff 1994,115)

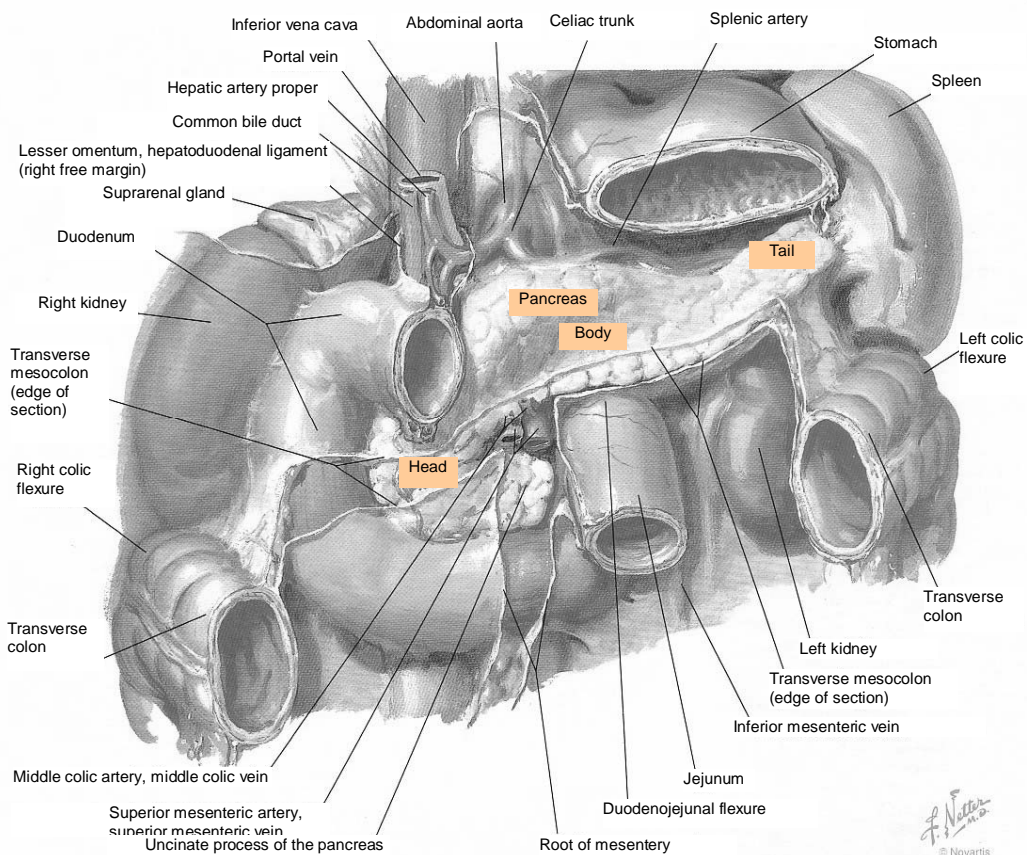


Fig. 1: Pancreas with surrounding organs

The pancreas consists of the head, the body and the tail.

“The head of the pancreas, caput pancreaticus, lies in the concave curve of the duodenum. A hook-shaped prolongation of the head, the uncinata process, is tucked behind the superior mesenteric vasa. These vessels lie at the posterior surface of the pancreas and pass forward in the pancreatic notch.

The body of the pancreas, corpus pancreaticus, lies at the level of the first or the second lumbar vertebra and passes from the right towards the left side of the body. The posterior surface of the body is continuous with the posterior abdominal wall, the anterior surface is covered by the peritoneum and lies in the posterior wall of the omental bursa.[...] The part of the body of the pancreas that lies anterior of the vertebral column and the aorta and bulges farthest into the omental bursa is called omental tuber.

The body gradually tapers to form the tail, cauda pancreatis, which extends as far as the spleen (splenorenal ligament).” (Benninghoff, 1994, 928)

“The main pancreatic duct, ductus pancreaticus major or Wirsung’s duct, is about 2 mm thick. It traverses the entire gland near the posterior surface. In most cases the pancreatic duct merges with the common bile duct and they jointly open into the duodenum at the major duodenal papilla (papilla of Vater)”. (Benninghoff, 1994, 928)

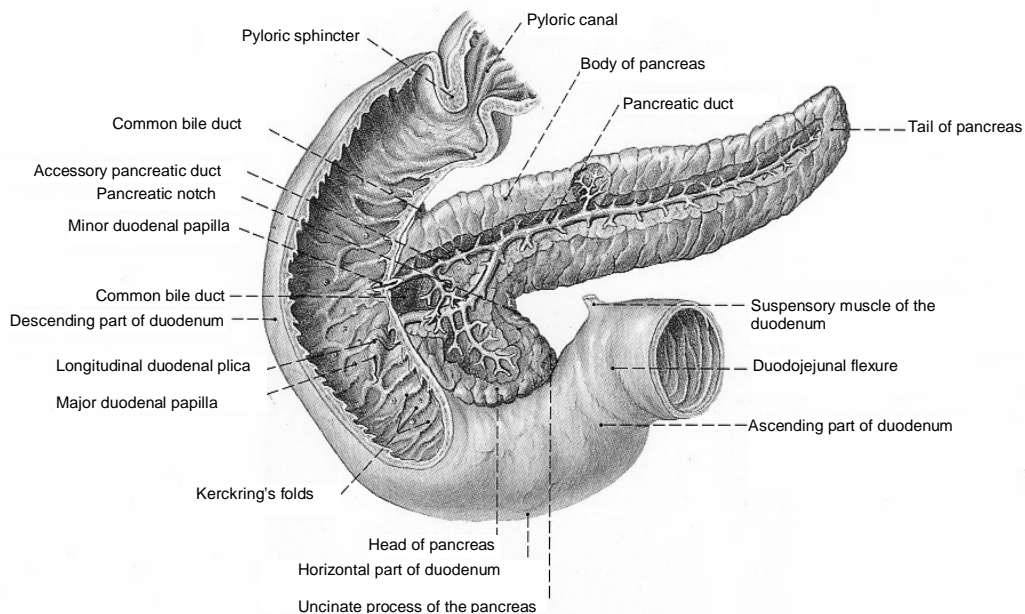


Fig. 2: Showing the pancreatic ducts

2.1.2. Blood supply:

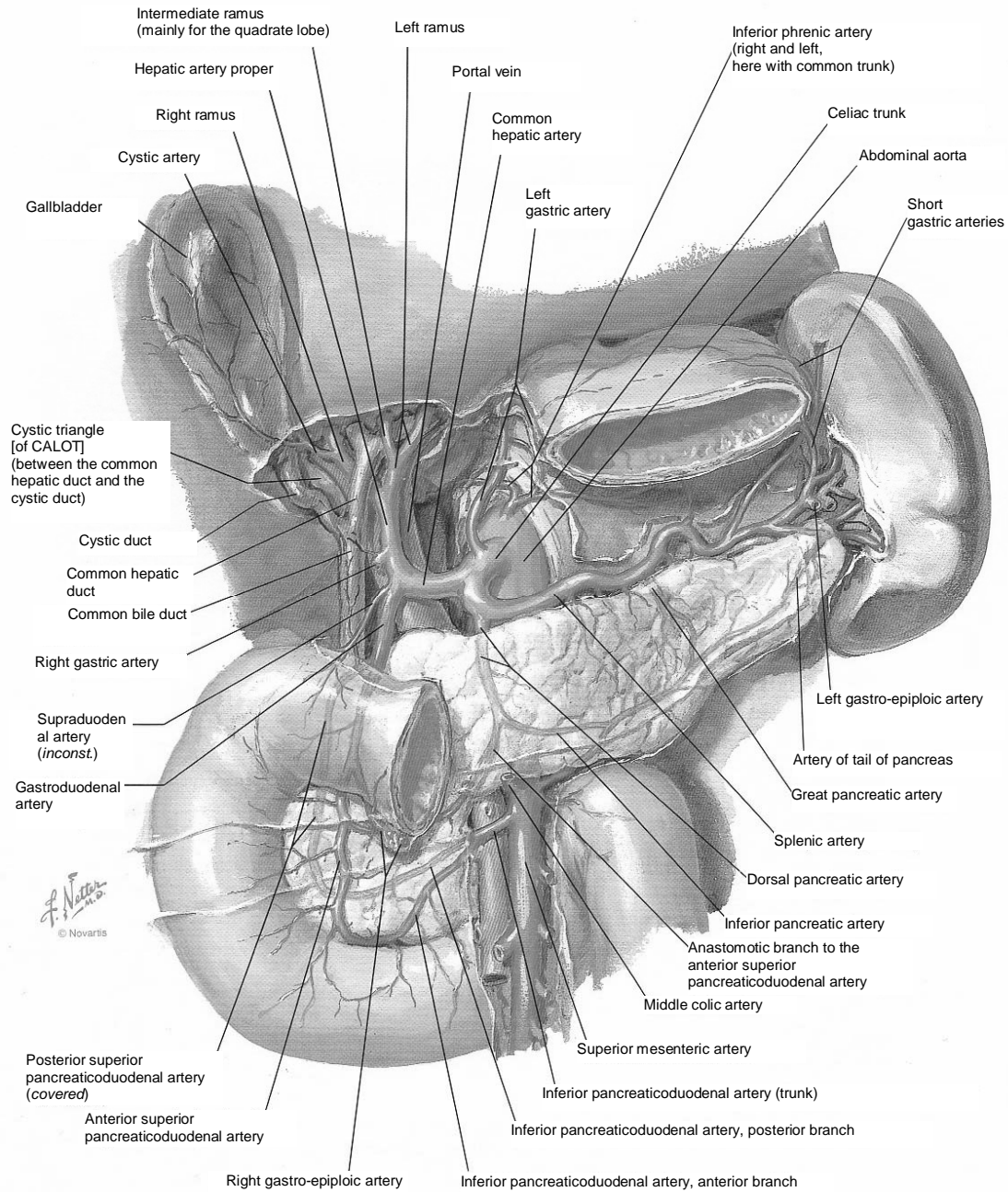


Fig. 3: Arteries of the pancreas, duodenum, spleen, liver – liver lifted

The arteries of the pancreas, duodenum, spleen and liver flow out of the celiac trunc.

The pancreas is supplied with blood by several arteries: Both the head of the pancreas and the duodenum are supplied by a double arterial circle, which consists of an anterior and a posterior superior pancreaticoduodenal artery (from the gastroduodenal artery) and an anterior and a posterior branch of the inferior pancreaticoduodenal artery (from the superior mesenteric artery).

The body and the tail receive vessels that arise directly from the splenic artery as great pancreatic artery or as pancreatic branches.

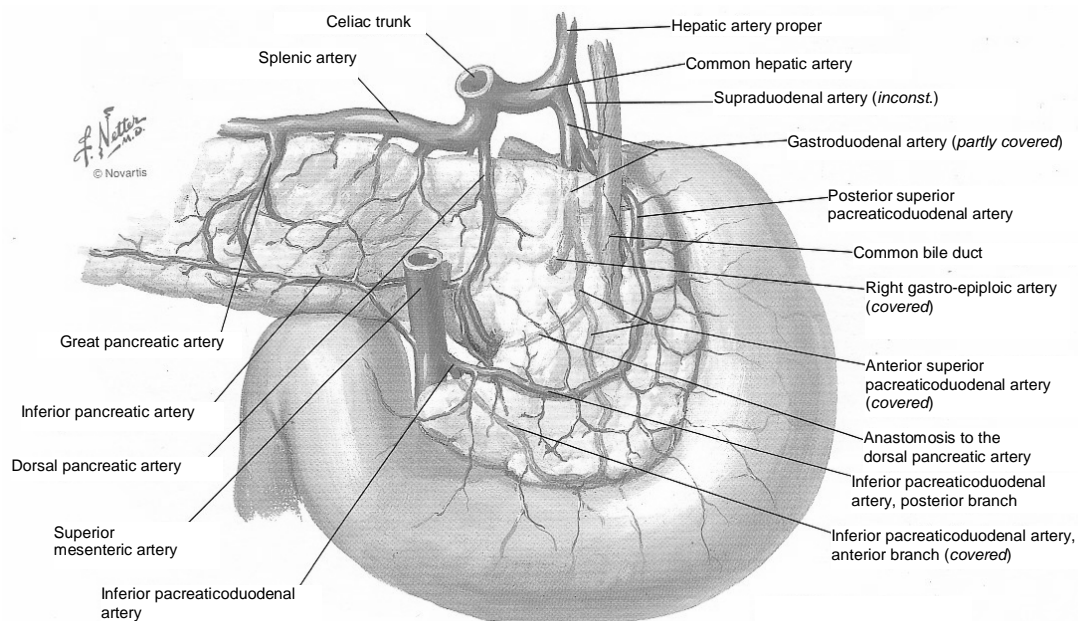


Fig. 4: Arteries of the head of the pancreas and duodenum

“According to Mc Catty, free circulation of the fluid is as important in physiology as unimpeded oil flow is in a car motor. If the oil passage is blocked, the machine will not work efficiently. With the body it is similar: nutrition, discharge of metabolic products, energy storage, conduction etc. – all that depends on a well functioning though complicated balance of the fluid circulation, so that the organism as a whole can maintain its homoeostatic integrity. If the blood flows freely and without impediment in the body, thus sufficiently supplying the tissue and nerves with blood, the tissues are able to function optimally. A. T. Still found that natural blood flow ensures health whereas local or general disturbances of the blood flow cause diseases. Therefore it is absolutely necessary to know the paths of the arterial and venous vessels in order to understand why certain disturbances occur, how these disturbances are related to other structures of the body and what is necessary to eliminate the impediments of the free flow. In osteopathy

the therapist tries to relieve the nerves, arteries, veins, etc. from pressure to restore the preconditions for a healthy physiology. He or she does so by correcting abnormal bone positions and enabling release of the strain on fasciae, ligaments, membranes, etc., partly with the help of the leverage of the bones, partly by directly communicating with the tissue.” (Liem, 1988,189)

The pancreas is drained by veins that open into the superior mesenteric and splenic veins and partly directly into the portal vein. The superior mesenteric and the splenic vein merge into the portal vein mostly behind the head of the pancreas.

2.1.3. The lymphatic supply

The lymphatics of the pancreas pass towards lymphatic nodes, which are situated along the arteries. The lymph flows from the head of the pancreas to the anterior and posterior pancreaticoduodenal lymphatic nodes. From there it reaches partly the hepatic lymphatic nodes and partly directly the mesenteric lymphatic nodes.

