Influence of Osteopathic Treatment

on Congestive Menstrual Disorders and

Premenstrual Syndrome

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1. Introduction

1.1. Presentation of the problem

Throughout my practising years as an osteopath I often noticed that young women, having come for osteopathic treatment of some different kind, reported that their menstrual complaints or PMS diminished or in some cases completely disappeared. With the emphasis of the manipulation being in a different area, I saw this as a positive side effect.

Bearing this in mind I became more and more interested in the matter and decided to present my practical experiences systematically in a thesis.

Sarah Wallace liked the idea and in a first supervision we talked things over and she helped me to arrange my ideas.

I decided to limit the scope of the thesis to congestive menstrual disorders and to put it in relation to symptoms of PMS since both sets of problems characteristically disappear when a period starts.

My first look into the relevant literature confirmed my decision. In fact, recent literature always seems to consider congestive menstrual disorders as part of the premenstrual syndrome.

Generally speaking, the medical literature deals with this problem in a very superficial way and treats it just the same. The only book giving a comprehensive overview, a good theoretical basis and a holistic therapeutic approach is a book written by Trickey[1] (1998). Even in osteopathic literature, the subject is only touched upon occasionally. Most of the information I used in this thesis is based on Barral[2] (1993).

[1] Trickey,R. (1998): Women, Hormones & The Menstrual Cycle .St. Leonhards/ Australia: Allen & Unwin
[2] Barral, J.P. (1993): Urogenital Manipulation. Seattle: Eastland Press

It seemed important to treat patients always in the same phase of the cycle, e.g. between day 7 and 10 before the onset of menstruation since the symptoms are most evident at that time and more easy to include in a questionnaire.
In preparing the questionnaires, I came to the conclusion that the symptoms could be categorised in two groups. This way it is more ease to determine which symptoms or groups of symptoms can be influenced and to which extend.
A short case history about the menstruation is part of my normal case history form, the data of which I include in my study.

Since even my gynaecologist could not find any questionnaire suitable for my thesis, I started looking on the internet and in the literature. I could not find a questionnaire but what I did find was a list of groups of symptoms to which I just had to add a few extra details.

The discussion with my patients revealed that most of them don't get enough help in dealing with their specific problem. Most gynaecologists just prescribe pain killers or oral contraceptives. Household remedies often don't help.

If we can prove that osteopathic treatment helps women suffering from these ailments, we could considerably improve the quality of life of these women.

1.2. Hypothesis:

With osteopathic treatment being able to have an impact on different levels of the body we may safely assume that

- dysfunctions causing intensified congestion in the lesser pelvis can be reduced thus causing less pain. This fact should be proven by subjective / individual interviews based on a questionnaire.
- This may also positively influence pain and the symptoms of PMS.

Dysfunction in an osteopathic sense is a malfunction emerging in osseous, fascial and muscular structures as a reaction of the body to internal or external influences.

This definition also determined my treatment approach: Meert[3] (2003)

The osteopath has to find the dysfunction and treat it, instead of using just individual techniques that don't necessarily correspond to the diverse origins and the mechanism of the dysfunction

1.3. Aims:

The aim of this thesis is to prove that a holistic osteopathic treatment and the resulting positive influence on the mechanical, visceral and cranio-sacral functions of the body reduce the premenstrual symptoms and the pain related to these symptoms.

With this thesis I would like to find answers to the following questions:

- Is it possible to improve PMS pain through osteopathic treatment or is there no difference compared to the control group?
- Are all groups of symptoms concerned by the improvement? (see questionnaire)
- Is it possible to define certain specific lesion patterns?

[3] Meert, G.F. (2003): Das Becken aus osteopathischer Sicht München: Urban & Fischer

2. Fundamental anatomical and physiological principles

This chapter contains a classification of dysmenorrhea and PMS, it presents an overview of its main symptoms, causes and epidemiology.
It mainly concentrates on the anatomical and physiological principles underlining the relationship developed by the subject of the present thesis.

What are congestive dysmenorrhea and PMS?

Painful menstrual complaints are called **dysmenorrhea**. There are two distinct categories:

- *Functional complaints* in which the uterus is perfectly healthy but behaves abnormally. This type of problem is called primary or essential dysmenorrhea.
- In those cases where pain is caused by *abnormal medical conditions* we speak of a secondary or acquired dysmenorrhea. The most common causes are endometriosis or chronic inflammations in the pelvic area with different causes[4].

Premenstrual Syndrome (PMS) covers a whole range of symptoms emerging during the days before a menstruation period. It is defined as a set of disruptive physical, psychological and behavioural symptoms which are not caused by any organic disease and which come up regularly during the same phase of menstruation and diminish significantly or disappear completely during the remaining cycle[5]

[4] Trickey, R. (1998): Women, Hormones & The Menstrual Cycle. St. Leonhards /Australia: Allen & Unwin
[5] www.toppharm.ch/ratgeber/krankheitsbilder/746.html 26.01.01

Classification of dysmenorrhea and PMS is not consistent.

In some publications dysmenorrhea and PMS are treated as two different types of disorders with two different sets of symptoms [6] [7], whereas in others primary dysmenorrhea is defined as being one symptom of PMS, for example Berninger-Schäfer and Larbig[8] (1996).

These authors describe primary dysmenorrhea as follows:

- ***Spastic dysmenorrhea*** *: pain starts on the first day of a menstruation period with severe cramps accompanied sometimes by nausea, vomiting and fainting. Pain is restricted to the area of back, thighs and lower abdomen.*
- ***Congestive dysmenorrhea*** *: is part of PMS and begins approximately 4 days before a period. Congestive dysmenorrhea manifests itself with a feeling of heaviness and dull pain in the lower abdomen or other parts of the body such as chest, back, thighs or joints. Very often bodily symptoms are accompanied by emotional symptoms such as lethargy, depression and general irritability.*

These two types of primary dysmenorrhea may appear together which means that the dull, congestive pain may at times be accompanied or even masked by the sharp pain of spasms.

Trickey[9] also assigns primary dysmenorrhoea to PMS.

She uses the term PMS in order to describe emotional and physical symptoms and divides them up into five groups, in the same way Abraham[10] (1980) had originally done.

[6] Greer, I. Cameron, I. Kitchener, H. Prentice, A. (2001): Obstetrics & Gynecology. London: Mosby
[7] Stamm, H.E. Stamm, H.(1987): Leitfaden der praktischen Gynäkologie. Landsberg: Ecomed
[8] Berninger-Schäfer, E. Larbig, W. (1996): Menstruationsschmerz. Stuttgard: Schattauer S.10
[9] Trickey, R. (1998): Women, Hormones & The Menstrual Cycle. St. Leonhards /Australia: Allen & Unwin

His grouping, dating back to 1983, is based on hormonal, biochemical and/or food related causes for PMS which he subdivided into the following five groups.

1. **PMS – A (A = anxiety):** feelings of irritability and nervousness prevail
2. **PMS – C (C = cravings):** accompanied by hypoglycemic symptoms and premenstrual food cravings for sugar
3. **PMS – H (= hyper-hydration):** fluid retention is very pronounced
4. **PMS – D (D = depression):** going along with depression and the need to withdraw
5. **PMS – P (P = pain):** increased sensitivity to pain, pain being the foremost problem

Women suffering from PMS may be afflicted by several of these groups of symptoms.
Trickey[11] describes *congestive dysmenorrhea* in the following way:
Congestive dysmenorrhea is extremely common and women feel it as a "dull pain in the background" linked with a feeling of heaviness. The pain may be masked by episodic cramps (spastic dysmenorrhea).
Pain is mainly felt in the lower abdomen, sometimes it can spread to the inguinal region, the back or the legs. Some women describe it as a dull boring pain in the vagina or a bloated feeling in the bowels. Very often it is described as a feeling of "everything will fall out".

Congestive menstrual pain is also characterised by the following: Pain starts before bleeding begins and is lifted once bleeding has set in properly.

[10] Abraham, G.E. (1980): Premenstrual Tension. Curr Probs Obstet Gynecol 3 (12) S.1-39
[11] Trickey, R. (1998): Women, Hormones & The Menstrual Cycle. St. Leonhards/Australia: Allen & Unwin S.225

When bleeding sets in, builds up until the flow of blood intensifies or blood clots are released, and then it tapers off.

2.1. Causes[12] [13] [14]

The exact causes of PMS are still elusive. There are, however a number of possibilities being discussed

- Excess or lack of progesterone
- Disturbance of estrogen release
- Excess or lack of cortisone, androgen or prolactin
- Excess or lack of an anti-diuretic hormone
- Lack of minerals (magnesium)
- Lack of vitamins (A, B1, B6)
- Hypoglycemia
- Excess or lack of prostaglandin
- Excess of serotonin
- Emotional, social or genetic factors

A lot of theories are put forward which is usually done when causes are not clear. These theories are widely discussed with controversial results. A few facts, however, have been established beyond any doubt:
Most women suffer from some kind of symptoms before menstruation and PMS is linked to ovulation and becomes apparent in ovulatory cycles only.
The bleeding itself does not seem to have a part in all this since symptoms persist after a hysterectomy unless the ovaries have been removed as well.

[12] www.toppharm.ch/ratgeber/krankheitsbilder
[13] www.healthanswers.co
[14] www.webmed.com/content/dmk_article_40070

In spite of all these uncertainties I would like to have a closer look at some of the possible causes thereby giving a more detailed account of the five previously mentioned groupings of symptoms[15]

2.1.1. PMS - A (anxiety)

This type of PMS is linked to a *relative imbalance of estrogen and progesterone* with a relatively increased level of estrogen and a relative lack of progesterone.
There are two theories for this imbalance:

- increased production of estrogen leading to an increased availability of noradrenaline in the brain which in turn leads to symptoms such as irritability, aggressiveness and fear
- lack of progesterone inhibiting aldosterone and facilitating fluid retention in the tissue. Another reason may be a disturbance in the area of progesterone receptors which reduces the flow of progesterone to the cells. Higher levels of adrenal hormones caused by stress may block these receptors and diminish their capability of taking up progesterone.

Progesterone receptors are found in all regions of the body linked to PMS (brain, nose, respiratory tract, uterus, skin, breasts …).
One reason for high estrogen levels is the inefficient *processing of estrogen in the liver*, a fact which may be crucial for osteopathic manipulations.
Symptoms for this group are nervous tension, mood swings, irritability and anxiety.

[15] Trickey, R.(1998): Women, Hormones & The Menstrual Cycle. St.Leonhards/Australia: Allen & Unwin

2.1.2. PMS - C (cravings)

Symptoms of this subgroup are often combined with PMS - A. They are related to hypoglycemia caused by a lack of magnesium, a heightened sensitivity to insulin or an imbalance of prostaglandin.
Women were found to test abnormally high in glucose tolerance in their premenstrual phase whereas after the period it was back to normal. The conclusion was that glucose tolerance must be linked to the increase of estrogen during the luteal phase.
The most common symptoms emerging in this group are headache, cravings for sugary foods, increased appetite, heart palpitations, fatigue, dizziness or fainting.

2.1.3. PMS - H (hyper-hydration)

Fluid retention is the most common aspect of this group of symptoms. It is very probably caused by an increased level of *aldosterone* which is responsible for the sodium level and the water balance in the body. It plays a major role in regulating blood pressure and fluid balance. An increase in aldosterone may be triggered by low progesterone levels, high estrogen levels, lack of magnesium, irregularities in serotonin and dopamine levels or stress. Serotonin and dopamine are substances of the brain affecting irritability, nervous tension, concentration but they influence the ability to relax as well.

If the main problem is discomfort in the breasts, the cause may be an elevated prolactin level which in turn is linked to estrogen and dopamine.
The most frequent symptoms are tensions in the breasts, bloating, weight gain and swellings of the lower extremities and the eye lids.

2.1.4. PMS - D (depression)

This type of PMS is accompanied by depression and withdrawal and is linked to a *lack of estrogen.*

Depression, forgetfulness, crying spells, disorientation and sleeplessness are symptoms characteristic of this group.

2.1.5. PMS - P (pain)

The main problem of this category of PMS is an increased sensitivity to pain. An *imbalance in the prostaglandin level* is thought to be the cause. Dietary habits may play a role since prostaglandin levels are known to rise with a heightened intake of animal fat.

The symptoms are pain, a lowered pain threshold and dysmenorrhea.

2.2. Epidemiology[16]

The range of frequency distribution for PMS is rather wide. About 50% – 75% of all women are believed to suffer from PMS with frequency and intensity being very different. More than 30% of these women suffer from pronounced symptoms.

In this context physicians point to the fact that women with marked symptoms – pain being the most distinct one – are considerably restricted in their daily activities eg. decreased work or social performance.

A German study found that 30% of professionally active women per year stayed away from work because of difficult menstruation.

[16] www.intmedcom Dr. Elnekheli

The present chapter describes the different categories of dysmenorrhoea, its causes and main symptoms. We also consider some epidemiological aspects to see how many women suffer from menstrual pain and PMS.

The next chapter will be dedicated to the anatomical and physiological findings again in relation to the subject of this thesis.

2.3. Anatomy of female genitalia[17] [18] [19] [20]

2.3.1. Uterus

The uterus is a hollow, muscular organ situated in the middle of the lesser pelvis.

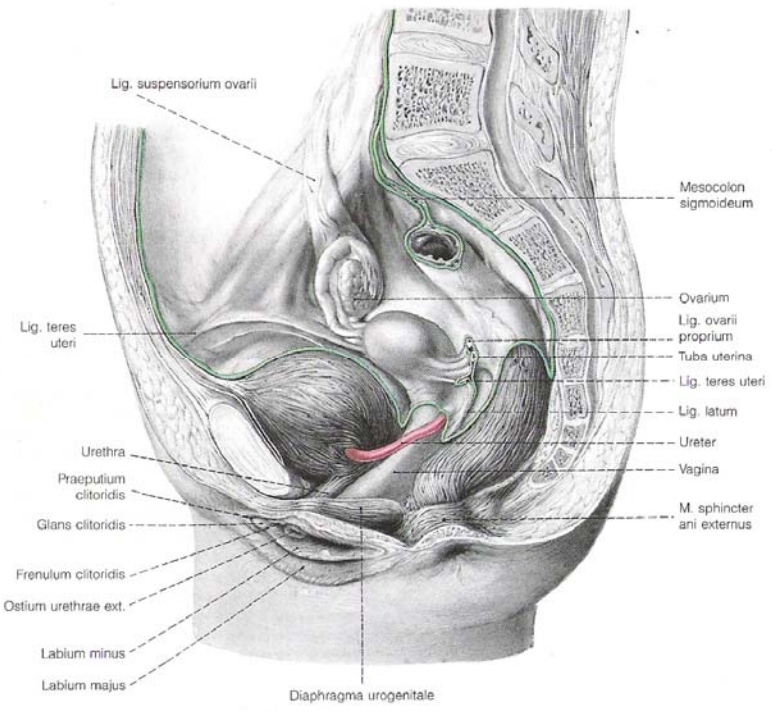


Abb. 1: Female pelvic organs: Drenckhahn, D. Zenker, W. (1994): page 124

[17] Drenckhahn, D. Zenker, W.(1994): Benninghoff Anatomie Band 2. München: Urban & Schwarzenberg
[18] Trickey, R. (1998): Women, Hormones & The Menstrual Cycle. St. Leonhards /Australia: Allen & Unwin
[19] Barral, J.P. (1993): Urogenital Manipulation. Seattle: Eastland Press
[20] Paoletti, S. (2001): Faszien. München: Urban & Fischer

The uterus muscle (myometrium) is one of the strongest muscles of the body. It is able to dilate enormously which is necessary for pregnancy and it has the capacity to contract rhythmically which is necessary to expel the baby from the womb. Intensity of the contractions, however, also influences menstrual blood flow, postpartal bleeding and intensity of menstrual pain.

The uterus is piriform in shape with its apex pointing caudally and dorsally. It measures 7 – 9 cm in length.

The upper two thirds are termed the **body of uterus** (corpus uteri) and the lower third is the **cervix** (cervix uteri). Between the body and the cervix of the uterus there is a slight constriction termed the **isthmus** (isthmus uteri) where the **internal orifice of uterus** (ostium internum uteri) is located. The **external orifice** (ostium externum uteri) lies at the vaginal end of the cervix.

The body of uterus : *corpus uteri*

The vesical or anterior surface, *facies vesicalis*, of the uterus is flattened and covered by peritoneum. In the area of the isthmus the peritoneum is reflected onto the posterior wall of the urinary bladder to form the vesicouterine excavation (excavatio vesicouterina).

The interstitial or posterior surface of uterus, *facies intestinalis*, is convex and is also covered by peritoneum which is continued caudally over the posterior wall of cervix to the rectum to form the *rectouterine excavation* (Douglas's space).

Laterally, in the area of the interstitial portion of the tube, the uterine tubes (tubae uterinae) pierce the wall of the body of uterus.

The uppermost part of the body is known as fundus (fundus uteri).

The cavity of the uterus, **cavitas uteri**, is a mere slit, triangular in shape and standing on its apex.

The close link between uterus, bladder, rectum and peritoneum is crucial for osteopathic diagnosis and treatment. Inflammatory processes in the area of the

peritoneum, for example, may result in adhesions restricting uterine mobility. These in turn may trigger functional disorders including menstrual complaints.

The uterine wall measures 1 – 2 cm in thickness and consists of three layers:

- Endometrium
- Myometrium
- Perimetrium

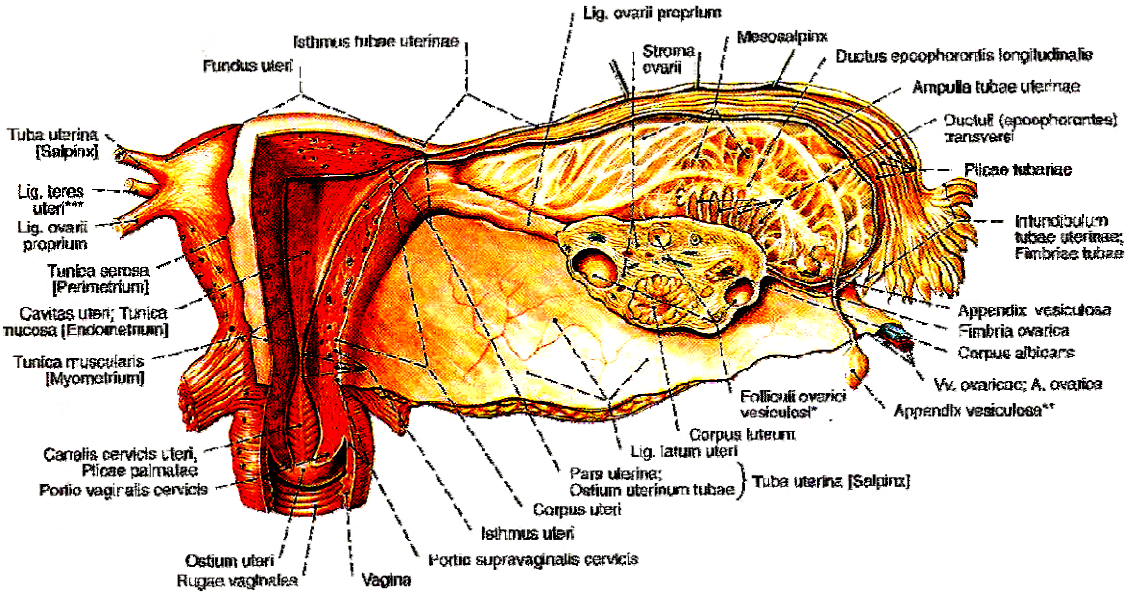


Abb. 2: Female internal genital organs: Putz, R, Pabst, R. (1993) page 193

Endometrium:

The uterine mucous lining is influenced by sexual hormones and subject to a cyclical change of its structure.

Myometrium:

Myometrium is approximately 1 cm thick. It consists of a plexus of smooth muscles and vessels.

Myometrium contracts regularly. Intensity and frequency of contractions depend on hormones. They are increased by estrogens and inhibited by gestagens.

Basic tone of uterus is crucial in causing dysmenorrhea. If tone is normal, contractions happen at a regular rate; they are neither too strong nor too weak and there is a recovery phase between contractions. This muscular activity does not cease, even when the uterus is inactive.

During menstruation or labour uterine activity is heightened, but if the basic tone is normal, contractions and inactive phases do not exceed a normal amount of pain.

The inactive phase is very important. In normal cases blood flow through the myometrium transports oxygen and nutrients, but if the myometrium is not able to relax sufficiently in the inactive phases, there will not be enough oxygen which causes pain. Severity of this pain corresponds to pain described by women suffering from dysmenorrhea.