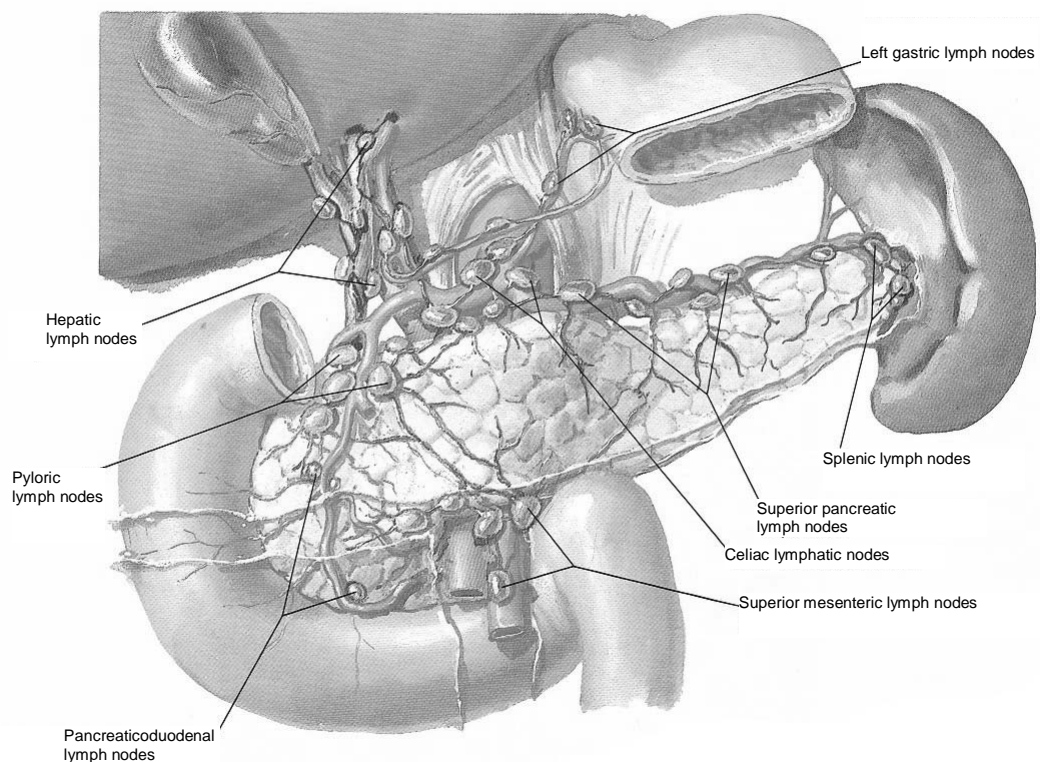


Fig. 5: Lymphatics and lymphatic nodes of the pancreas

The lymph from the body and tail of the pancreas is collected in nodes, which are situated along the splenic artery as superior pancreatic lymphatic nodes and along the inferior border of the organ as inferior pancreatic lymphatic nodes. From there it flows to the big group of lymphatic nodes at the junction of the celiac trunk and the superior mesenteric artery.

The drainage of the lymph system is also important for undisturbed physiological processes. Any blockage leads to a collection of metabolic products in the extracellular space.

“Factors responsible for lymphostasis:

Muscle activity: No or reduced muscle activity or hypertonic musculature [...]

The diaphragm: is the primary lymphatic pump. In the pulmonal inspiratory phase the lymph is pumped forward. In the expiratory phase the deep lymphatic plexus fills up.

Intestinal peristalsis: It acts as a pump for the main part of the lymph.

Arterial pulsation: The pulsation of the vessels acts as a lymphatic pump.

Innervation: The autonomic nervous system innervates the lymphatic vessels and causes rhythmic contractions at the level of the thoracic duct and other big lymphatic vessels.[...]

Tensions: of the connective tissues and fasciae

Thoracocervical diaphragm: The lymph returns in the left (and right) venous angle behind the sternoclavicular artery to the venous blood circulation. Therefore, the condition of the thoracocervical diaphragm, of the sternocleidomastoid muscle, the sternoclavicular artery, the upper thoracic vertebrae and the superior sternocostal arteries is of particular interest with regard to the lymph return.” (Liem, 1988, 197)

2.1.4. Innervation:

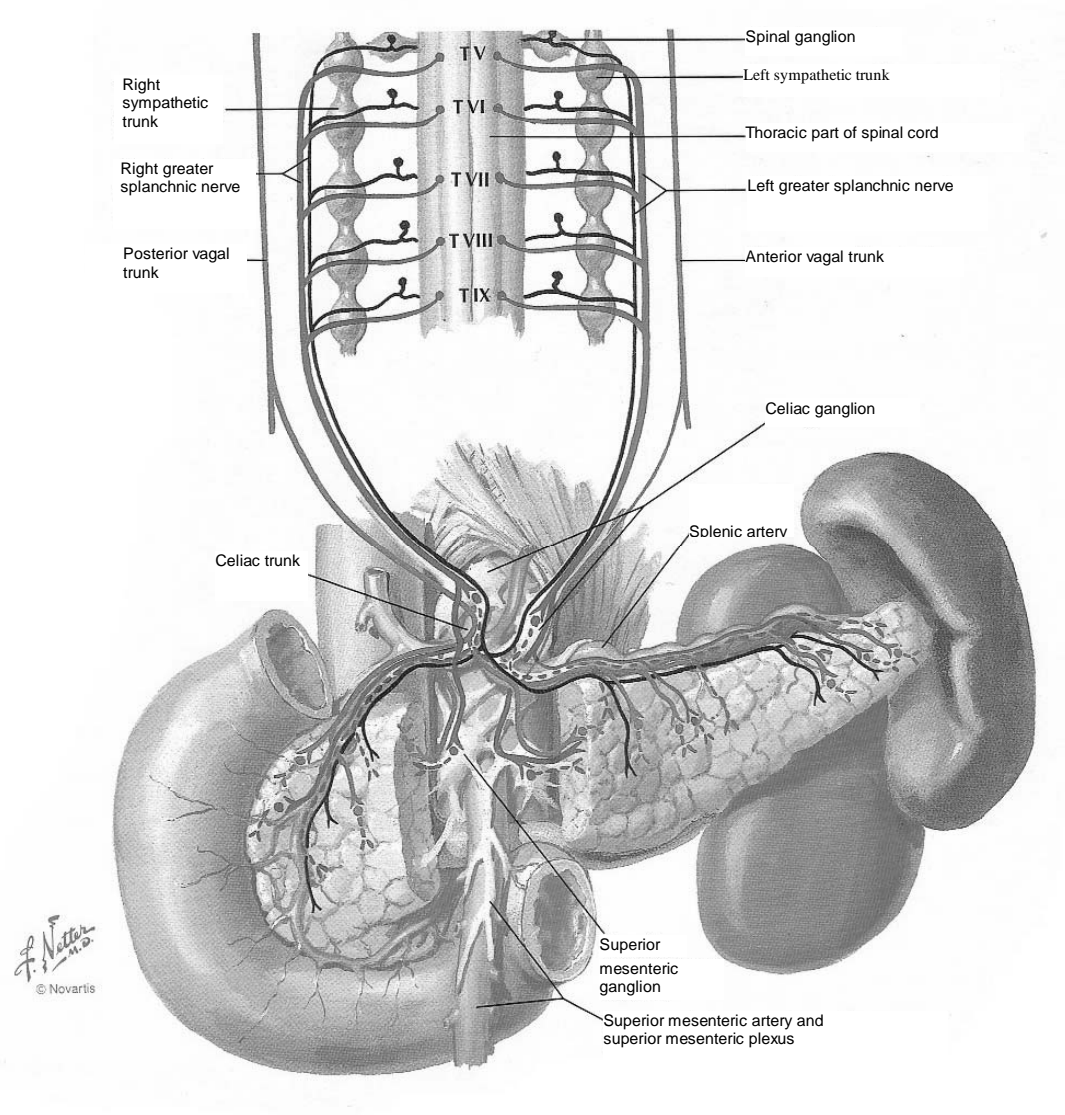


Fig. 6: Innervation of the pancreas

The pancreas is innervated by numerous autonomic nerve fibres. Some of the sympathetic fibres reach the organ through dense plexuses, which arise from the celiac ganglion and run along the vessels, another few fibres pass directly from the ganglion to the gland.

As shown in Fig.6, the sympathetic fibres emerge from the thoracic vertebral segments 5 to 9. This is important for the structural part of osteopathic evaluation and treatment.

Further neural supply is provided by parasympathetic fibres from the celiac trunk of the vagus nerve and from the gastric branches of the vagus.

This leads us to the examination of the origin of the vagus nerve and, particularly, to the evaluation of the exit orifice at the cranium. The jugular foramen (exit orifice) is situated in the occiput near the foramen magnum. Dysfunctions can be caused by compression of the temporal bone upon the occiput (the temporomandibular joint, for example, may be the initial cause), dysbalances of the membranes (falx, tentorium), interosseous tensions of the occiput, etc.

2.1.5. Associated organs and tissue structures

head of the pancreas:

- duodenum
- anterior surface: peritoneum, transverse mesocolon, omental bursa, coils of the jejunum
- posterior surface: pancreaticoduodenal artery, common bile duct, vessels of the right kidney, inferior vena cava, aorta

body of the pancreas:

- anterior surface: posterior surface of the stomach, omental bursa
- posterior surface: Treitz's fascia, aorta, superior mesenteric artery, left crus of the thoracic diaphragm, splenic vein, suprarenal gland, left kidney with vessels, fatty capsule of the kidney

- inferior surface: transverse mesocolon, duodenojejunal flexure, coils of the jejunum
- superior border: celiac trunk - hepatic and splenic artery
- anterior inferior border: divergence of the two layers of the transverse mesocolon
- inferior border: superior mesenteric artery

tail of the pancreas:

- splenorenal ligament , spleen

2.1.6. Fixations of the pancreas

- connections duodenum – gall-bladder
- exit of the pancreatic ducts in the duodenum (sphincter Oddi)
- vascular anchorage, which attaches it to the gastroduodenal, splenic, superior mesenteric arteries and to the splenic, superior mesenteric and portal veins.
- peritoneal cover: fixed posteriorly by the Treitz's fascia, anteriorly by the posterior peritoneum, the root of the transverse mesocolon, the gastrocolic ligament, the right mesocolon; The tail lies between the two layers of the pancreaticosplenic ligament.
- turgor effect and abdominal pressure

Aquiring a good knowledge of anatomy is important for the osteopath in order to be able to perceive dysfunctions.

If, for example, the mobility of the transverse colon or its fixation at the thorax (left and right colic flexure) is restricted, the mobility of the pancreas will also be reduced. A lack of movement, even on the smallest scale, always means reduced blood flow, lymphatic drainage, bradytrophia... .

2.1.7. Thoracolumbar diaphragm

With diabetes mellitus the diaphragm must not be disregarded.

“The diaphragm carries out 20.000 movements per day, each time causing the lungs and the abdominal organs to move, too. [...]

The constant change of the abdominal cavity’s shape as it moves to and fro between two extreme positions – the end position of the inspiratory phase and the end position of the expiratory phase – causes gliding movements between the individual organs.” (Barral, Mercier, 1994, 20-21)

The osteopath tries to sense the tension of the diaphragm and to remove dysharmonies. The diaphragm is a dome-shaped fibromuscular septum between the abdominal and the thoracic cavity. Nearly all structures of the body are directly or indirectly related to the diaphragm. Its muscles arise in a circle from the thoracic outlet and arch upwards to reach a central aponeurosis.

The muscular parts of the diaphragm can be subdivided into the lumbar, the costal and the sternal part.

“Orifices of the diaphragm

- Esophageal hiatus: left of the aortic hiatus, at the level of the tenth thoracic vertebra, composed of the muscle fibres of the lumbar part. The esophagus and the vagal nerves traverse the esophageal hiatus.
- Aortic hiatus: a bit left of the median line at the level of the twelfth thoracic vertebra, composed of the median arcuate ligament of the lumbar part. The descending aorta traverses the aortic hiatus anteriorly and the thoracic duct posteriorly.
- Vena cava hiatus: a bit right of the median line at the level of the ninth thoracic vertebra, composed of fibrous structures of the central tendon. The inferior vena cava and the right phrenic nerve traverse the vena cava hiatus.” (Liem, 1988, 361)

Here the question arises, in what way the diaphragm will influence the dynamics of the surrounding structures if it is too tense (blood vessels, lymphatics, esophagus, pancreas, liver, stomach, etc.).

2.1.8. Embryology

“During embryonal life several cellular changes occur: We start with a fertilized ovum and end up with thousands of millions of cells. These cells do not develop arbitrarily, but follow a defined pattern within space and time. There is a „coordinator“ that forces the cells to adapt and leads them to a development in full harmony.

The cell has a brain.”(Barral, Mercier 1994,25)

In the course of the development the embryonal organs shift. The stomach, for example, undergoes two rotations - a horizontal one and a frontal one. Embryologic theory claims that the visceral tissue maintains this memory and reiterates these movements rhythmically (Motility). (cf. Barral, Mercier1994)

The embryologic axes

“Our investigations are based on clinical and experimental facts. Each organ was examined by assessing mobility and motility. Exactly determined axes were allocated to these movements. We were astonished to realize that the directions of these movements were identical with those of the embryonic development. We had started the project unbiased and without a study hypothesis. It was just empiricism that discovered this phenomenon. Therefore, we can claim that the cells do not forget... .” (Barral, Mercier1994, 25)

If we want to learn how we can understand and feel motility better, we need embryologic knowledge. In this work I will confine myself to the rotations of the abdominal tract.

The gastrointestinal tract develops from a swelling of the rectal tube.

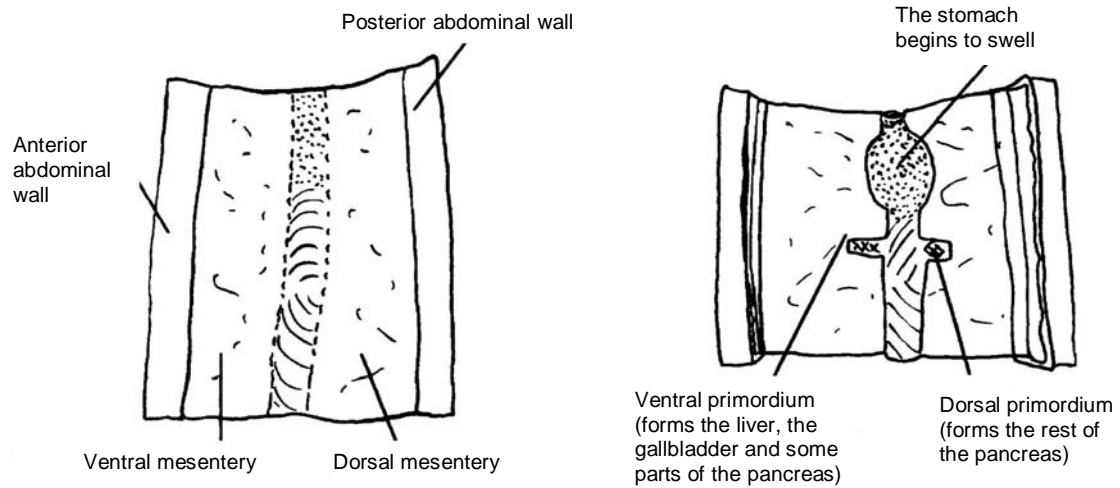


Fig. 7: lateral view of the rectal tube / the stomach with the pancreatic buds and the liver

The bud that develops between the layers of the ventral mesentery and grows towards the anterior abdominal wall forms the liver, the gall bladder, the bile duct and a part of the pancreas. The other bud that develops between the layers of the dorsal mesentery and grows towards the posterior abdominal wall forms the rest of the pancreas.