Perimetrium:

Perimetrium consists of a peritoneal coating and a subserous layer.

The smooth surface enables the uterus to shift freely along adjacent structures.

Perimetrium invests the uterus at its anterior, posterior and cranial surface. Laterally it is replaced by parametrium .

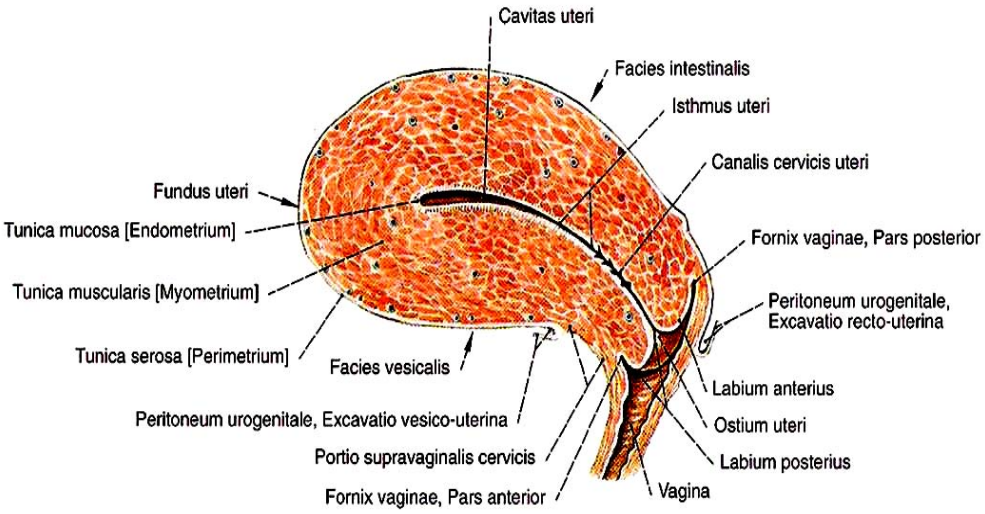


Abb. 3: Uterus and vagina: Putz, R. Pabst, R. (1993) page 194

Suspending and supporting structures of uterus :

There are a number of ligaments and other types of connective tissue which fasten or support the uterus.

Peritoneum:

The peritoneum plays a minor role in suspension but peritoneal restrictions may disrupt uterine mobility.

Lig. teres uteri:

The round ligament of uterus (lig.teres uteri / rotundum) passes on either side of the lateral angle of the uterine tubes (cornua uteri) through the abdominal inguinal ring and along the inguinal canal to the greater pudendal labia. It *anteverts* and *stabilises* the uterus to a small extent.

Lig. latum:

The broad ligament (Lig. Latum) connects the uterus to the lateral walls of the pelvis. The posterior part is in relation with the small intestine. The two broad ligaments form a suspension sling for the uterus. Around the cervix the broad ligament becomes larger and stronger, it is now called parametrium.

The broad ligament contains all the vessels leading to the uterus (arteries, veins, venous plexus, lymphatics) nerves as well as the ureter.

The broad ligament forms the transverse support for the uterus and a cross-shaped structure with the lamina sacro-recto-genito-pubicalis.

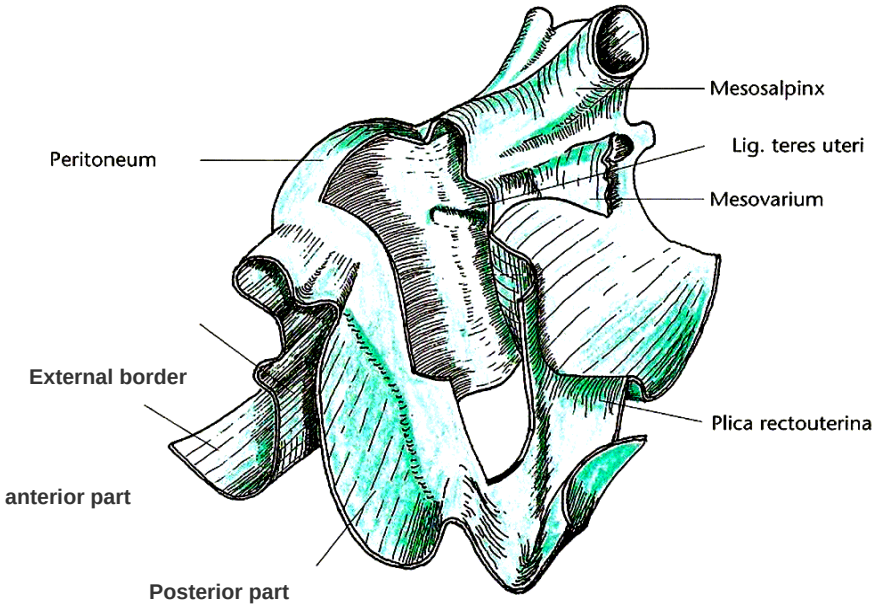


Abb. 4: Lig.Latum (Broad ligament): Paoletti, S. (2001) page 69

Lamina sacro-recto-genito-pubicalis

These fibrous laminae correspond to the *plexus of vessels of the Aa. iliacae internae* / internal iliac arteries. They pass sagittally from posterior to anterior, eg. from the sacrum (anterior area of the for. sacralia) to the posterior side of the os pubis.

The lamina is formed by the following ligaments and structures:

- Pubovesical ligament
- Vesical fascia
- Uterovesical ligament
- Sacrouterine ligament

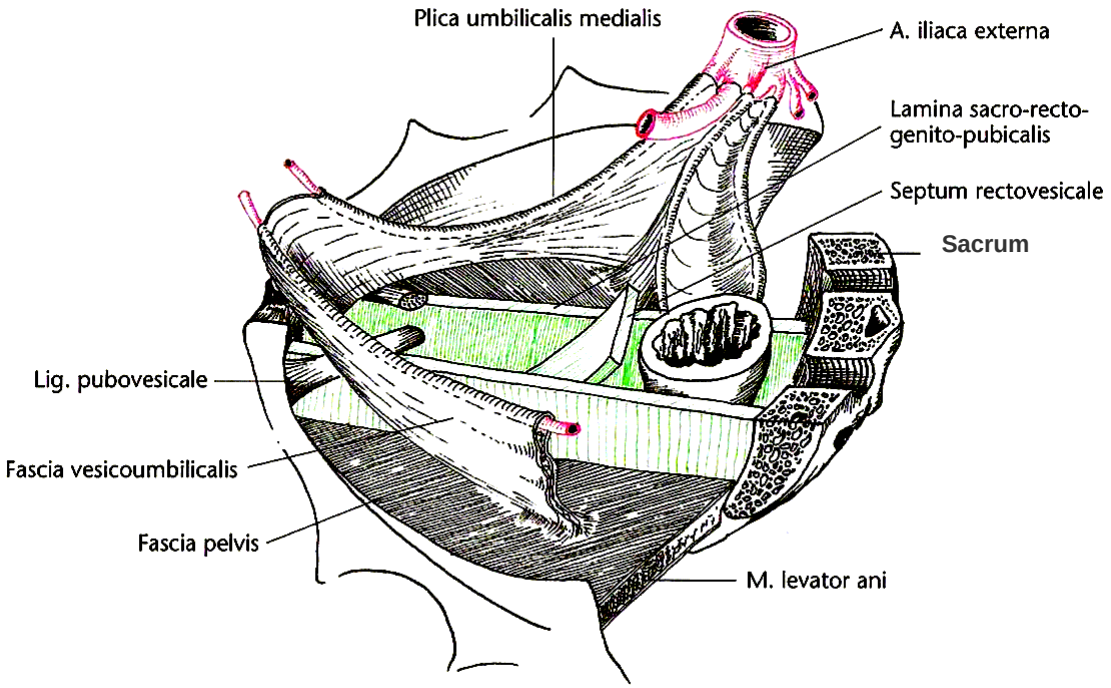


Abb. 5: The fascial structures of the small pelvis: Paoletti, S. (2001) page 68

Pelvic floor:

The *osseous pelvic aperture* is anteriorly formed by the two pubic rami and the symphysis, laterally by the ischial tuberosity and posteriorly by the sacrotuberal ligaments and the apex of the coccyx.

It is secured by a special closing apparatus made of muscles and fasciae.

The closure of soft tissue consists of three layers:

- Pelvic diaphragm
- Urogenital diaphragm
- Outer layer of sphincter muscle (perineal muscles)

The pelvic floor forms the inferior and lateral walls of the pelvis and thus plays a major role in supporting the pelvic organs and distributing the forces within the pelvis.

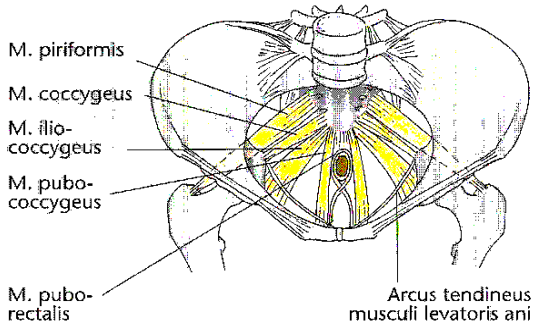


Abb. 6: Schematic view of the upper level of the pelvic floor, from ventro-cranial: Meert, G. (2003) page 16

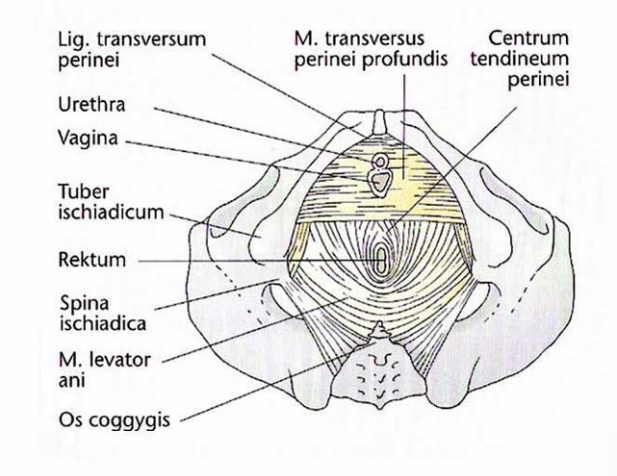


Abb. 7: Schematic view of the urogenital diaphragm, view from caudal: Meert, G. (2003) page 17

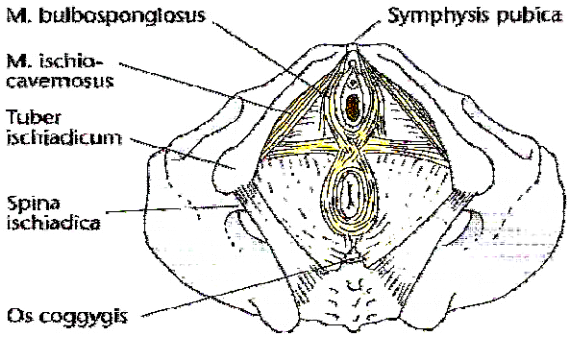


Abb. 8: Schematic view of the lower level of the pelvic floor: Meert, G. (2003) page 17

Cervix: *cervix uteri*

The cervix is the lower, constricted segment of the uterus. The apex projects freely downward into the vagina.

The cervix serves as a *closing mechanism* which, on the one hand, prevents ascending bacilli from reaching the uterus and on the other hand, prevents spontaneous abortion and miscarriage.

Moreover, the cervix is the *central point of attachment* for the supportive structure of the inner genitalia.

2.3.2. Uterine tubes and ovaries:

Uterine Tube: *tuba uterina*

At the ovary the egg cell (ovum) is drawn in the infundibulum (infundibulm tubae uterinae) of the uterine tubes from where it is conveyed to the uterus.

The uterine tube is suspended between the superior angle of the uterus and the ovary or rather the *suspensory ligament of ovary*. Additionally, it is attached to the broad ligament which, in that part, is termed the *mesosalpinx*.

The ovaries are muscular and contract rhythmically in order to transport the ovum to the uterus.

Ovary : *Ovar*

The ovary is the **"female gonad".** It contains egg cells which are released at certain times as fertile ova.

The ovary is also an endocrine gland producing sexual steroids such as *estrogen* and *progesterone* in the first place but other hormones as well.

The ovary controls the cycle and influences the whole body of a woman, particularly through estrogen. Its superimposed system is the hypophysis but the autonomic nervous system exerts some influence on the ovary as well.

The ovaries are roughly the size of an almond. By means of the *mesovarium* they are attached to the broad ligament. With the **proper ligament of ovary** it is attached to the cornua uteri. It contains the ovarian branch of uterine artery as well as smooth and elastic muscle fibres.

The **suspensory ligament of ovary** fixes the ovary to the pelvic wall and the lumbar aponeurosis. It contains the ovarian vessels (vasa ovarica) and the nerves leading to the ovary.

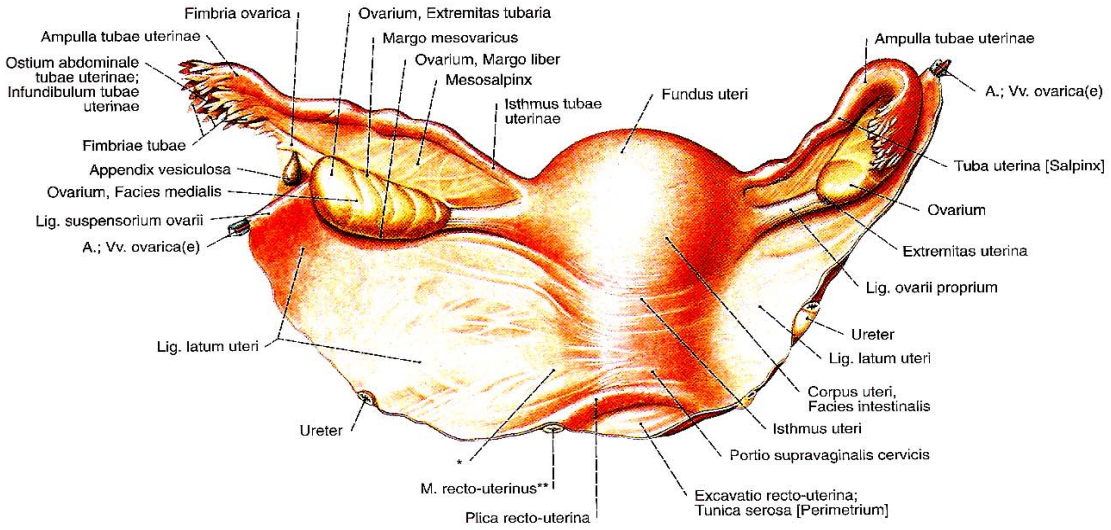


Abb. 9: Internal genital organs in the women: Putz, R. Pabst, R. (1993) page 193

2.3.3. Vagina

The vagina is situated between the urinary bladder or rather the urethra and the rectum. As an acid barrier the vagina protects the inner genitalia and it is a passageway for cervical discharge and menstrual blood.

2.3.4. Arterial supply

There is no central blood vessel supplying the inner genitalia but there are a number of paired arteries anastomosing with each other or with the opposite side through their terminal branches.

Being arranged in this way the vessels almost always ensure blood supply of the inner genitalia.

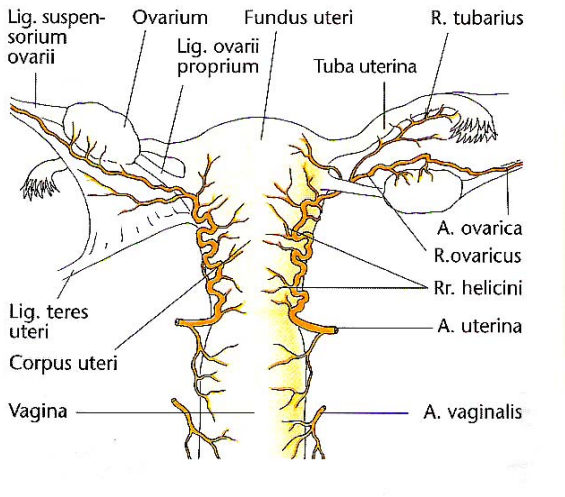


Abb. 10: Arteries of the female genital organs: Meert, G. (2003) page 80

The most important vessels are:

- **Ovary**: ovarian artery, uterine artery
- **Tube**: ovarian artery, uterine artery
- **Uterus**: uterine artery
- **Vagina**: uterine artery, inferior vesical artery, middle rectal artery, internal pudendal artery

2.3.5. Veins

The course of the veins corresponds to that of the arteries of the inner genitalia. Veins are avalvular and form numerous venous plexuses.

There are lateral plexuses to the cervix (*plexus venosus cervicalis uteri*), to the uterine body (*plexus venosus uterinus*) and in the area of the vagina (*plexus venosus vaginalis*). Another venous plexus is situated at the hilum of ovary (*plexus ovaricus*).

Vaginal venous plexus is in relation with the vesical venous plexus. Venous blood of the inner genitalia is mainly drained by the internal iliac vein. Blood flows from the right ovarian vein directly to inferior vena cava, from the left ovarian vein it flows through the left renal vein to the inferior vena cava.

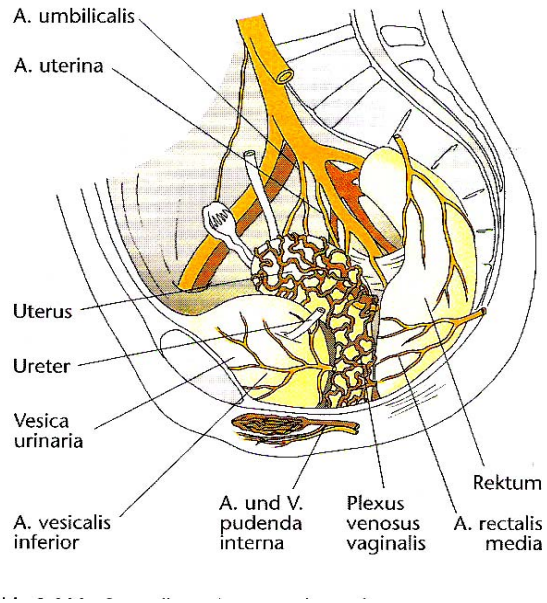


Abb. 11: Circulatory system of the small pelvis: Meert, G. (2003) page 80

2.3.6. Lymph vessels

The lesser pelvis has an extensive system of lymph vessels.

- **Ovary and tube:**

  Lymph vessels in the suspensory ligament of ovary extend with vasa ovarica to the *lumbar lymph nodes* at the level of the inferior pole of the kidney.

- **Body:**

  Lymph is collected in the uterus in an extensive subserous plexus (*plexus*

lymphaticus uteri, *plexus lymphaticus cervicis uteri*). The main drainage of the corpus to the vasa ovarica and the *lumbar lymph nodes* is done via the superior lymphatic vessels. Lymphatic vessels of the anterior side of the uterus pass from the interstitial portion of the tube via round uterine ligament to the *superior inguinal lymph nodes*. The lateral lymph vessels drain into *external iliac lymph nodes*.

- **Cervix:**
  Lymph flows to the *external iliac lymph nodes* or the lymph nodes of the *fossa obturatoria* via the broad ligament.
- **Vagina:**
  Lymph flows to *external iliac lymph nodes* and the *superficial inguinal lymph nodes*.

2.3.7. Nerve supply

Innervation of the inner genitalia is autonomous only.

Sympathetic nervous system:

Cell nuclei for the *sympathetic innervation* lie at the level of the spinal cord at **T10 – L2/3** (data varies). As *splanchnic nerves* they reach the major para-aortal ganglial plexuses (celiac plexus, inferior and superior mesenteric plexus, renal plexus).

Nerve fibres derived from the *superior mesenteric plexus* and *renal plexus* surround the ovarian vessels and form the *ovarian plexus.* They are continued in the *suspensory ovarian ligament* and branch off to the ovary and the tube where they join with branches of the uterovaginal plexus.

Fibres from the *inferior mesenteric plexus* are continued to the *inferior and superior hypogastric plexus* and finally to the cervix and vagina.

Laterally and posteriorly of the cervix they form the *uterovaginal plexus*. From there, fibres run on to the cervix, vagina and uterus.
The sympathetic nerve trunk presents four paired ganglia and an unpaired one (*ganglion impar*).

Parasympathetic nervous system:

Pelvic viscera are innervated by the *sacral parasympathetic nervous system* with the exception of the ovary. Parasympathetic fibres arise from the sacral part of the spinal cord and reach the *inferior hypogastric plexus* and *uterovaginal plexus* as *pelvic splanchnic nerves*. From there they mainly run to the cervix.