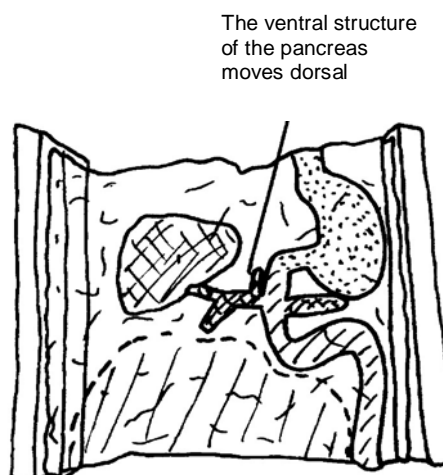


Fig. 8: lateral view, movement of the ventral pancreatic bud

In Fig. 8 the hepatic bud, as well as the curvature of the intestine – in order to adjust to its new length - can already be clearly made out. In this phase all organs are situated in a sagittal plane.

According to Stone, at first, the anterior part of the pancreas and, therefore, the bile duct and the duodenum, too, rotate in posterior direction. The anterior and the posterior part of the pancreas fuse and become situated retroperitoneally at the dorsal abdominal wall.

Afterwards the liver moves to the right and the stomach to the left (the greater curvature of the stomach rotates to the left)

During this movement the dorsal mesentery has to expand in order to allow rotation. The result is a forward bulge of the mesentery between the stomach and the posterior abdominal wall. This forward bulge is called greater omentum.

The small and the large intestine start rotating counter-clockwise around the arterial axis, superior mesenteric artery. The colon coils itself to the right around the small intestine and appears to lie superior of it. During this rotation there seems to be too much of a peritoneum, which, however, enables the small intestine to rotate freely. The peritoneum undergoes absorption and forms the root of mesentery. (cf. Stone, 1996)

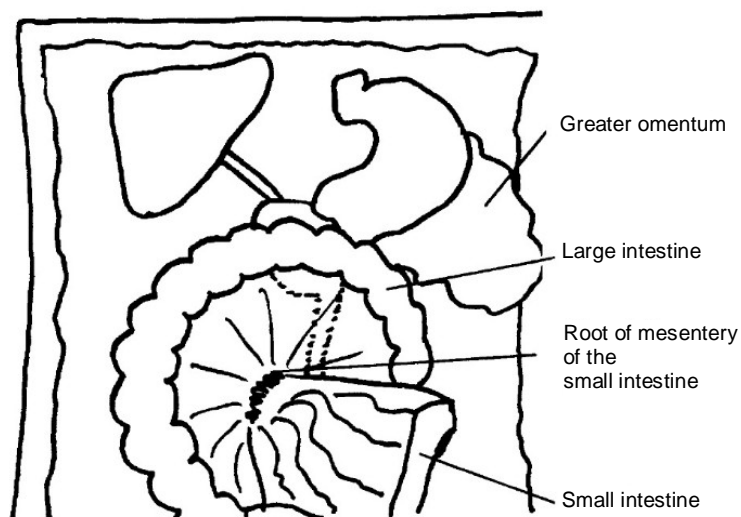


Fig. 9: Root of the small intestine

The small intestine is attached to the posterior abdominal wall by the root.

Drews (1993) described a further counter-clockwise rotation of the intestinal tract on account of the fixation of the colon. Again the axis is the mesenteric artery– the small intestine positions itself in the frontal plane.

“A theory stated: If there are too many fascial tensions or adhesions in the abdomen, part of these tensions might exist in the fasciae around the blood vessels. This could lead to irritations of the blood vessels and therefore cause disturbed visceral function.“
(Stone,1996, 67)

2.2.Physiology

The pancreas consists of eccrine and endocrine glandular tissue.

With problems of the pancreas Barral(1994) recommends manipulation of the Oddi's sphincter (where the hepatic, biliary, and pancreatic ducts join each other) as a first step of local treatment. Pancreatic secretion can amount to up to 2 litres per day. This fluid should be able to move easily from the pancreas to the duodenum. It seems to be important for the metabolism.

Therefore, I will start by describing the exocrine glandular tissue.

2.2.1.Exocrine glandular tissue

The eccrine glandular tissue consists of several thousands of lobules, which are separated from each other by thin septa of connective tissue. The lobules are composed of several hundreds of acini each. An acinus consists of about 70 glandular cells. 2 to 4 acini form an acinar complex, which is connected to the ductal system through a common intercalated duct. (cf. Benninghoff, 1994)

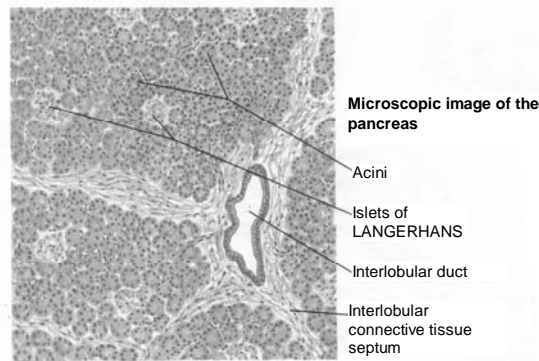


Fig. 10: Showing the acini

The secretion of the acini is conducted through the intercalated ducts into interlobular ducts. The interlobular ducts merge into larger ducts and lead to the main ducts, the major and the minor pancreatic duct.

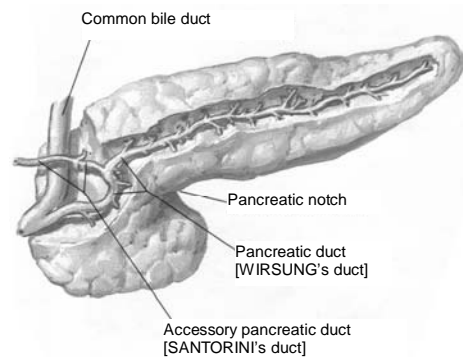


Fig. 11: The pancreatic ducts and the common bile duct

The pancreatic juice contains a number of enzymes, which are necessary for the digestion of proteins, fats and carbohydrates.

A proteolytic enzyme is trypsin, for example

A lipolytic enzyme is lipase, for example

The enzyme responsible for carbohydrate digestion is L-amylase (cf. Thews, 1990)

“ The pancreas also secretes pancreatic juice in small amounts outside of digestive phases (basal secretion). The stimulation of secretion through eating is achieved by neural and hormonal means.

The phase of pancreatic secretion that is under the influence of nervous impulses from the central nervous system is called cephalic secretion phase. Before and during eating olfactory and gustatory stimuli involuntarily trigger secretion. The sight or imagination of food also stimulates secretion. Here the efferent nervous impulses are conducted through the vagus nerve. [.....]

The following gastric phase of pancreatic secretion is triggered when the food reaches the stomach. Especially mechanical expansion stimulates increased secretion through local reflexes [.....].

The third phase, the intestinal phase, is triggered by the passage of acidic contents of the stomach (pH < 4,5) or of fat and protein catabolic products into the duodenum.” (Theews, 1990,318-319)

2.2.2. Endocrine glandular tissue

The physiological mechanism important for the regulation of blood sugar lies in the endocrine glandular tissue of the pancreas.

The endocrine part of the pancreas is composed of all of the islets of Langerhans or pancreatic islets. These develop mainly in the dorsal pancreatic primordium, therefore most of them can be found in the pancreatic tail. During the first years of life numerous new islets develop. The totality of the islets in an adult is between 500 000 and 1.5 million (about 2% of the pancreas' weight). (cf. Benninghoff, 1994)

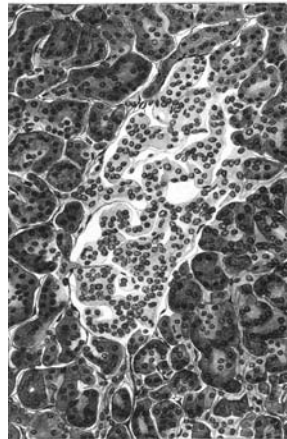


Fig. 12: Section through an elongated islet of Langerhans

The islets lie mainly in the exocrine glandular lobules and only occasionally in the interlobular connective tissue. The endocrine cells are arranged in ramified bar-shaped clusters, which locally can be in direct contact with acinar cells of neighbouring acini. The bar-shaped islet-cell clusters are bathed in capillaries of wide lumen. The insular capillary plexus is supplied by one to two arterioles, which reach at the islets through the interlobular septa of connective tissue. The efferent capillaries emerging from the islets open into the capillary system that surrounds the exocrine acini. (cf. Benninghoff, 1994)

This orientation of the microvascular pathway is called the portal system of the pancreas. On its account, the hormones secreted by the islets at first reach the acini of the exocrine pancreas in high concentration where they partly influence the acinar function before they are conducted by the pancreatic veins to the portal vein and to the liver.(cf. Benninghoff,1994)

Four different types of endocrine cells can be regularly established in the islets of Langerhans in humans:

“A-cells (glucagon cells) ¹

These lie mainly at the periphery of the islets and at the margins of the islet-cell clusters, i.e. close to the capillary system of the islets and the periinsular connective tissue.[...] The cells contain the hormone glucagon.[...] Glucagon reaches the liver through the blood of the portal vein, where it stimulates the release of glucose from glycogen (which stores glucose at a high level of food intake) through receptors and the production of glucose from amino acids received from the bloodstream. On account of this, glucagon leads to an increase of the blood sugar level. The release of glucagon from the A-cells is stimulated by a drop of the blood glucose level (< 100 mg/ml).”(Benninghoff,1994, 934)

“B-cells (insulin cells) ¹

B-cells constitute approximately 80% of the islet cell population. They spread out nearly evenly over the whole islet.[...] The secretion of insulin is triggered by glucose flowing into the B-cells.[...] A disturbed insulin-release mechanism in the B-cells leads to the onset of diabetes mellitus type II.[...] B-cells normally store the insulin requirement for two days. If this amount of insulin were released at once it would cause a lethal hypoglycaemia. If the amount of circulating insulin is too high, the blood glucose level can drop so heavily (hypoglycaemia) that it can result in unconsciousness and respiratory paralysis (The brain gets the most part of its energy requirement from the blood glucose.) Several hormones inhibit the secretion of insulin and counteract hypoglycaemia: the somatostatin of the D-cells [...] and the adrenalin of the adrenal medulla. Two hormones from the endocrine cells of the intestinal epithelium, insulinotropin and the glucose-dependent insulinotropic peptide, stimulate the secretion of insulin.”(Benninghoff,1994, 936)

“D-cells (somatostatin cells)

These cells constitute about 5% of the islet cell population and lie mainly at the margins of the cell clusters near the capillaries. Somatostatin inhibits the secretion of insulin and glucagon. Glucagon for its part stimulates the release of pancreatic somatostatin. Insulin, however, inhibits the somatostatin cells.”(Benninghoff,1994, 937)

¹ A-cells / B-cells or α -cells / β -cells, depending on the author

I want to give a better understanding of the regulation of the blood sugar because it is basic knowledge with regard to the disease diabetes mellitus.

The following information is from Thews:

The two main pancreatic hormones regulating the blood sugar are **Insulin** and **Glucagon**. Insulin is a vital hormone. Facilitated by a membrane-bound insulin receptor it influences the metabolism of carbohydrates as well as of protein and fat.

Insulin

- improves the uptake of glucose (blood sugar) into the cells of most of the tissues (with the exception of neurons)
- enhances oxidative glucolysis
- enhances the production of glycogen (glycogen stores glucose) in the liver and in the muscle
- stimulates the production of proteins and fats from glucose.

All these processes lower the blood glucose level under the influence of insulin. Moreover, insulin is important for fat metabolism: in the fat tissue and in the liver it supports the uptake of free fatty acids that are then stored as triglycerides. As insulin counteracts fat mobilization and lipolysis, it prevents ketone bodies, which are detectable with hypo-insulinism, from occurring. Furthermore, insulin enhances the uptake of potassium ions into the cells and reduces the catabolic effect of glucocorticoids (produced in the suprarenal gland) and thyroid hormones.(cf. Thews, 1990)

Glucagon

Glucagon constitutes an antagonist to insulin with regard to the blood glucose level. Glucagon, like adrenalin (from the adrenal medulla), enhances glycogenolysis in the liver. Moreover, it supports gluconeogenesis from lactate (the salt of the lactic acid) so that the blood sugar level increases.

Glucagon has a twofold effect on fat metabolism: On the one hand, it increases the oxidation of fatty acid in the liver, on the other hand, it supports the storage of fatty acids as triglycerines. Summing it up, its functions can be described as follows: Glucagon supplies glucose when it is needed and allows consumption of fatty acids, if possible.

In protein metabolism glucagon enhances desamination of amino acids and their utilization for gluconeogenesis. (cf.Thews, 1990)

The blood glucose level can be seen as a resultant of glucogenic and glucolytic processes in the organism.

glucogenic: glucose supply through food

glycogenolysis (glucose is released)

transformation of galactose (ground substance of the carbohydrates)

transformation of fructose

gluconeogenesis (new formation of glucose from non-sugar elements)

Glucolytic: glucose oxidation

glycogen synthesis

lipogenesis

“By activating or inhibiting the individual mainly hormonally controlled processes the system remains constant at the desired value (0,6 to 1,0 g/l) of the blood sugar level. [...] If the blood glucose level (after food intake, for example) increases, more insulin will be released in order to bring back the increased blood sugar level to the desired value. It is assumed that the glucose level is measured directly in the pancreas and the secretion rate of the B-cells is directly controlled there. Moreover, somatotropin (in the anterior lobe of the hypophysis) enhances insulin secretion, whereas somatostatin (in the hypothalamus) inhibits insulin secretion. [...]

Adrenalin, glucagon and somatotropin are also involved in blood sugar regulation. Adrenalin and glucagon support above all the release of glucose from the depots, somatotropin inhibits the uptake of glucose into the cells. These three antagonists of insulin are secreted increasingly, if the blood glucose level sinks below the normal value, thus initiating an increase of the glucose level. The secretion of adrenalin, glucagon and somatotropin is controlled by the hypothalamus, where hypothetical glucose receptors register the decrease of the glucose level.” (Thews, 1990, 439-442)

Finally I want to summarize which organs and systems are involved in the regulation of blood sugar.

pancreas: insulin – blood-sugar decreasing

glucagon – blood-sugar increasing

somatostatin – blood-sugar increasing

liver: stores glucose

suprarenal gland: adrenalin – blood-sugar increasing

intestine: insulinotropin (endocrine cells of the intestinal epithelium) – blood-sugar decreasing

insulinotropic peptide (endocrine cells of the intestinal epithelium)- blood-sugar decreasing

muscle: stores glucose

autonomic nervous system

hypothalamus – hypophysis: superior regulators of the hormone system

This shows how complex the regulation of blood sugar is and how many organs are involved. This corresponds to the complex osteopathic point of view. The osteopath does not treat symptoms or just one organ, but the whole system.

„ When you're working on any one part, you're in contact with all parts. You're listening and feeling through one part, but you are hearing the whole as you work. If you keep that open-door attitude toward the work, it becomes more than just a unit – it becomes a whole unit.” (Becker, 1997, 8)

2.3.Pathophysiology

Diabetes mellitus is derived from Greek. „Diabetes“ stands for the fast flow – the increased urine production with high blood sugar levels, „mellitus“ stands for the honey-like taste of the urine.

Diabetes mellitus is the most frequent disease of metabolic disorders. By definition, it is called diabetes if a blood sugar level of 120mg/dl is permanently exceeded. There are different kinds of diabetes. Type I and type II occur most frequently. This study only deals with patients suffering from type II. Therefore, the pathophysiology of type I will merely be briefly described in order to know the main criteria for differentiation.

2.3.1 Type I

Diabetes type I results from the destruction of B-cells, which causes an absolute hypo-insulinism. It is assumed that endogenic antibodies inadvertently aim at the B-cells in the tissue of the pancreatic islet. (cf.Ruhland, 1985)

According to Krück (1988), the destruction of the B-cells is a result of the interaction between endogenic and exogenic factors. “Long before the clinical manifestation an insulinitis with lymphocytic infiltration exists as an expression of an auto-immune mechanism directed against B-cells that disappears after total destruction of the cells. The B-cell mass gradually decreases, and a clinically relevant hypo-insulinism occurs when more than 80% of the cells are destroyed. Sometimes a fibrosis occurs. [...] The gradual destruction of B-cells becomes clinically manifest when the B-cell mass falls below a critical point and the B-cell function is overtaxed by additional strain (infection, etc.). This infection, however, should not be regarded as the initiator of the underlying destruction of the B-cells, which must have existed much longer. This is also confirmed by the detection of ICA (Islet-Cells Antibodies) months or years before the onset of diabetes. Sometimes islet-cell antibodies disappear again without diabetes occurring.” (Krück, 1988, 364)

By comparison, Berger (1995) says: “Recent epidemiological findings indicate that particularly the conditions of the first years of one’s life influence the individual diabetes risk. The widely held assumption that virus infections or environmental toxins cause the onset of insulinitis, however, has not been proved yet.

“Chronic progressive insulinitis seems to be the direct cause of β -cell destruction. First studies in humans supported the assumption that the development of the disease starts soon after birth. Immunohistochemical analyses of insulinitis with manifest diabetes showed that all essential types of immunocytes were present in the islet. [...] At present it is undecided if diabetes type I constitutes an auto-immune disease. That much is certain, however, that immune reactions essentially contribute to the destruction of β -cells. [...]

A genetic predisposition is an indispensable precondition, too.” (Berger, 1995, 209)

2.3.2. Type II

The pathophysiological conditions with diabetes type II are much more complex than with diabetes type I. Destruction of B-cells is not in the foreground here, but disturbed secretion of insulin. In spite of sufficient insulin production hyperglycaemia (high blood-sugar) can occur because the insulin is ineffective (insulin resistance). Moreover, increased hepatic glucose release contributes to hyperglycaemia. Diabetes type II is much more likely to be hereditary than diabetes type I. Overweight plays a more important role than with diabetes type I. The higher the body weight is, the more resistant to insulin the organism becomes. The tolerance is tremendous, however, since only 10% of overweight people develop diabetes type II in the end.

According to Krück(1988), too little effect of insulin is the case in about 80% to 90% of the patients with diabetes mellitus type II. This can develop due to insufficient secretion of insulin from the B-cells, or because the hormone does not fully take effect at the peripheral target organs.

“As long as the B-cell function has not decreased to below 20% of the standard, the high sensitivity of the still functioning B-cells is sufficient to keep the fasting blood sugar within the normal range. Any further decrease of this function, however, results in a sharp increase of the blood sugar, which in turn stimulates the remaining B-cell function so heavily that the basal insulin level can be kept nearly at standard. If finally the B-cell function decreases to about 5% to 10% of the standard, the basal insulin level will be further reduced despite hyperglycaemic stimulation. At this stage even a further slight increase of insulin resistance, for example due to adiposity, leads to a considerable fasting hyperglycaemia. Dietetic measures and/or the stimulation of the B-cell function by sulfonyl urea can prevent or reverse the increase of insulin resistance.

Depending on the relative involvement of B-cell dysfunction and insulin resistance, a diabetes mellitus type II can be accompanied by a low, normal or increased insulin level.[...]

The true molecular disturbances are not known yet. A receptor defect or a reduction of the insulin receptors prevails in the cells of the peripheral target organs, which results in impairment of glucose transport activity. With increasing fasting hyperglycaemia the possibility of a postreceptor defect, which is regarded as the main cause of insulin resistance, has been discussed.

In addition to this, insulin secretion stimulated by glucose is primarily delayed in the first secretion phase and less in the late phase. Thus, the peripheral insulin resistance cannot be compensated.[..]

Which defect occurs first is not clear yet. Frequently both defects exist at the same time. A disturbance of glucose identification might be present, which leads to inhibition of secretion in the B-cells and to decrease of glucose transport activity in the peripheral target organs. [...]

Morphologically, the islets can be enlarged and the number of the B-cells increased, but a slight reduction, which never surpasses 50%, however, is also possible. Amyloidosis in the islet cells (degeneration of tissue with storage of amyloid) has frequently been found. [...] It is possible that the amyloid originates from an insulin waste product that is primarily produced by aging or diabetic (type II) islet cells.” (Krück, 1988, 364-365)

The osteopath might be able to influence this storage mechanism. Presumably he / she can slow down the storing process, however not by causal therapy but by accompanying treatment such as measures stimulating the blood circulation, mobilization of the diaphragm, mobilization of the flexures of colon, mobilization of the root of mesentery, etc. This will be dealt with in greater detail in the chapter „Discussion“.

Furthermore, Krück (1988) says that strong genetic factors cause a predisposition for diabetes type II. One of these factors might be the regulation of the insulin receptors and, on account of that, the regulation of insulin resistance or the insulin response to glucose stimulus.

According to Berger (1995), at least three substantial defects in key functions for glucose homeostasis in different organ systems exist. They are manifest with fully developed diabetes type II. In the course of the disease's development, however, they can occur one after another and cause or increase each other.

“These three defects are:

1. changed, abnormal insulin secretion in response to glucose stimulus
2. reduced capability of insulin to stimulate the peripheral uptake of glucose into the tissue (insulin resistance)
3. increased hepatic glucose production, more difficult to inhibit, despite already existing hyperglycaemia” (Berger,1995, 369)

Ad 1. Insulin secretion

Insufficient insulin secretion by the B-cells for the requirements of the metabolism constitutes a fundamental defect with diabetes type II. In most of the adipose type-II-diabetics insulin secretion is increased at the initial stage of the disease, but decreases during the progression of the disease. The absence of the early release of insulin by the pancreas at the beginning of the stimulation by glucose is noticeable.(cf. Berger,1995)

“The continuous change in the insulin secretion capacity in the course of diabetes type II is characterized by the increase of the circulating insulin level at the initial stage and the decrease to subnormal levels despite increasing hyperglycaemia at the late stage of the disease.[...] Hyperglycaemia occurs with diabetes type II only when insulin secretion – at

first excessively increased in a compensatory reaction, becomes unproductive.

The compensation (hyperinsulinaemia) takes place because, among other reasons, the number of insulin receptors of the peripheral cells in which glucose should be discharged is too low." (Waldhäusl, 1993, 28-29)

Berger further described that glucose carriers are involved in all key functions for the regulation of glucose homeostasis.

"Insulin secretion itself depends on the intact glucose-induced „insulin secretion-coupling“-mechanism. [...] Glucose sensors of the β -cells, as well as glucose carriers in the β -cells are a prerequisite for continuous “measuring“ of the blood sugar level – which in its turn is a precondition for the commensurate regulation of the secretion of islet cell hormones. [...]

Transportation of glucose molecules necessitates the binding to special glucose carriers (GLUT). GLUT 1, for example, is the carrier for erythrocytes, kidneys, brain, placenta and colon. GLUT 4 is the carrier for muscle, fat and heart [...]

Despite many recent findings the details of these signal-transduction-mechanisms are unknown." (Berger, 1995, 369-370)

Ad 2. **Insulin resistance**

Berger (1995) defined insulin resistance as a reduced insulin activity.

"With diabetes type II insulin resistance can be simplifyingly equated with reduced uptake of glucose in the peripheral insulin-dependent tissues. Reduced uptake of glucose compensatorily leads to higher insulin levels, hyperinsulinaemia, and, as things develop, to hyperglycaemia, if the compensation by the β -cells is not sufficient anymore. Therefore, glucose and insulin levels can often be regarded as indirect indicators of insulin resistance. [...]

Interaction between insulin secretion and insulin resistance should be understood as a dynamic, reciprocally influencing relationship. Increased insulin secretion, for example with adipositas, causes a decrease in the number of receptors by downregulation of the receptors. This results in reduced insulin activity or insulin resistance. Naturally, decreased insulin secretion with the same number of receptors causes reduced insulin activity. The decrease of insulin activity and insulin resistance, on the other hand, lead to

increased insulin secretion of the β -cell, hyperinsulinaemia, in an effort to compensate the resistance. The increase of insulin activity or increased insulin sensitivity, however, causes normalization or decrease of the excessively increased insulin secretion. This is the case with weight reduction in adipose patients or after sports activities and improved fitness.[..]

For the understanding of the pathogenesis of diabetes type II it is irrelevant in the end, whether the primary defect is an insulin secretion defect or insulin resistance. The clinical picture of the fully developed syndrome diabetes type II is characterized by the existence of both defects at the same time, which reciprocally increase each other in a vicious circle. [..]

In most cases, primary insulin resistance, which secondarily leads to hyperinsulinaemia, is considered the onset of the syndrome diabetes type II. The extent and localization of this primary insulin resistance, however, can differ considerably from patient to patient.” (Berger,1995, 377-379)

Ad 3. hepatic glucose production

Berger (1995) mentions enzymatic causes, hyperglucagonaemia (hyperfunction of the A-cells), insulin resistance as possible causes for unrestrained hepatic glucose production.

Siegel, Jakobs, Riemann(2001) in a contribution to the journal „Der Internist“, discuss relationships between diabetes mellitus and liver diseases. The liver ensures glucose production according to requirements . During fasting the liver mobilizes glucose from the storage cells in order to maintain the blood glucose level. In the case of a surplus the liver stores glucose. Impairment of this mechanism can be found with diabetes type II as well as with the so-called hepatogenic diabetes (for example with hepatic cirrhosis).

Waldhäusl defines the disturbed hepatic glucose production as an insulin resistance of the liver.

The relation between liver and diabetes type 2 is summarized in the article of Lecube, Hernandez, Genesca, et al.(2004). The aim of this study was to compare the prevalence of developing diabetes type 2 at patients with Hepatitis C virus infection (HCV+) to people with other liver diseases but Hepatitis C virus negativ (HCV -).

Examination has shown that people with (HCV+) develop a type 2 diabetes more than three times likely than those without Hepatitis C infection, but with other liver diseases.

I selected these articles about liver diseases in order to make conscious of the interplay of organs. No organ is working only by itself.

Fridyland, Philipson (2006) described the connexion of oxidative stress and insulin resistance. They write about coordinated development of oxidative stress and insulin resistance and B-cell dysfunction. Oxidative is defined as an imbalance between free radicals and antioxidant defences. They suppose cell damage as a result of oxidative stress. Several experimental studies showed that free radicals lead to insulin resistance.

Bell, Polonsky(2001) engaged in the theme of genetically programmed defects. The diabetes type 2 doesn't appear as an only genetic disease. Genetic pre-causes diabetes type 2. The outbreak of the diseases includes many causes such as obesity, stress, smoking, alcohol,... . Multiple interacting genes and environmental factors determine whether diabetes will develop or not, and at what age. Two main genes involved in type 2 diabetes are mentioned.

“A common amino-acid polymorphism (Pro12 Ala) in peroxisome proliferator-activated receptor- γ (PPAR γ) has been associated with type 2 diabetes.[..] Genetic variation in the gene encoding calpain-10, a ubiquitously expressed cysteine protease, has also been associated with type 2 diabetes, [...]” (Bell, Polonsky, 2001, 788)

Bell and Polosky described the identification of the genes that increase to develop diabetes Type 2 as a very arduous task and as a slow process.

A very dramatic development in the incidence of diabetes type 2 worldwide announced Zimmet, Alberti, Shaw (2001) in their article. They speak about a diabetes epidemic due to human behaviour and lifestyle in conjunction with genetic susceptibility. Diabetes mellitus is one of the main threats to human health in the 21st century.

By these informations we can only hope that clinical research will go on. Human beings have to be encouraged to overthink their lifestyle.

I am happy to be a small part of this information factor with my study.

Two more important terms for the basic knowledge of diabetes mellitus have to be mentioned:

Hypoglycaemic coma

Hyperglycaemic coma

With a **hypoglycaemic coma** the blood sugar level decreases rapidly, leading to a loss of consciousness in most cases. Before that, disorientation and disturbance of speech and vision occur. Possible causes are too intensive fasting, too high glucose consumption during muscular activity, serious diseases of the liver, islet-cell tumours, improper insulin medication, etc.

A **hyperglycaemic coma** is characterized by acidosis (including electrolyte shift, dehydration and insufficient blood supply of the brain). Warning signs are a strong feeling of thirst, increased urination, lack of impulsion, nausea, vomiting, etc.

Other causes for diabetes mellitus:

Pancreatitis (more likely chronic than acute), resection of the pancreas (segmental and total after tumourectomy, for example), diseases of the liver, etc.

2.3.3. Therapy of diabetes mellitus

Type I

In insulin-dependent type-I-diabetics insulin must be administered by insulin injection. In addition, a diet and much exercise are necessary.

Type II

About 80 to 90 per cent of the type-II-diabetics are overweight. Most of them can do without medication, at least at the onset of the disease, instead they have to be on a low-caloric diabetic diet. The individual daily amount of food is calculated in bread units. One bread unit is defined as that amount of food that is equivalent to 12g glucose with regard to its effect on the metabolism of the diabetic.

One bread unit is for example 250 g buttermilk or
100 g apple or
20 g wheat wholemeal flour

A diet only makes sense as long as the B-cells still work. Food rich in roughage delays the uptake of carbohydrates from the intestine.