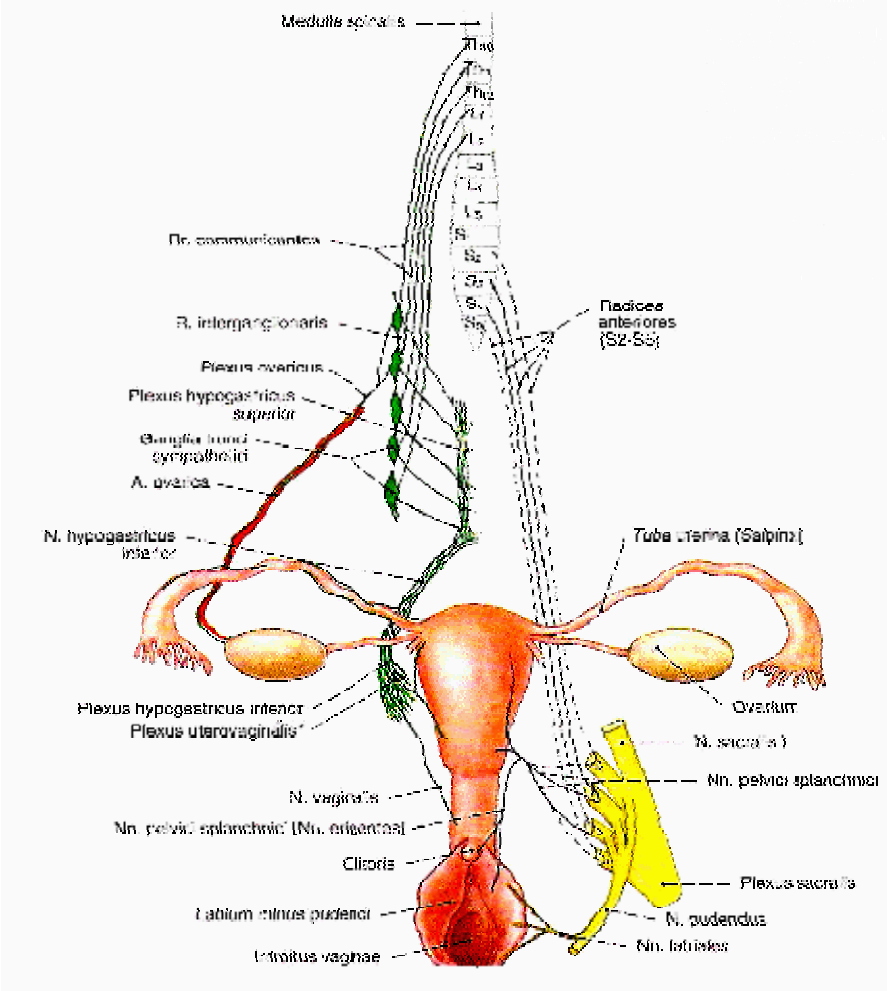


Abb. 12: Schematic view of the autonomous innervation: Putz,R. Pabst, R. (1993) page 213

Afferent sensory nerve supply

Sensory nervous fibres join the autonomic nervous fibres. Sensory fibres from ovary, tube, fundus and corpus reach the spinal cord at level T10 – L1 together with fibres of the ovarian plexus.

Afferent fibres from the cervix and particularly from the orifice of uterus run along the hypogastric and uterovaginal plexuses to T11 – T12.

There are neural relations between the pelvic organs leading to a mutual influence of intestines, uterus, kidney and urinary bladder.

2.3.8. Fascial relations

The fascial system of the body can be likened to a *mechanism of transmission* which may play a major role in causing PMS or congestive dysmenorrhea.

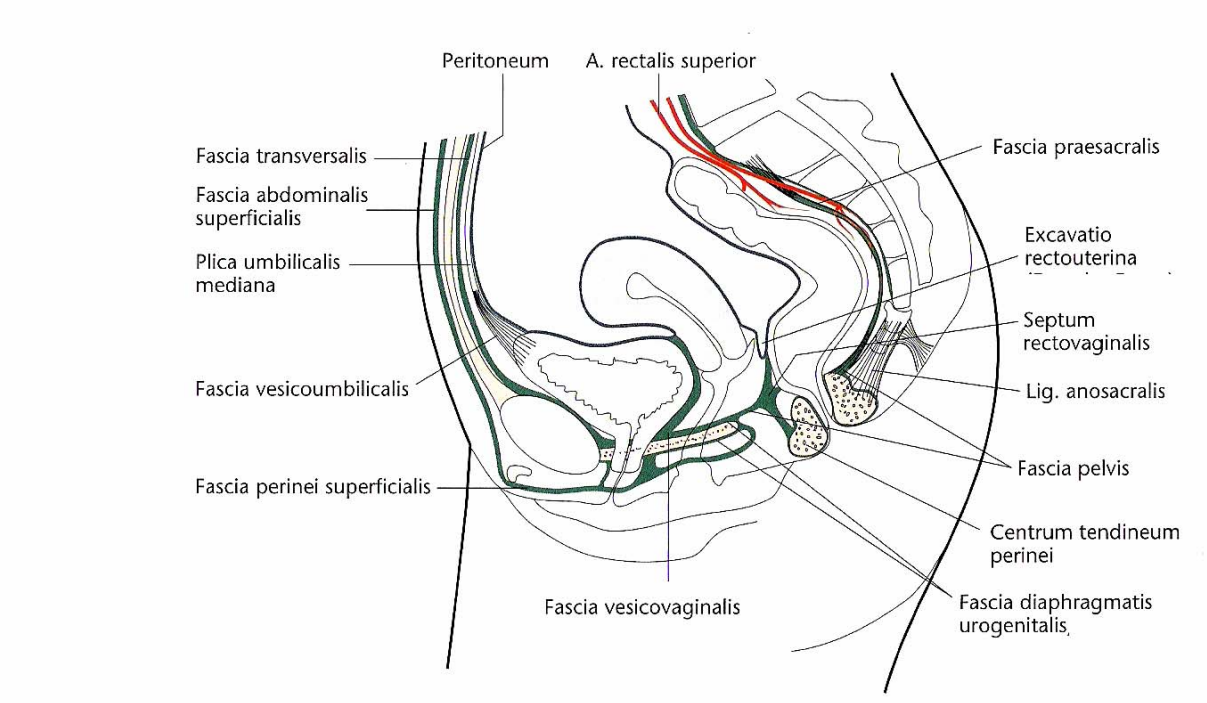


Abb. 13: Fascia of the female small pelvis: Paoletti, S. (2003) page 64

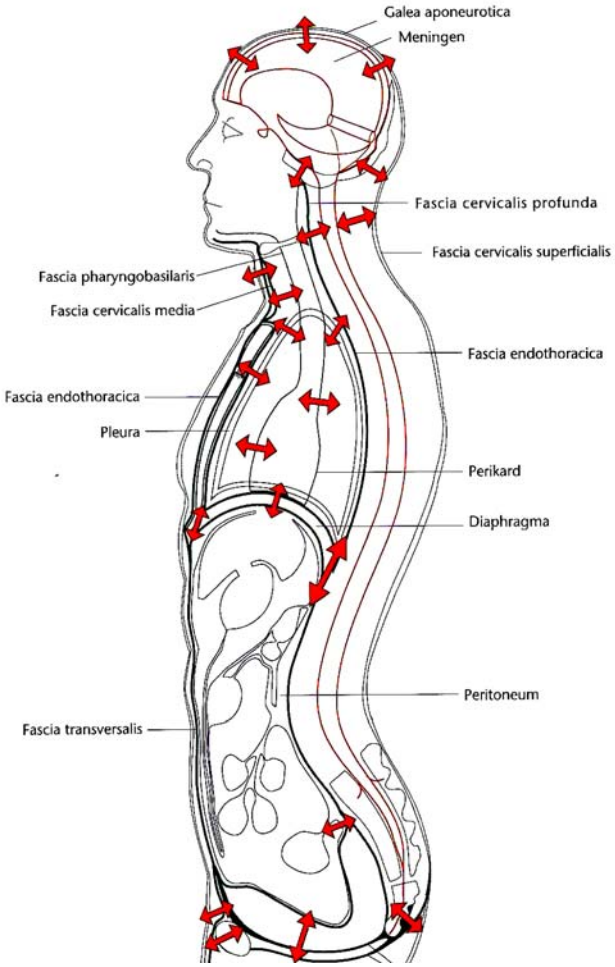


Abb.14: General view of the fascial organisation and their relations: Paoletti, S. (2003) page 111

2.4. Physiology of menstrual cycle[21] [22] [23] [24] [25] [26]

2.4.1. Course of a cycle - Phases

The course of a cycle depends on hormones. *Hypothalamus*, *hypophysis (pituitary gland)* and *ovaries* are the endocrine glands which regulate the cycle and are responsible for hormone production.

[21] Silbernagel, S. Despopoulos, A.(1979): Taschenatlas der Physiologie. Stuttgart: Thieme
[22] Faller, A. (1980): Der Körper des Menschen. Stuttgart: Thieme
[23] Trickey, R. (1998): Women, Hormones & The Menstrual Cycle. St. Leonhards/Australia: Allen & Unwin
[24] Drenckhahn, D. Zenker, W. (1994): Benninghoff Anatomie. München: Urban & Schwarzenberg
[25] h-devil-www.mc.duke.edu/h-devil/women/cramps.htm 1994-99 Duke Unuversity
[26] Liem, T. (1998): Kraniosakrale Osteopathie. Stuttgart: Hippokrates

These endocrine glands function as a unity and communicate with each other by way of a feed-back system.

The **hypothalamus**, at intervals, discharges gonadotrophin-releasing hormone (*GnRH*) which travels through a special vessel system (portal system) to the **anterior lobe of the pituitary gland**. The pituitary gland in turn produces luteinizing hormone (*LH*) and follicle-stimulating hormone (*FSH*) which affects the **ovaries**. The ovaries produce estrogen and progesterone thus influencing the hypothalamus.

If any one of these glands suffers any changes in hormone release, it will immediately affect the next gland of this chain. This is how the *feed-back system* works.