In addition, exercise has a blood-sugar decreasing effect, because muscular activity causes glucose consumption. However, the diabetic must pay attention to the right measure. Short, very intensive muscular activity, for example a sprint, increases the blood-sugar level. In contrast, too long-lasting muscular activity (a bicycle day's tour) has a sugar decreasing effect, particularly in an insulin-dependent diabetic. Therefore, regular well-dosed training is the most appropriate thing to do. Progress must be followed by measurements.

Only if the blood-sugar levels remain unsatisfactory despite dietetic and physical efforts, the diabetic must turn to medication.

According to Ruhland, 1985, there is a choice of three groups of drugs:

1. Biguanides

Ruhland mainly describes Metformin from this group of drugs. It increases the insulin sensitivity, delays the uptake of sugar from the intestinal canal and inhibits gluconeogenesis in the liver. Biguanides cannot lead to low blood sugar.

2. Acarbose

It serves mainly to smooth out the blood-sugar peak after meals. Moreover, it slows down carbohydrate digestion. It cannot lead to low blood sugar, either.

3. Sulfonyl urea

Sulfonyl ureae stimulate B-cells that are still able to produce some insulin to increase the release of it. These substances, however, can lead to low blood sugar. Regular checks are useful.

If the oral therapy is exhausted, change-over to insulin therapy by injection becomes necessary.

2.3.4. Consecutive symptoms of diabetes mellitus

These are diseases that result from bad blood-sugar stabilization over years. The most affected organs are the following:

Blood vessels

Erythrocytes: The haemoglobin releases oxygen, which is necessary for energy consumption in the cells, worse with chronic hyperglycaemia.

Thrombocytes: High blood sugar causes the thrombocytes to agglutinate with each other and to clump.

Leucocytes: They are the defence cells of the head. They lose power with chronic hyperglycaemia –susceptibility to infections increases.

Early arteriosclerosis affects particularly coronary arteries, blood vessels supplying the brain and arteries of the leg. The results are myocardial infarction, apoplectic stroke, necrotic foot – even leading to leg amputation.

Kidneys

Metabolic imbalance harms the renal corpuscles – they become ever more permeable for protein. This leads to sclerosing of the renal corpuscles. The filtration function cannot be ensured anymore. Moreover, blood pressure regulation, which is partly controlled through the kidney, cannot function anymore.

Eyes

The most frequent changes in the eyes caused by diabetes occur at the retina. The blood vessels dilate. Micro-aneurysms occur. The organism reacts to the chronic disturbance of circulation by vasculogenesis. These new vessels, however, lift off from the retina and pull it with them. This can result in blindness.

Nerves

Glucose and catabolic products concentrate in the nerve tissue – diabetic neuropathy develops. Long nerves are more often affected than short ones. Therefore, chronic hyperglycaemia becomes more likely noticeable in the legs. The symptoms are burning feet, impairment of the sense of touch, or pain.

Still, it should be the aim that affected people, with their metabolism optimally stabilized, feel diabetes not as a disease but as a certain “state“ that they can well cope with.

The osteopath should know the pathophysiology of diabetes in order to recognize the stage of the disease and to draw conclusions from a loss of vitality. He/she can thus understand the interaction of the organs liver – pancreas, muscle tissue, hormone system, etc., better. The targets for improvement differ depending on the stage and vitality of the patient. Pathophysiology should contribute to this understanding.

In spite of pathology it seems sensible to include the following quotation in the osteopathic way of thinking.

“To find health should be the object of the physician; anyone can find disease”. A. T. Still (Liem,1988, 1)

3. Methodology

The osteopathic leitmotif in this thesis is movement – most subtle movement, at different levels. The gear-wheel mechanism– the movement of a wheel causes the movement of another wheel. Applying this mechanism I have asked myself whether the improvement of mobility positively influences the metabolism.

The results will be determined by the following two measurement methods.

3.1.Measurement Methods

The most commonly used measurements are the HbA1c-measurement and the blood-sugar measurement. The measurements are carried out by general practitioners and laboratories.

3.1.1. HbA1c-value

The HbA1c–value gives information on the binding of glucose to haemoglobin. Haemoglobin constitutes the main part of the erythrocytes (95 % of the dry substance). Erythrocytes are freely permeable to glucose. Glucose can permeate into the erythrocytes with the aid of a carrier. The HbA1c-value depends on the concentration of the surrounding blood glucose. Due to the limited life-span of erythrocytes (about 100 days), the HbA1c-value gives information on a period of 6 to 8 weeks.

The general practitioner takes blood and sends it to the laboratory, where HbA1c can be determined by centrifuging and haemolysis. The measurement will be carried out 4 times with intervals of 12 weeks.

3.1.2. Blood-sugar measurement

In contrast to the HbA1c-value, the blood-sugar measurement only determines the daily status. Thus, it is not so objective. The diabetic patient might adhere more strictly to his/her diet for 1 or 2 days in order to improve the values.

The doctor takes capillary blood from the lateral finger pad and uses it to measure the blood-sugar level with a test strip. For this study the fasting morning blood sugar will be measured 3 times with intervals of 12 weeks.

3.2. Selection of the Probands

10 probands are treated 6 times with intervals of 4 weeks.

Inclusion criteria are non-insulin-dependent diabetics, medicamentously stabilized type-II diabetics, and age 45 to 75 years.

Exclusion criteria are insulin-dependent diabetics, type-I diabetics, and diabetics suffering from other severe diseases (apoplectic stroke, myocardial infarct, multiple sclerosis,...). 10 diabetics selected by the general practitioner serve as a control group. The same inclusion criteria and exclusion criteria are applied here. I do not know the patients of the control group.

3.3. Procedure of the Treatment

The test persons are asked not to change any other factors (e.g. sports, keeping to the diet, taking of medicaments) in order not to influence the result of the osteopathic treatment. They should keep to their usual way of dealing with the disease in the test period.

General osteopathic treatment is used. I try to treat the patient individually and understand him/her in his/her totality. The tests (mobility tests, fascia tests, ecoute or local listening, motility-listening) are carried out with the patients standing, sitting and lying. The craniosacral system is tested, if not otherwise necessary, by occiput–sacrum-listening, by feeling the tension in the membranes (falx – tentorium) and perceiving the primary respiratory rhythm. In addition, the motility of the pancreas, the mobility of the diaphragm, and the condition of the tissue of Treitz's fascia will be tested in each proband.

The treatment is carried out in accord with the testing and the patient's tissue. Testing and treatment overlap time and again.

I want to emphasize the description of the local treatment of the pancreas according to Barral:

The first step when treating the pancreas should be the opening of the duodenum and Oddi's sphincter, using direct and indirect techniques. The peritoneum should be „released“. The root of the transverse mesocolon is attached to the anterior side of the pancreas. It is important to mobilize the root by stretching, starting from the two flexures of the colon. Only then should we question the „fragile“ organ pancreas by induction or listening. The ball of the thumb lies on the head of the pancreas, the rest of the hand lies alongside the head and tail of the pancreas. At the beginning of expiration we move the ball of the thumb backwards, then the metacarpus downwards, followed by the fingers. In the inspiration phase our hand is moved, from the fingertips to the ball of the thumb. The hand can be moved by the organ – not the other way round. Barral describes this technique as „rocking technique“. During rocking we give the tissue the opportunity to „release“.

In addition, I tried to communicate with the tissue by using ecoute at the pancreas. While doing so I asked myself several questions concerning motility, thickness of the tissue, elasticity, blood supply, lymph supply, innervation.

3.4. Critical reflections on the method

It has already been mentioned that the repeated measurement of the HbA1c-value during a period of 100 days leads to sound data. This value is significant, and it comprises the level of glucose, the effectiveness of drugs and how people deal with the disease.

The measurement of blood sugar levels by means of diurnal profile can easily be manipulated by short-term diets. Therefore, this value is a random value: only the blood sugar level of one day is recorded. The significance of the value is restricted to a short period of time.

In addition, the objectivity of the osteopathic test and treatment has to be considered. The treatment depends on my current osteopathic skills and my osteopathic knowledge. The powers of my perception during the treatment also depend on my daily mood. This can have a negative influence on the objectivity of the results. Furthermore, the patient's daily mood varies too. Not only can excessive eating and lack of exercise, gymnastic.. lead to worse levels of glucose, but stress factors, for example, bad news and family disputes, can also have a negative effect on the metabolism. Although I am trying to work to the best of my knowledge, the method is thus not entirely objective.

4. Results

4.1. HbA1c – Measurement Results

Graphs show the curve of each proband compared to the curve of a control patient. The HbA1c-value is given in %.

According to an information sheet by the hospital „Barmherzige Brüder“ in Graz, % corresponds to mg/dl as follows:

5 %	80 mg/dl
5.5 %	97 mg/dl
6 %	114 mg/dl
6.5 %	130 mg/dl
7 %	147 mg/dl
7.5 %	163 mg/dl
8 %	180 mg/dl
8.5%	199 mg/dl
9 %	214 mg/dl
9.5 %	231 mg/dl
10 %	247 mg/dl
10.5 %	264 mg/dl

Times of measurement

t0: HbA1c-value at baseline

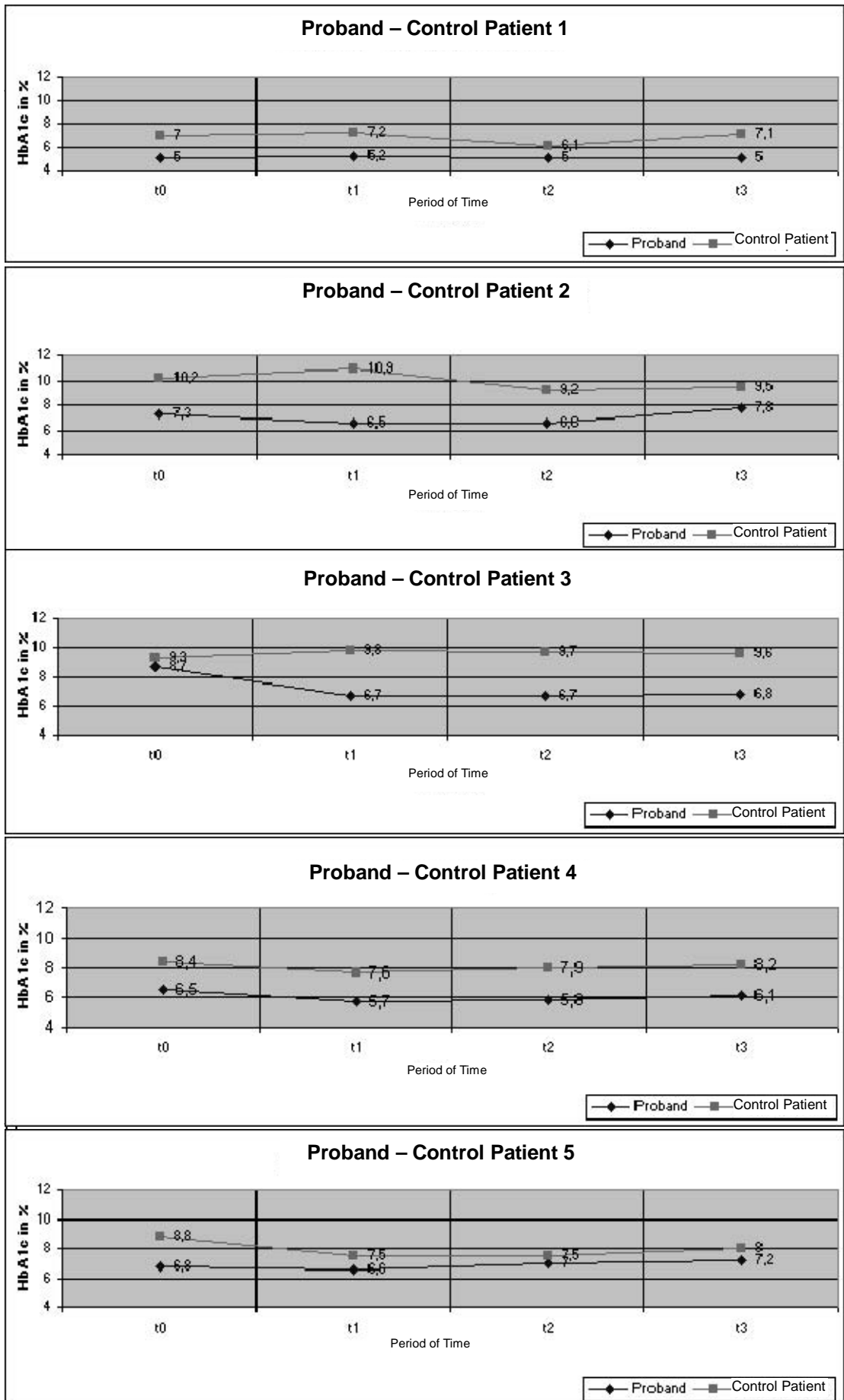
t1: HbA1c-value 12 weeks after the first osteopathic treatment

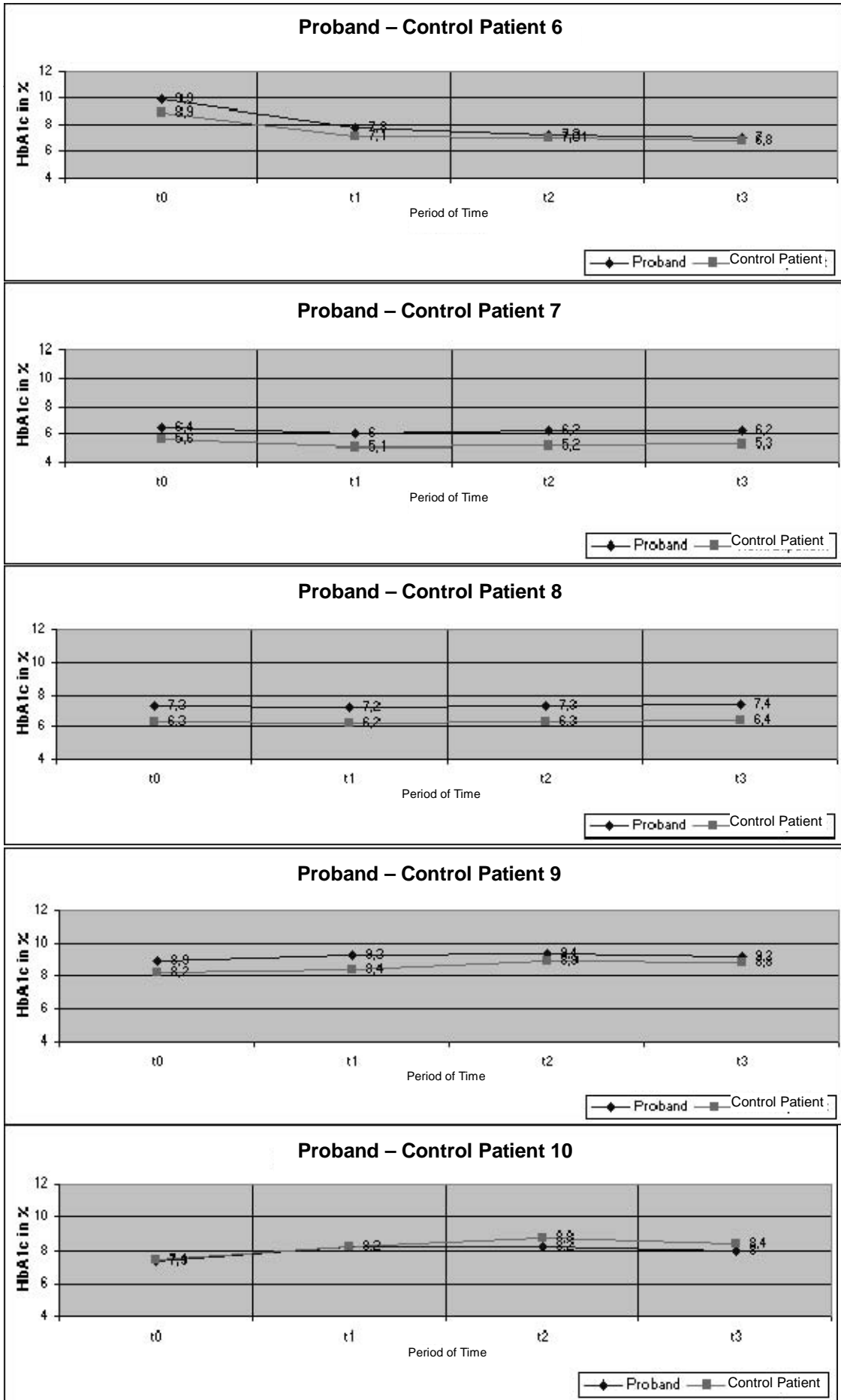
t2: HbA1c-value 24 weeks after the first osteopathic treatment

t3: HbA1c-value 36 weeks after the first osteopathic treatment

The probands were treated 6 times with intervals of 4 weeks. 4 treatments were carried out between t0 and t1, 2 treatments between t1 and t2.