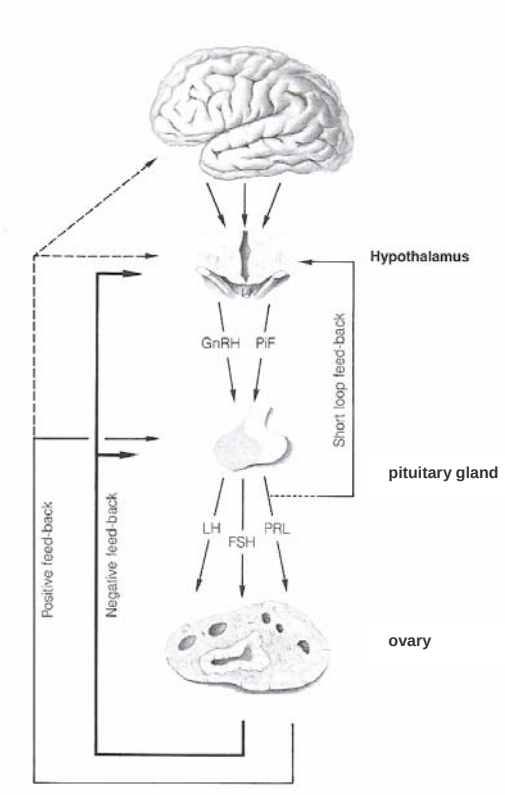


Abb. 15: Feedback-regulation between the pituitary gland, the hypothalamus and the ovary: Drenckhahn, D. Zenker, W. (1994) page 147

First let me describe the five phases of the menstrual cycle: follicular phase, proliferative phase, luteal phase, ischemic phase and desquamative phase.

2.4.1.1. Follicular phase:

In the course of the cycle estrogen levels decline and the hypothalamus releases *GnRH*. This signals to the pituitary gland to release *FSH* in order to stimulate follicle growth.

10 – 20 tertiary follicles grow but one of them only matures enough to become an ovum. The remaining follicles degenerate and at the time of ovulation one ovum is ready to be released.

2.4.1.2. Proliferative phase: (part of the follicular phase)

During maturation of the follicle more estrogen is produced. The endometrium is caused to grow and thicken. *LH* levels increase considerably for 16 – 24 hours before ovulation which in the end triggers ovulation.

FSH levels drop dramatically whereas LH levels decline slowly.

2.4.1.3. Luteal phase: Secretory phase

This phase starts after ovulation has occurred and the follicular epithelium left in the ovary develops into the *corpus luteum*.

Characteristic changes take place in the glands of the uterine mucous membrane (endometrium). These glands and their blood vessels now present a contorted or waved appearance and begin secretion. Increasing progesterone secretion of the corpus luteum causes the endometrium to stop proliferation. Progesterone stimulates secretion of the endometrial glands which proliferated under the influence of estrogen. After an initial drop corpus luteum produces steady amounts of estrogen.

LH keeps up normal functioning of corpus luteum but *LH* production is constantly declining while progesterone levels are rising. If fertilisation does not occur, corpus luteum degenerates after 8 – 10 days after ovulation which is most probably due to the influence of prostaglandin.
Corpus luteum starts to degenerate after two weeks of producing estrogen and progesterone and in this way hormone production declines.

2.4.1.4. Ischemic phase: part of luteal phase

Shedding of endometrium is believed to have several reasons. Firstly, declining estrogen and progesterone levels are held responsible, and secondly, prostaglandin seems to play a major role.
Prostaglandin level rises during the secretory phase which causes uterine contractions to intensify. Spiral arteries which ensure blood supply to the endometrium react to prostaglandin. They begin to contract rhythmically which results in a heavily reduced blood supply (*ischemia*).
The endometrium degenerates.

2.4.1.5. Desquamative phase: early follicular phase

Relaxation of spiral arteries allows blood to flow back into the ischemic area.
Capillary walls burst and endometrial cells fall apart. These, together with parts of spiral arteries, are shed as menstrual blood.
Uterine contractions facilitate shedding of endometrium through the cervix.
At the same time the estrogen level is so low that hypothalamus secretes *GnRH* and the cycle resumes its course.

2.4.2. <u>The role of prostaglandin in causing menstrual pain</u>

Here, I would like to go into more detail about the mechanism causing menstrual pain.

How does prostaglandin cause menstrual pain?

Prostaglandin is a hormone-like substance present in almost all the body cells. It regulates muscle tone of smooth muscles for example in blood vessels, uterus and intestines. If prostaglandin increases excessively, smooth muscles react by a heightening muscle tone and contractions.

Prostaglandin level is very high before menstruation and is at its peak when bleeding sets in.

Thus *uterine contractions* increase and women feel pain and cramps.

The uterus contracts so heavily that its blood vessels are compressed and *blood supply to myometrium* is strongly reduced.

Vigorous blood flow makes for a sufficient supply of oxygen and nutrients – bad circulation results in lack of oxygen and thus leads to pain.

It is also conceivable that the increased prostaglandin concentration goes into blood circulation and influences smooth muscles of other organs or prostaglandin receptors which are present in the whole body. In this way an increased prostaglandin level may also be related to symptoms accompanying menstrual pain such as headache, dizziness, hot flashes, diarrhea and nausea.

2.5. Hormones and the liver

In the liver estrogen is turned into different, less active forms and released into the small intestine via bile.

One part is excreted with the stool, another part is bound to special enzymes (beta-glucuronidase). These enzymes are able to turn estrogen back into a more active form. In this process one part is excreted with the stool and one part is reabsorbed into the blood stream. This process is termed *entero-hepatic circulation*.

Heightened levels of estrogen in the blood occur when excretion via liver and small intestine is reduced.

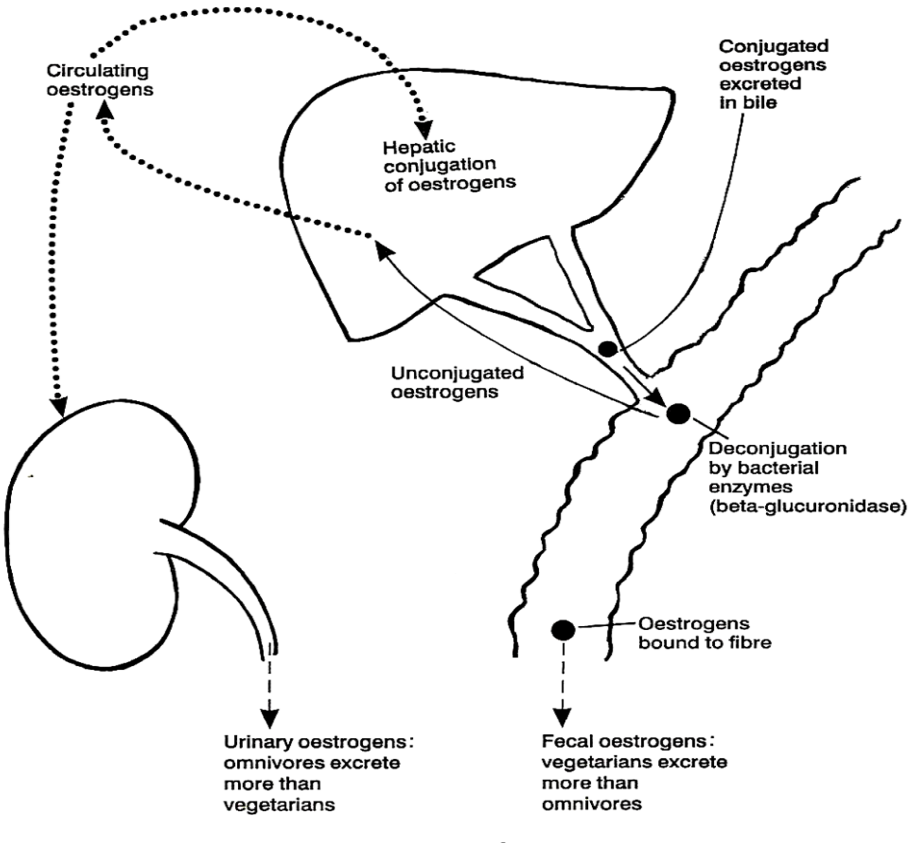


Abb. 16: Entero-hepatic circulation of estrogens: Trickey, R. (1998) page 61

2.6. Hypothalamus – Hypophysis – Third ventricle

The *pituitary gland* (hypophysis) is a vital endocrine gland. It produces numerous hormones which affect incretory glands in a way that hypophysis can be seen as a superior regulatory force for endocrine activity.

Hypophysis itself is regulated by substances built in the *hypothalamus* and reaching it by way of an intricate portal system.

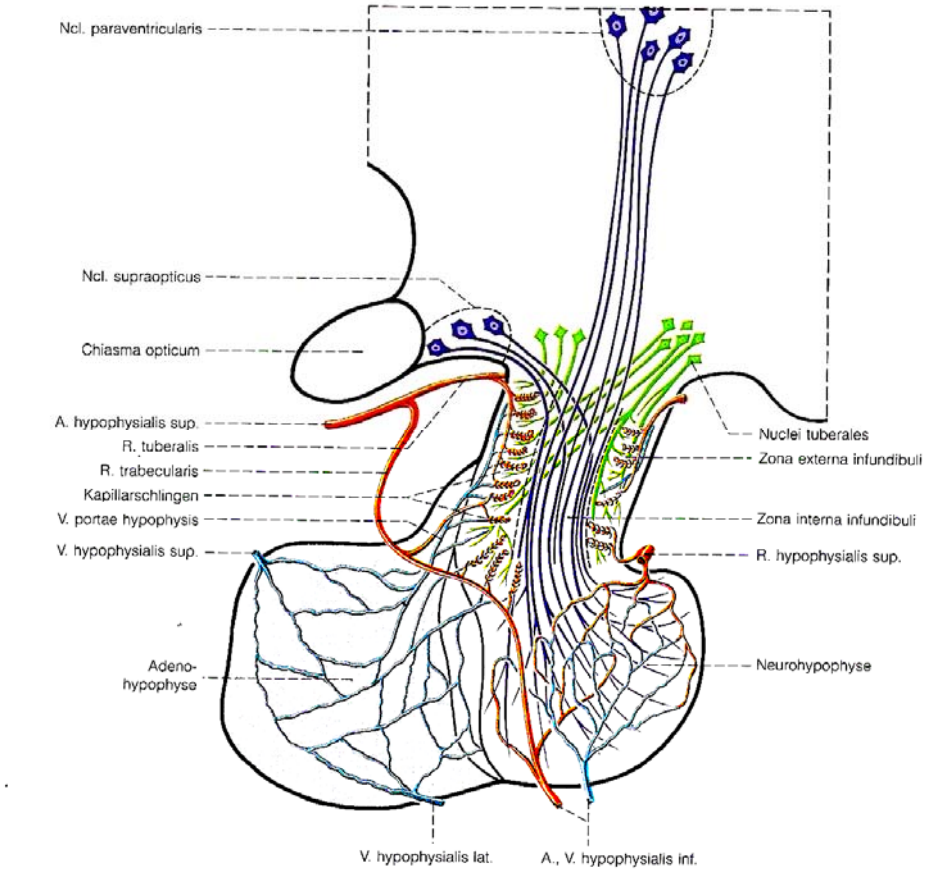


Abb. 17: Portal system between neurohypophysis and adenohypophysis: Drenckhahn, D. Zenker, W. (1994) page 187

In my opinion it is very important to include the third ventricle into this functional unity since, from an embryological, anatomical and physiological point of view the third ventricle is in close relationship with hypophysis and hypothalamus.