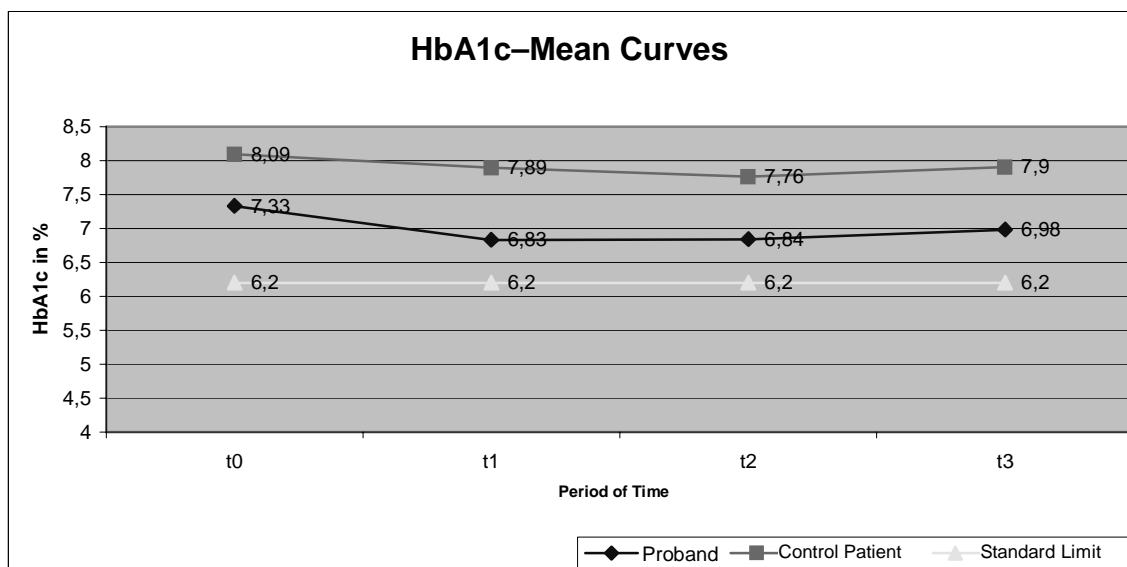
4.1.1. Graphs showing the HbA1c- measurement results





The results were evaluated by a graph showing the mean in relation to the blood-sugar standard limit (120 mg/dl – 6,2%)

Mean Curves of the HbA1c-Measurement Results:



4.1.2. Graph analysis:

C = Control Patient

P = Proband

S = Standard Limit

HbA1c	t0	t1	Change	t2	Change	t3	Change
C	8.09	7.89	-0.2	7.76	-0.33	7.9	-0.19
P	7.33	6.83	-0.5	6.84	-0.49	6.98	-0.35
S	6.2	6.2	0	6.2	0	6.2	0
P							
Absolute Deviation	1.13	0.63	-0.5	0.64	-0.49	0.78	-0.35
% above Standard Limit	18.23%	10.16%	-8.06%	10.32%	-7.90%	12.58%	-5.65%
	100.00%		-44.25%		-43.36%		-30.97%
C							
Absolute Deviation	1.89	1.69	-0.2	1.56	-0.33	1.7	-0.19
% above Standard Limit	30.48%	27.26%	-3.23%	25.16%	-5.32%	27.42%	-3.06%
	100.00%		-10.58%		-17.46%		-10.05%

In the osteopathically treated group the deviation of the HbA1c-value from the standard limit decreased by 44.25 % from t0 to t1, by 43.36 % to t2, and by 30.97 % to t3.

In the control group the deviation of the HbA1c-value from the standard limit decreased by 10.58 % from t0 to t1, by 17.46 % to t2, and by 10.05 % to t3.

In the osteopathically treated study group the deviation from the standard limit decreased by 33.67 % more than in the control group from t0 to t1, by 25.9 % more to t2, and by 20.92 % more to t3.

In this study the osteopathically treated patients of the study group improved by 33.67 %, 25.9 %, and 20.92 % more than the patients of the control group did.

In other words: In this study, osteopathy influenced diabetes in the patients of the study group.

But the significance of the study is questionable. The group of probands is too small. The results only show trends. The effect of osteopathic treatments is not evidenced by this study.

4.1.3. Possible osteopathic mode of action

The metabolic disease diabetes type II is characterized by reduced insulin activity at the receptors of the target organs (muscle, liver, etc.), by reduced insulin secretion from the B-cells of the pancreas, and by increased hepatic glucose production or hepatic insulin resistance. It is assumed that there is a receptor defect in the cells of the target organs. Hyperglycaemia can occur despite sufficient insulin secretion because the insulin is not active – the receptors do not understand their task anymore. Moreover, the dialogue between blood-sugar decreasing hormones (insulin from B-cells, insulinotropin in the intestine, somatotropin in the pituitary gland) and blood-sugar increasing hormones (glucagon from A-cells, adrenalin from the suprarenal gland, somatostatin from D-cells of the pancreas, from the hypothalamus) seems to be disturbed.

What could improve all these chemical dialogues and deficiencies?

In my opinion the “body fluids“ (liquor, lymph, blood supply), acting as mediators, are most important. The fluids carry glucose carriers into the B-cells of the pancreas to the glucose sensors, and into the hypothalamus to the glucose receptors. The more “mobile“ the fluids are, the better they circulate, the higher their quality ought to be, the more reactive the chemical dialogue ought to be.

What improves the mobility of the fluids?

Barral (2000) speaks of 20,000 inspiratory and expiratory movements of the diaphragm per day. A restriction of the diaphragmatic movement evidently involves a dramatic loss of the abdominal movement (organs, fluids, every tissue). The lymphatic pump – as Liem describes the diaphragm – was in a status of dysfunction in all probands. Stretching or

releasing improved the diaphragmatic movement again and should therefore change the surrounding area (pancreas, large intestine, small intestine, cisterna chyli, solar plexus, portal vein...).

In diabetic patients it is essential to treat the diaphragm. As can be seen in the following graphs showing the common dysfunctions, especially the left flexures of the colon are affected. The mobilization of the flexures releases the diaphragm – it can carry out its pumping function unrestrictedly. The exchange of lymph, liquor, blood functions well. The chemical dialogue has a better quality.

On the one hand, it is necessary to improve the mobility and motility around the pancreas (psoas muscle, Treitz's fascia, large intestine, small intestine, diaphragm, ...) and of course at the pancreas itself (mentioned in greater detail in the chapter "Discussion"), on the other hand, the craniosacral system must be in balance in order to secure the chemical dialogue, the hormonal coupling. Balanced membranes and a balanced sphenobasilar synchondrosis enable the undisturbed function of the pituitary gland and the hypothalamus. The regulators of the hormonal system can work more efficiently. Moreover, improved liquor flow ensures better conditions for the chemical dialogue.

The innervation of the pancreas (Th 5 – 9) often had to be treated. The neural sympathetic stimulation (by Thrust technique or the Muscle Energy technique by Mitchell at the vertebrae) and the parasympathetic supply (vagus nerve) of the pancreas are also necessary for the metabolism. The jugular foramen (exit orifice of the vagus nerve) had to be opened mainly on the left.

As can be seen in the following graphs showing the common dysfunctions, the tissues and organs are affected mainly on the left (left kidney, left suprarenal gland, left psoas muscle, left jugular foramen, ...). The irritation of the pancreas is transmitted through the fasciae. Fasciae enclose every muscle, every vein, every nerve and all the organs in the body. They constitute the interrelationships between the structures. If one link in the system goes wrong, chain reactions develop.

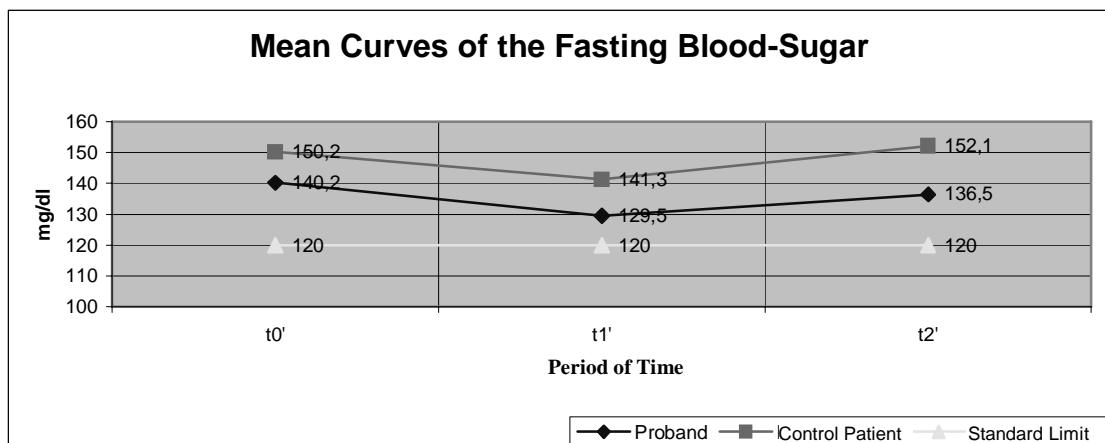
Particularly important for the balance within the entire body was the good positioning of the kidneys, so that they could glide in the fascial compartments. Further fascial tissue thus released automatically.

Further particularities will be discussed in the chapter "discussion".

4.2. Fasting Blood-Sugar Value Results

Only the mean curves are shown here. The blood-sugar value can change after a one-day diet only and is therefore not appropriate for statistical evaluation. Nevertheless, I will show the mean curves and their calculation, since I included them in my concept and the measurements were done. However, the values do not serve as a basis for further discussion.

4.2.1. Graphs showing the fasting blood-sugar results:



4.2.2. Graph analysis:

C = Control Patient

P = Proband

S = Standard Limit

Fasting Blood-Sugar	t0	t1	Change	t2	Change
C	150.2	141.3	-8.9	152.1	1.9
P	140.2	129.5	-10.7	136.5	-3.7
S	120	120	0	120	0
P					
Absolute Deviation	20.2	9.5	-10.7	16.5	-3.7
% above Standard Limit	16.83%	7.92	-8.92%	13.75%	-3.08%
	100.00%		-52.97%		-18.32%
C					
Absolute Deviation	30.2	21.3	-8.9	32.1	1.9
% above Standard Limit	25.17%	17.75%	-7.42%	26.75%	1.58%
	100.00%		-29.47%		6.29%

The deviation of the fasting blood-sugar value from the standard limit decreased by 52.97 % (t0 to t1) and by 18.32 % (t0 to t2) in the osteopathically treated group.

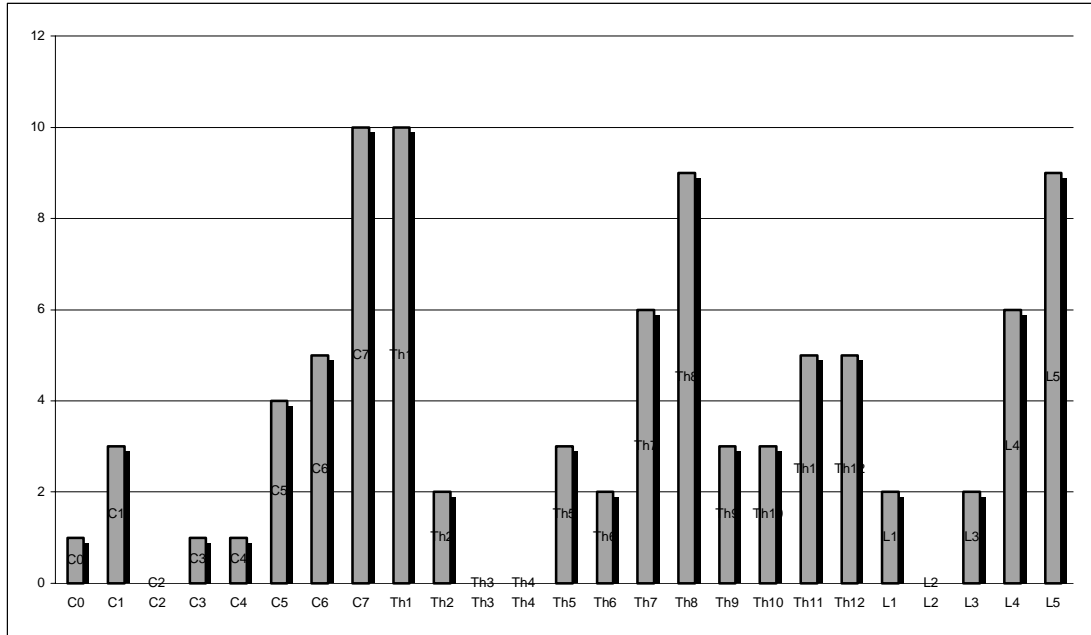
In the control group the deviation of the fasting blood-sugar value from the standard limit decreased by 29.47 % from t0 to t1, but increased by 6.29 % from t0 to t2.

The mean curves and the results in per cent are similar to the HbA1c-value results. Still, I base my thesis only on the HbA1c- value results in the following discussion. The fasting blood-sugar values were calculated to complete the picture.

4.3. Common Dysfunctions of the Probands

Results of the Mobility Tests for the Vertebral Column – Loss of Mobility

This diagram shows the results of osteopathic testing at vertebra column. These vertebral segments are most blocked and reduced in mobility.



C0		Th1	10	L1	2
C1	3	Th2	2	L2	
C2		Th3		L3	2
C3	1	Th4		L4	6
C4	1	Th5	3	L5	9
C5	4	Th6	2		
C6	5	Th7	6		
C7	10	Th8	9		
		Th9	3		
		Th10	3		
		Th11	5		
		Th12	5		

The most frequent concerned parts of vertebra column are seventh cervical vertebra, first thoracic vertebra, eighth thoracic vertebra and fifth lumbar vertebra.

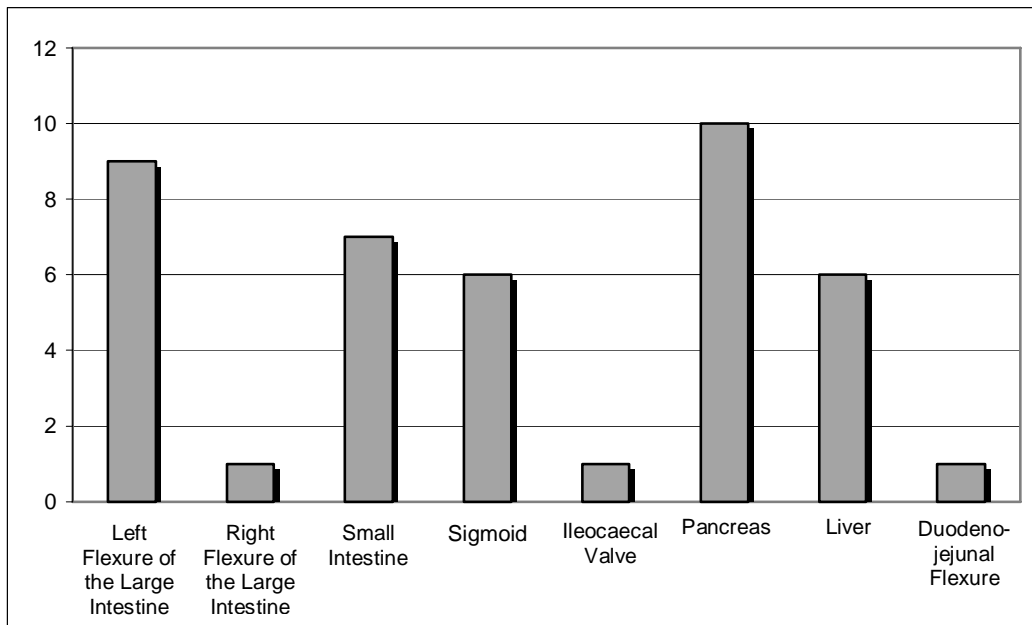
Moderate concerned are seventh – eleventh -twelve thoracic vertebra, fourth lumbar vertebra and fifth – sixth cervical vertebra.

Less concerned are occiput, first – third - fourth cervical vertebra, fifth – sixth- ninth – tenth thoracic vertebra and first – third lumbar vertebra.

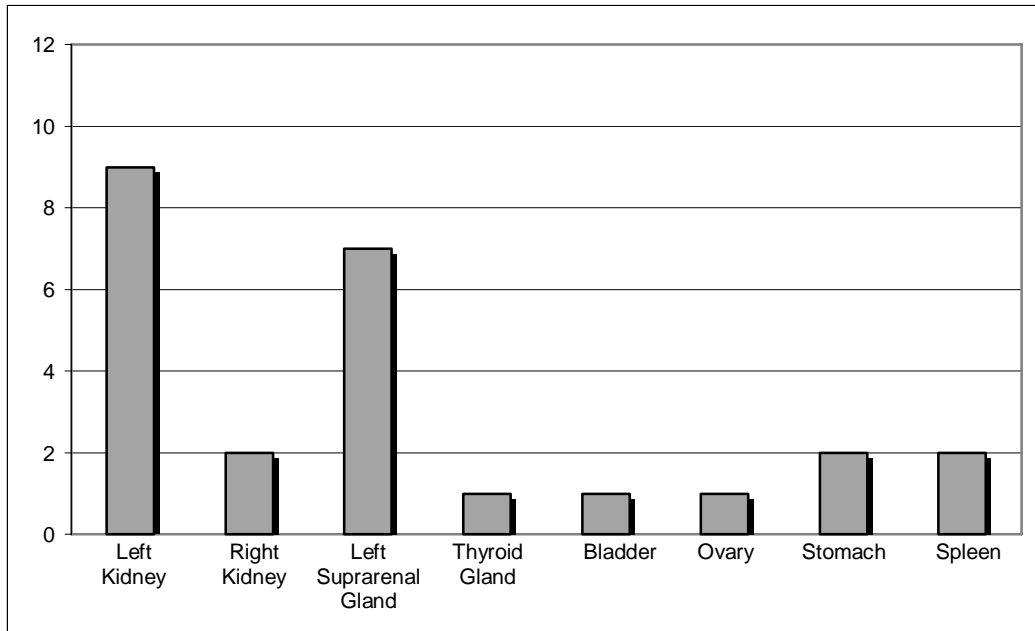
Not concerned is second cervical vertebra, third – fourth thoracic vertebra and second lumbar vertebra.

Results of the Visceral Examinations:

The next two diagrams are the results of osteopathic testing at viscera. They give us an impression how much organs and tissues are involved at the patients of the study group



Left Flexure of the Large Intestine	9
Right Flexure of the Large Intestine	1
Small Intestine	7
Sigmoid	6
Ileocaecal Valve	1
Pancreas	10
Liver	6
Duodenojejunal Flexure	1



Left Kidney	9
Right Kidney	2
Left Suprarenal Gland	7
Thyroid Gland	1
Bladder	1
Ovary	1
Stomach	2
Spleen	2

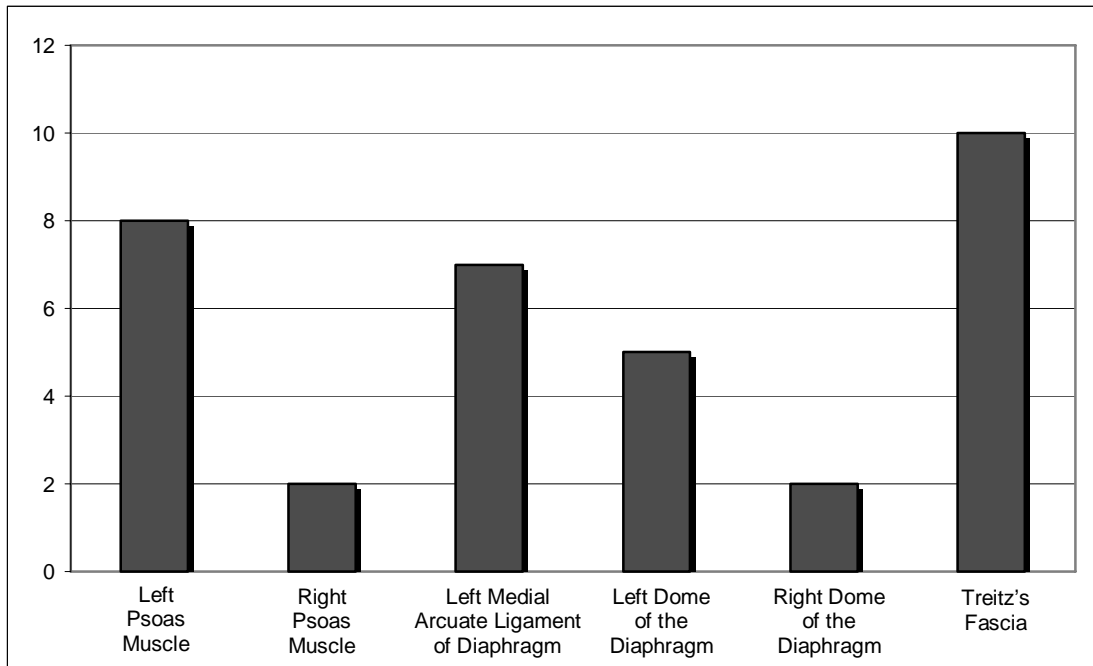
The pancreas is noticed to 100% at visceral testing. Left flexure of the large intestine and left kidney is noticed to 90%. Small intestine and the left suprarenal gland have positive results of 70%, liver and sigmoid 60%. Right kidney, stomach and spleen are concerned to 20%.

Ovary, bladder, thyroid gland, duodenojejunal-flexure and ileocaecal valve diverge from normal tissue tension to 10%.

Obviously is the left side of the abdominal viscera more concerned that the right side.

Loss of Mobility of Muscle and Fascia:

The osteopathic testing shows variations in muscle and fascia. The tissues are tight. The most concerned tissues are represented in this diagram.



Left Psoas Muscle	8
Right Psoas Muscle	2
Left Medial Arcuate Ligament of Diaphragm	7
Left Dome of the Diaphragm	5
Right Dome of the Diaphragm	2
Treitz's Fascia	10

The examination shows 100% loss of mobility in the Treitz's Fascia, 80% in the left psoas muscle, 70% in the left arcuate ligament of the diaphragm. The left dome of the diaphragm diverges from normal tissue tension to 50%. A little difference from normal tissue tension is seen at the right psoas muscle and the right dome of the diaphragm.

The tendency of positive results in higher tissue tension continues again on the left side. The results of the left side are more dominant.

Dysharmony in the Cranial Membrane System – Sphenobasilar Synchronosis:

Testing and working with the probands the cranial system attracted attention. All of the patients had dysharmonic tension in the membran system and at the sphenobasilar synchronosis. Especially the left side was concerned. The cranial system seems to be a mirror of the body. The tension in the body is transmitted to the head like a chain reaction. An other presumption arises that the cranium is steady busy to compensate the tension in the body.

5. Discussion

5.1. General Discussion:

Before I started this study I had my doubts whether osteopathy can influence a metabolic disease of such proportions, a disease that also extends over a considerable period of time, sometimes over several generations by hereditary transmission. In addition, permanent irritation caused by patients not keeping to the diet makes work more difficult. According to their own statements two out of ten probands kept strictly to the diet, three moderately, two a little, two nearly not, and one not at all. None of the probands did sports. As for physical exercise, daily activities in the household and at work were stated.

However, even from one treatment to the other I realized, that the tissue can be changed, that a rigid diaphragm can be softened, that a fixed flexure of the large intestine can be mobilized,..... . Still, I could feel a permanent tension between irritation (pancreas, liver, hormonal system, ...) on the one hand and the body's recollection of health (by osteopathy) on the other hand.

Therefore, it is easier to work with diabetic patients who keep to their diet well and do sports – the osteopath and the patient work in the same direction.

In the probands nine and ten the curves clearly rose. Proband nine did not keep to his diet at all, proband ten nearly not.

The HbA1c-mean curves in general, however, show that osteopathy positively influenced this study group.

This confirms the assumption that improvement of mobility positively influences the metabolism. The gear-wheel mechanism shows its effect. Improvement of mobility means better trophic effect, means better metabolism.

The study group improved by 33.67 %, 25.9 %, and 20.92 % (3 measurements at different times) more towards the standard limit than the control group did.

It is remarkable, that both the study group curve and the control group curve drew nearer to the standard limit.

The study group drew closest from t0 to t1 (HbA1c-Mean Curves in the chapter “Results”).

The control group showed the biggest change from t2 to t3. Both curves rose again at measurement t3, i.e. deviated from the standard limit. The study group curve rose slightly less than the control group curve.

The patients of the study group were treated four times from t0 to t1, and two times from t1 to t2. No treatment was carried out from t2 to t3. The best improvement towards the standard limit was achieved from t0 to t1, too. The rise of the curve at t3 indicates that the effect of osteopathy does not last due to the irritation in the diabetic patients (if not keeping to their diet). If the patient and the osteopath work in the same direction (i.e. the patient cooperates in aiming at health), the improvement will probably last longer.

Several reasons can be responsible for the decrease of the blood-sugar level in the study group:

1. The probands kept more strictly to their diet and medication during measurements although they were not particularly encouraged to do so.
2. The selection of patients was right. This means that the better improvement is pure chance.
3. The blood-sugar value is never constant. The decrease of the value is pure chance.
4. Osteopathy provides the possibility of better vitality for the patients, can remind the body of its health, fosters the self-healing mechanism.

“ The individual has a dual set of mechanism working concurrently throughout life – a voluntary capacity to work, play, and rest and a complex involuntary mechanism designed to maintain health and adapt to trauma and/or disease.” (Becker, 1997,84)

Becker (1997) also speaks of a potential for self-organization.

The following reasons can be responsible for the decrease of the blood-sugar level in the control group:

1. The control patients did not know that they were participating in a study. We cannot assume any change in their attitude towards their disease. It cannot be ruled out, though, that the doctors who treated them tried harder to improve the disease. This is, of course, quite right but unsuitable for the study.
2. The selection of the control patients lead to these values. This means that the decreasing values are pure chance.
3. The blood sugar is never constant. The decrease of the value is pure chance.

This means that influences cannot be ruled out in the study group as well as in the control group. Any influences can falsify the results. Moreover, it is clear that no definite conclusions can be drawn from this study due to the small number of probands.

Still, a tendency can be seen that the osteopath can influence diabetes, not causally but as an accompanying treatment. The gear-wheel mechanism – the movement of one wheel causes the next wheel to move – positively influences metabolism.