Embryologically seen, neurohypophysis develops from a recess of the third ventricle floor which is termed processus infundibularis. This process of the third ventricle later blends into the infundibulum and the posterior lobe (neurohypophysis) itself.

Anatomical importance is given by the fact that lateral walls of the third ventricle are formed above by the thalamus and below by hypothalamus, whereas the floor is constituted by infundibulum and corpora mamillaria. The **physiological** relation is established by the cerebrospinal fluid and by embryolocical and anatomical facts.

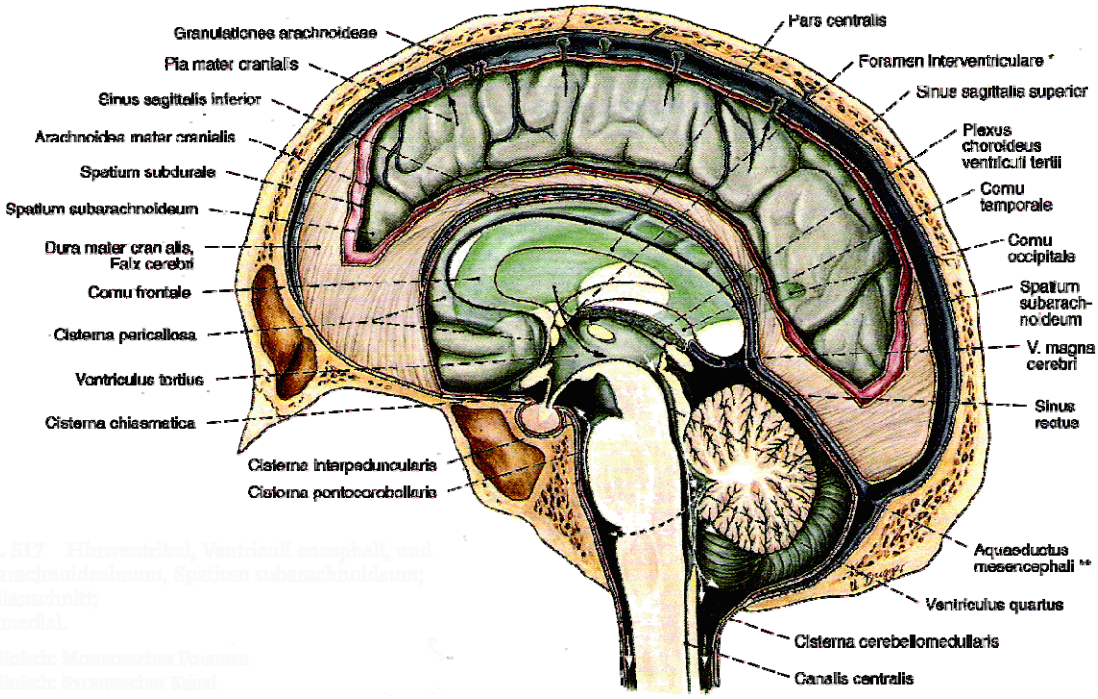


Abb.18: The ventricular system of the brain: Putz,R. Pabst, R. (1993) page 296

The third ventricle is part of the inner cisterns, so are the second lateral ventricles and fourth ventricle.

Cerebrospinal fluid (CSF) is mainly produced in *choroid plexuses*. These are vascular fringed processes of the pia mater reaching into the ventricles. Plexuses are most dense in lateral walls but appear as well in the roof of the third and the fourth ventricle.

CSF is also built in capillaries of the subarachnoidal space in the area of the cranium or the spinal cord but to a much lesser extent.
CSF circulates from lateral ventricles through the interventricular foramen (Monro's foramen) to the third ventricle and from there to the fourth ventricle by way of the cerebral aqueduct (aquaeductus cerebri).
At the fourth ventricle CSF flows through lateral and medial apertures into the outer cistern and subarachnoidal space of spine and skull.
Arachnoidal villi reabsorb CSF which then flows into the intracranial sinus. Good circulation of CSF is imperative for the many functions it has to fulfil. *Transporting hypothalamic and neurohypophysial substances* is but one of them.

3. Considerations and approach to osteopathic treatment

PMS and dysmenorrhea have been of central interest lately. There are numerous approaches, theories and pearls of wisdom, nevertheless, a lot of insecurity remains.

Each person should be seen as an individual. Thus, very likely, every woman has a history of her own as to how her menstrual problems emerged.

Based on this individuality manipulations should be "customised" to each woman.

There are, however, certain regions of the body which deserve being looked at more closely.

At first, it is necessary to get a general overlook of the posture followed by a more detailed examination of certain regions in the parietal system, visceral system and craniosacral system.

In doing so, I would like to take into account the various physiological and anatomical aspects which bear considerable weight in context with PMS and dysmenorrhea.

3.1. Posture:[27] [28]

A good posture is essential for the proper functioning of the body.

A body functions well, if mechanic, visceral and craniosacral mobility is not restricted.

We can define different posture patterns which are used in order to compensate gravity. It is important to analyse them, since they may explain certain compensatory factors and the presence of tension centres which affect the body.

[27] Mitchell, F.L.(1995): The Muscle Energy Manual Volume one. Michigan: Met press

[28] Richard, J.P. (1994): Die Wirbelsäule aus der Sicht der Osteopathie. Kötzing: Verlag für Osteopathie

On the *frontal plane* (seen from the front) relative symmetry of the body is a factor of equilibrium. On the *sagittal plane* (seen from the side) an asymmetric order of structures tends to result in imbalance.
This imbalance invariably establishes either a balance pattern or one of the functional patterns (anterior or posterior).

3.1.1. Basic postural pattern:

The basic postural patterns are the pattern of equilibrium, the anterior pattern and the posterior pattern.

3.1.1.1 Pattern of equilibrium:

There are two factors which cause an imbalance of the body:

- The first factor is the *Transmission of body weight* via sacrum onto the pelvis is done behind the point the hip joint rests upon.
  Thus the pelvis gets into retroversion, the hip into extension and the knee into flexion.
  Body weight on the pelvis can only be kept in balance by flexing hip flexor muscles and knee extensor muscles. Considering the fact that the whole lower extremity forms one unity this means that the foot has to be an integral part of the balance pattern.

- The second factor is the Position of the gravitational centre of the head which causes its flexion. Balance of the head is maintained by constant activity of dorsal neck muscles. Cervical lordosis is then physiologically more pronounced.

This tonic postural activity is intermittent and does neither induce muscles to tire nor nerves to overstrain.

The body as a whole gives an impression of stability and unconstraint; abdominal and thoracic tension is in balance.

The gravitational line which is an indicator for the postural pattern runs as follows, with the patient seen from the side:
It starts trough the external auditory canal and continues to the acromial bone, the body of the third lumbar vertebra, the greater trochanter and passes slightly in front of the lateral ankle.

3.1.1.2. Anterior pattern:

This is the most common postural pattern. The gravitational line runs down in front of the third lumbar vertebra, the hip joints, transverse axis of the knee joint and towards the forefoot.
The body is ventrally off balance and reacts with hyperextension of the knee joint. Body weight is on the area of the forefoot, hip is bent, pelvis inclined forwards. Curvature of cervical spine and lumbar spine is increased.
Heavy tension rests on the lumbosacral junction, the iliosacral joints and T11/12.
Between thorax and abdominal cavity there is an imbalance of pressure.
Diaphragm is low and abdominal organs show a disposition to ptosis.
Posterior myofascial muscle chains keep this postural pattern up.

3.1.1.3. Posterior pattern:

The gravitational line runs behind its physiological points of orientation which means behind the hip and the knees and reaches is lowest point at the heel.
The body is dorsally off balance.
Flexion of the knee, extension of the hip and the pelvis inclining backwards are the results of this posture.

Curvatures of cervical and lumbar spine decrease.

There is a heavy strain on the iliosacral joints and the cervicothoracic junction.

Diaphragm is high and there is an imbalance between thorax and abdomen, because the thorax is slightly drawn in which increases intra-abdominal pressure.

Anterior myofascial muscle chains keep this postural pattern up.