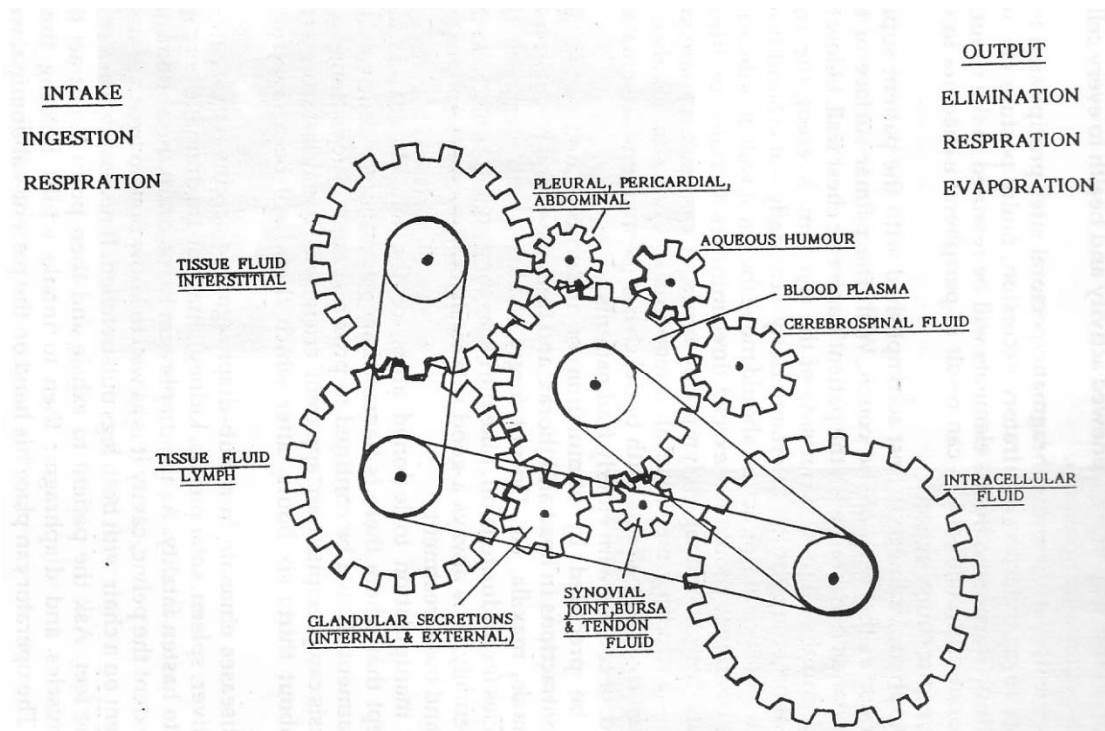


Fig. 13: Body Fluids

I conclude from this study that diabetic patients who deal with their disease seriously should undergo osteopathic treatment at least every two months over a long period. This, together with diet, drugs, and much physical exercise, can slow down the course of the disease.

By no means does osteopathy take the place of diet and drugs. Osteopathy accompanies and supports the patient in dealing with the disease.

Now I want to discuss the results from the structural, visceral, and cranial point of view. It is clear that these areas cannot be separated from each other in the treatment.

5.2. Discussion from the Structural Point of View:

The body presents itself best through its structure. To me, however, the structure is not the first criterion. This is absolutely not objective, of course. I have experienced time and again, however, that the visceral influences structure more than the other way round. Nevertheless, it is necessary to create vitality and mobility in the soft tissues (e.g. psoas muscle), the vertebral joints, and the costal articulations.

The bar chart of muscle and fascia shows that the left psoas muscle is affected in 80 % and Treitz's fascia in 100 % of the patients.

The bar chart of the vertebral column shows that the transitional zones between the vertebral regions are strongly affected. The vertebral segments Th7 and Th8 are more often affected, too, as the nerves supplying the pancreas arise between Th5 and Th9.

Structure, viscera, and cranium are interrelated through neural and lymphatic supply.

5.3. Discussion from the Visceral Point of View:

It is remarkable that dysfunctions in the organs were dominant on the left: left kidney 90 %, left flexure of the large intestine 90 %, left suprarenal gland 70 %. Liver, small intestine, and sigmoid were often in a state of dysfunction, too. The pancreas, of course, showed dysfunction in 100 % of the patients.

The general visceral impression was slowing down, adhesion, induration. The most remarkable thing was a sense of induration in the area of the tail of the pancreas. What can be sensed here seems to be the amyloid deposits, which have been discussed in chapter 2.3 "Pathophysiology", page 25.

In the first patient a change in the softness of the tissue already struck me during motility perception. This recurred in all probands. These findings are completely subjective, but I asked myself: “What will happen if I keep my hands and my concentration on this induration during the motility of the pancreas and additionally include the body fluids?” The answer is absolutely subjective, but the induration changes towards softness.

“[...]when you are reading those tissues, you stay within the tolerance available for diagnosis and treatment [...] “. (Becker,1997, 104)

“Our bodies are a dynamic flux of energy operating from the moment of conception throughout life, and within these energy fields are moments of time – moments of Stillness within these energy fields, fulcrum points in time for various physiological needs – and all centred with the Potency of Stillness as the motif power for the action that follows. We must understand the mechanism of this Stillness and use it in the care of our cases, [...]” (Becker, 1997, 31)

“According to Mc Catty, free circulation of the fluid is as important in physiology as unimpeded oil flow is in a car motor. If the oil passage is blocked, the machine will not work efficiently. With the body it is similar.“ (Liem, 1988,189)

5.4. Discussion from the Cranial Point of View:

Still describes the cerebrospinal fluid as the “highest known element“ in the body. Diabetes patients are not excepted either. The chart of the sphenobasilar synchondrosis shows dysharmony in 100 % of the patients. The hypophysis – the superior system to the hormonal system - lies in the Turkish saddle.

For a further thesis on this subject I would suggest a study that includes this to a greater extent in the practical work.

Involuntary filling and drainage of the ventricles, permanent production of the liquor, this motility must influence anybody, healthy or ill.

Becker speaks of a transmutation process during the “still point“. The exchange of all body fluids takes place during this period. The rhythmic rocking of the liquor ensures the exchange with the choroid plexus, the neurons of the central and peripheral nervous systems, in fact with all fluids and cells of the body. After all, the cranial rhythm can also be felt in the periphery.

This motility means vitality – a permanent cleansing mechanism that should also positively influence metabolism and therefore diabetes. Maybe this cleansing mechanism also has an effect on the cell receptors (“Pathophysiology”), which would then become more sensitive to insulin. This is just an idea and not an assertion.

5.5. Discussion of the hypothesis:

Does Osteopathy influence diabetes mellitus type 2?

It is obvious that osteopathic treatment has a positive effect on diabetes mellitus type 2. An enhancement of mobility is usually accompanied by a better cell interchange. Every agglutination of tissue, such as the bursa omentalis or the Treitz`sche fascia, every tense ligament, for example the ligamentum trigonum of the liver, the ligamentum phrenogastricum, and every blocked vertebra impairs the harmonic flow of the body fluids. If the osteopath unblocks a blocked vertebra of the area Th5-Th9, he or she enhances the elasticity of the vegetative nervous system, which supplies the pancreas. In other words, the nervous system can quickly and unhamperedly convey cell information to the target organ pancreas and to the brain.

If the osteopath relaxes the diaphragm, the rhythmical dynamics get more impulsive. The pump of mobility for the abdominal cavity as well as for the thorax cavity leads to better activity and liveliness. The blood supply is positively affected. Due to the relaxed diaphragm, the points of transit of the vena cava and the aorta get more flexible. The blood flow is unrestrained and there is no venous backpressure at this point. The dynamics of the diaphragm also reach the portal vein, which transports the venous blood more quickly to the liver. Thus, pancreas, liver and stomach profit from a relaxed diaphragm. Due to a free diaphragm, the extension of the motility of the pancreas in all directions is positively affected.

Distension of the suspension of the flexuraduodenajejunalis gives more scope to the intestinal network-plexus. Mobilisation of the radix mesenterii makes the ingestion in the small intestine more efficient. If the pancreas is able to position itself well in the fascial bed, and if the surrounding tissue works without tension, the impression of the motility improves immediately.

The results of the measurement of the bloodsugar support the hypothesis. In the osteopathically treated study group the deviation from the standard limit of HbA1c decreased by 33,67% more than in the control group from t0 to t1, by 25,9% more to t2, and by 20,92% more to t3. In this study, with this probands and controlgroup, Osteopathy influenced diabetes mellitus type 2.

5.6. Relevance for the research:

In spite of checking a small probands group and control group there is a result. This result shows some promise. Improving mobility and motility of the pancreas and its connected systems brings a better result of the measurement of bloodsugar. I think the earlier the osteopath sets in treatment the better results would be. It seems to me much more difficult to treat, if diabetes is very marked and lasts for a long time. The steady remembrance of the body to its health could keep a good HbA1c level. Osteopathic studies which deal with patients immediately, when the diagnose of diabetes exists, should be aimed. Remembrance for the pancreas and its surrounding on health could be a solution. The dialog of the body fluids that carry hormones, all chemical stuff is forced by osteopathic treatment. The organs and tissues which are involved at diabetes can be influenced well. But it doesn't stay. The patients have to be treated again and again. Therefore studies of long term should be done.

In my study patients with diabetes mellitus type 2 could be influenced well by osteopathic treatment. Tissue, body fluids, chemical dialog could be improved.

I believe that studies which set in very early at diagnose of diabetes, and studies where patients are treated for a long period of time would bring much more better results.

5.7. Discussion of literature:

At this chapter I'll give a few statements about the literature that supported my study.

Berger(1995), Krück(1988) and Waldhäusl(1993) gave me the possibility to understand the diabetes type 2 in its principles. The combination of pancreas, liver, vena portae system, muscles and brain comes clearly out of all three books. All three authors describe the disease of diabetes not only as B-cells destruction but also as a reduced distribution of Insulin with less effectiveness at the organ of aim to absorb glucose.

Berger(1995) reports additional about an increased and diminished function to stop hepatic production of glucose in spite of hyperglycämie.

An article of Siegel, Jakobs, Riemann(2001) points out the combination of liver diseases(especially liver cirrhosis) and diabetes mellitus. 15-20% of patients with liver cirrhosis develop a diabetes mellitus. In this report the disease of liver is supposed first and the diabetes mellitus is seen as a consequence.

Lecube, Hernandez, Genesca et al.(2004) compared Hepatits C Virus infected (HCV+) people with patients with other liver diseases but HCV- and diabetes mellitus type 2. The HCV+ patients develop three times more likely a diabetes type 2 than HCV- patients.

Berger (1995) and Waldhäusl(1993) further engage in the consequences of diabetes type 2 (such as eyediseases, destruction of the nerval tissue and vascular tissue very complete.

Krück(1988) deals with the indeed process of diabetes short and pregnant, and not with the consequences of the disease.

Very interesting for me is the article of Fridyland, Philipson(2006). Several experimental studies showed, that free radicals lead to insulinresistance. They speak about oxidative stress , which is a dysbalance between free radicals and antioxidantien.

Zimmet, Alberti, Shaw (2001) publised an article of diabetes epidemic. Diabetes mellitus type 2 is one of the main threats in human health in the 21st century.

The most important literature for me was the osteopathic book of Becker(1997). It changed my way to treat patients completely. Becker learned me to listen to the tissue, to follow the tissue , to follow the wisdom of the tissue, to stay a step behind to watch tissue like a butterfly. I am very thankful for the production of this book.

Stone(1996) shows how easy and simple embryonic information can be demonstrated with almost childlike drawings, as well as relative simple text, in spite of the very complicate embryonic stuff.

As a summary I have to add positive that I am astonished how complex diseases are explored. Chain reactions are described. It is the same way an osteopath tries to feel, think and see. A disease cannot be restricted to one organ.

To complete the discussion a negative statement must be remarked. A lot of literature can be found of the consequences of diabetes mellitus (such as eyedisease, vascular destruction, ...), but very less literature I could find of the beginning of the disease. To my opinion it is more important to research the beginning of a disease, rather than the description of after effects.

6. Abstract

Does osteopathy influence diabetes mellitus type II?

The aim of my study was to examine whether holistic osteopathic treatment can positively influence diabetes and whether mobilization – most subtle mobilization, at different levels, is beneficial to the metabolism.

Methodology: 10 probands were treated 6 times with intervals of 4 weeks. The HbA1c-value (4 times with intervals of 12 weeks) and the fasting blood-sugar value (3 times with intervals of 12 weeks) were measured. 10 patients who were not osteopathically treated served as a control group.

Results: The blood-sugar value decreased in both groups. The study group improved by 33.67 %, 25.9 %, and 20.92 % more than the control group did. The measurements were carried out at different times.

Common dysfunctions showed in the transitional zones between the vertebral regions and in the middle thoracic spine (innervation of the pancreas). The left side was particularly affected in the visceral area (left kidney, left suprarenal gland, left flexure of the large intestine). Liver, small intestine and, of course, the pancreas (100 %) were often affected, too.

The cranial system showed dysharmony at the sphenobasilar synchondrosis in 100 % of the probands. A further study on this subject that includes this to a greater extent (the hypophysis lying in the Turkish saddle – the superior system of hormone production) would be useful.

Discussion: The results might have been influenced by the patients' stricter keeping to diet and taking of medicaments. I did not, however, encourage them to do so.

The best improvement towards the standard limit was achieved between the first and the second HbA1c-value measurement. The patients were treated 4 times during this period and 2 times between the second and the third measurement. Between the third and the fourth measurement no treatment was carried out. The results worsened again during this period. This means that the effect of the treatment does not last.

The assumption that improvement of mobility positively influences the metabolism has been confirmed. Mobilization of the body fluids ought to improve the dialogue between the chemical processes and between blood-sugar decreasing hormones and blood-sugar increasing hormones.

Better results can probably be achieved if the osteopath (recollection of health) and the patient (keeping to the diet, exact medication, sports) work in the same direction.

By no means does osteopathy take the place of diet and drugs. Osteopathy accompanies and supports the patient in dealing with the disease.

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Definitions

Amyloid: an abnormal protein built up in the organism

Ecoute: „local listening“ at a tissue or an organ by using the hands

Fulcrum point: point of balance

Motility: inherent motion of an organ

Meso: folds in the peritoneum, only in the area of digestive organs (e.g. mesocolon)

Primary respiratory rhythm: relatively autonomous, involuntary motions of the central nervous system

Still point: that point in time of stillness, rest, motionlessness that is necessary to change from one direction of motion to another

Thoracocervical diaphragm: upper aperture of the thorax, the boundary of which is formed by the first ribs, scalenus muscles, pleurae, clavicles

Treitz's fascia: a fascia between large intestine, pancreas, duodenum on the one hand and the two kidneys on the other hand. The two lateral margins of the fascia are called Toldt's fascia

Medical History

Date:
Patient:
Age:
Address:
Phone:
Occupation:
Family:
Hobbies, sports:
Medical history:
Since:
First time:
Condition before the complaints:
Where is the pain:
What is the pain like:
What makes it better, what makes it worse:
Daytime:
Other treatments:
Standing:
Ecoute: Forewardbending:
Sitting:
Ecoute: Forewardbending:

History: mechanical/traumatic/medicinal/surgical:

General state: fatigue/sleep/nutrition/way of life:

Elimination: intestine/kidneys/bladder/gynecology (cysts, pregnancy, abortion...):

Assimilation: stomach/esophagus/liver/gallbladder:

Cardiovascular system:

Neck/thyroid gland/head (meningitis,...)/otorhinolaryngology:

Psyche (deaths, divorce,...):

Additional examinations:

Diagnosis	structural	visceral	cranial

Diabetic History

Type of diabetes:		
Since when:		
Course:		
Weight:	Height:	Blood pressure:

<u>Possible causes:</u>		
Nutrition:	before	now
Alcohol:	before	now
Heredity:		
Stress:		
Traumata:	mechanical psychical	
Smoking:		

Medicaments:		
Diabetic diet:	since when:	how strictly do you keep to it:
Have you ever lost weight:		
Do you do sports or much physical exercise:	before:	Now:

<u>Additional findings if available:</u>		
Kidney:		
Eyes:		
Neuropathy:		
Vessels:		