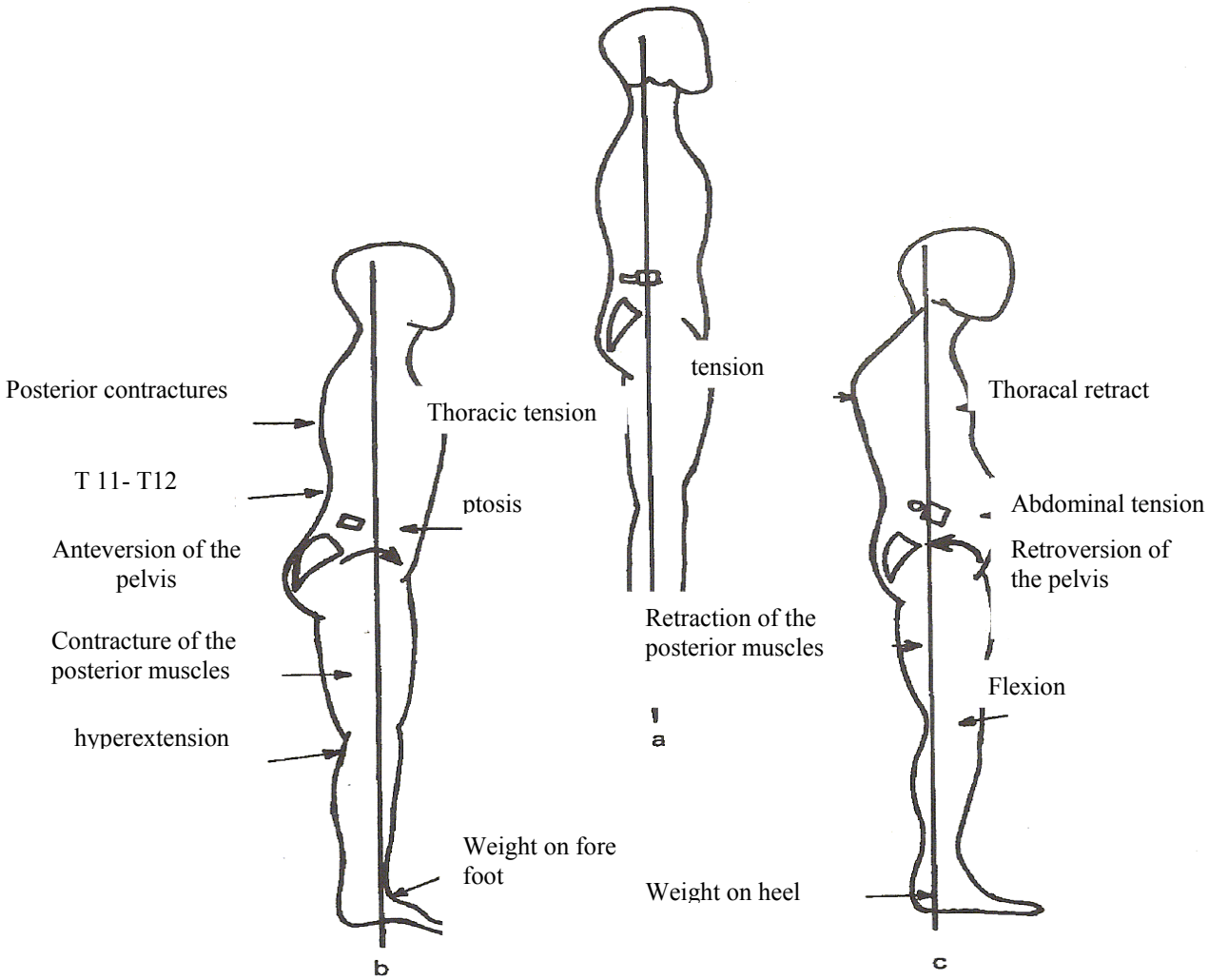


Abb. 19:Basic postural patterns: a. pattern of equilibrium / b. anterior pattern / c. posterior pattern: Richard, J.P. (1994): page 67.

How can these physiological facts be linked to PMS and dysmenorrhea?

Osteopathic manipulations can only be applied, if the mechanisms causing a certain postural pattern have been identified.

Causes may be found both in the parietal and the craniosacral system as well as in the organs.

In the following chapters I will go into more detail about the precise links.

3.2. Possible causes in the parietal system[29] [30]

It is very important to look at the parietal system as a lot of mobility problems start in this area.

The possible causes in the parietal system (musculoskeletal) are the pelvis, special spinal areas and the lower extremities.

3.2.1. Pelvis:

Due to its close contact to the inner genitalia the pelvis has an important function.

Mobility restrictions affect the organs via pelvic floor, on the one hand, and via ligaments and fasciae on the other hand.

Moreover, the close relationship to vessels and nerves play a major part.

Pelvic floor muscles and the connective tissue between the osseous structures build a strong link between the pubic symphysis, pelvic, sacral and coccygeal bones.

This means that each change in position causes a change in tension of the pelvic floor which may prove to be hypotone or hypertone.

[29]. Lason, G. Peeters, L.(1993): Handbuch für Osteopathie: Das Becken OSTEO 2000 b.v.b.a.
[30] Barral, J.P.(1993) : Urogenital Manipulation. Seattle: Eastland Press

Uterus and pelvic floor are linked by the broad ligament (ligamentum latum) which covers pelvic floor but the broad ligament is also in close contact with cervix and vagina.

Another very close relation is established by way of sacro-recto-genito-pubic lamina which runs sagitally and within the sacral foramen from sacrum to pubic bone. It contains the inferior hypogastric plexus which is responsible for the vegetative supply of the genital area.
One very important part of this lamina is the uterosacral ligament building the axis of movement for the uterus.
The uterosacral ligament may sometimes be severely damaged by lesions of the sacrum, whereas iliac lesions are transmitted by the broad ligament.
Another important player is the *coccygeal bone* or rather the *sacrococcygeal joint* for two reasons. It is part of the pelvic floor and the unpaired ganglion (ganglion impar) at the terminal part of the sympathetic trunk holds a key position.

Sacrococcygeal lesions influence the greatest gluteus muscle, coccygeal, levator ani and piriform muscles.
A sacrococcygeal lesion also results in a venous-lymphatic stasis in the lesser pelvis, uterine problems and, because of the close relation to the craniosacral system, in headaches, depression, exhaustion and hormonal dysbalance.

Relations to nerves, arteries and veins:
The most important links to nerves, arteries and veins are represented by the broad ligament, sacro-recto-genito-pubic lamina, suspensory ovarian ligament and presacral fascia.

The broad ligament contains all the vessels and nerves leading to or from the uterus, thus we can safely conclude that changes in tension affect these structures.
Structural changes influencing sacro-recto-genito-pubic lamina may also impair the inferior hypogastric plexus and the ovarian artery.
Lesions of the sacrum may influence vegetative supply due to the close relation of presacral fascia to the sacral plexus.
The ovarian suspensory ligament contains vessels and nerves which supply the ovary.
There is a close link to the broad ligament through the mesovarium which influences the ovary and also a close link to the lumbar aponeurosis.
This is how we can establish a link to the hormonal system which plays a major role in causing PMS. Barral[31] (1993) writes: *"It seems that all local tubo-ovarian manipulations have a general effect on the body".* He believes that local manipulations affect hypothalamus and hypophysis.
So there is according to Barral (1993) a close connection to hormonal regulation which is crucial in the mechanisms causing PMS.
And we can safely assume that changes of tension in the pelvic area provoke changes of uterus tone and thus promote congestive dysmenorrhea.

3.2.2. Spinal regions deserving special attention[32]

Cellular nuclei for the sympathetic nerve supply of inner genitalia lie at level T10 – L3. For the sacral parasympathetic nervous system they lie in the second to fourth sacral spinal segment and reach down to S5 and in the first coccygeal segment.

[31] Barral J.P.(1993): Urogenital Manipulation. Seattle: Eastland Press 1993 page 219
[32] Barral J.P.(1993): Urogenital Manipulation. Seattle: Eastland Press 1993

From experience we know that lesions of the uterus are often linked to restrictions in the area of T12 and L1, L5/sacrum and sacrococcygeal restrictions, whereas spinal restrictions at T11, L1, L3 and sacrum affect ovary and tube.

Lesions in the **lower thoracic spine and lumbar spine** usually lead to vasodilation in the lesser pelvis.
Restrictions in the **thoracolumbar junction** may influence the genital region not only via vegetative nervous system but also because of its close link of the diaphragm in this area.
Thus, a change of tension in the **diaphragm** may also result in a change of pressure balance between thorax and abdomen.
If diaphragm is low, abdominal organs show a disposition to ptosis which in turn exerts more pressure on the organs of the lesser pelvis.
If diaphragm is high, thorax is drawn in, intra-abdominal pressure increases and pressure balance between abdomen and lesser pelvis is upset.
The diaphragm additionally plays a key role in proper arterial and venous function and may, due to its close relation to organs, damage their function. Liver, kidney, small intestine and colon are of major importance for PMS and dysmenorrhea.
The diaphragm is also responsible for the mobility of pelvic organs since they move synchronically along with inhalation and exhalation.
Restrictions of the **middle and upper thoracic spine** may be significant in the clinical picture of PMS and dysmenorrhea because of the relation to the autonomic nervous system and because they may induce changes of the intrathoracic pressure and postural changes.
Pericardium presents a close link between thorax and the fasciae of the *middle body axis*.

This chain of fasciae continues upwards and connects to the cranial base which, in turn, is closely linked to the hormonal system.
Fascia of the middle body axis is also connected to the meninx (via cerebral nerves), to deep and middle cervical fasciae and consequently to thyroid, thymic space, pleura and endothoracic fascia as well as to the diaphragm.
Firstly, the **upper cervical spine** is connected to the sacrum and coccyx by the dura mater and lesions in this area may, via dura mater, affect high cervical lesions.
Secondly, the upper cervical spine is connected to the cranial base and the *hypothalamus - hypophysis* system. Nevertheless, cranial restrictions are invariably reflected at this level.
The upper cervical spine has a very important function as a compensatory mechanism for ascending lesion chains because this area ensures that horizontality of the eyes is maintained.
Restrictions at level **C3/4/5** may be linked to the diaphragm and the peritoneum via phrenic nerve. The close relationship of diaphragm and peritoneum to the uterus has already been mentioned.
All these examples contribute to emphasise the significance of the upper extremities.

3.2.3. Lower extremities

The lower extremities, the foot in particular, are a major point of departure for *ascending lesion chains*.
In general, any disturbance in mobility of the lower limbs may be transmitted to the pelvis or the whole body via fasciae. According to Jean Pierre Barral the most commonly affected are navicular bone, proximal and distal tibiofibular joints.

Fasciae of the lower limbs are associated with the thoracic and abdominal fasciae by gluteal fascia which goes over into thoracolumbar fascia.
And by piriform and obturator internus muscles there is a close relation to the superficial perineal and pelvic fasciae.
It is continued over psoas muscle to iliac fascia and in the area of the inguinal ligament to abdominal and transverse fascia.

3.3. Possible causes in the visceral system:[33] [34] [35] [36] [37]

The viscera are very closely connected e.g. if one organ doesn't work properly, its dysfunction will affect other organs.
Possible causes in the visceral system are: the uterus, ovarian tubes, ovaries, bladder, colon, small intestine, liver and kidneys.

3.3.1. Uterus-ovarian tubes-ovaries

Uterus has the following visceral articulations:

- *Superiorly* with peritoneum, small intestine and colon
- *Anteriorly* with peritoneum and via vesicouterine excavation with the bladder. Enteric interloops may slide between bladder and uterus, particularly if the bladder is empty.
- *Posteriorly* with peritoneum and via rectouterine excavation with rectum.
- *Laterally* with the broad ligament and subperitoneal pelvic tissue
- *Inferiorly* cervicoisthmic region articulates with bladder neck, trigone, base of bladder, vagina and perineal elements.

[33] Barral, J.P. Mercier, P. (1997) : Visceral Manipulation I,II. Seattle : Eastland Press
[34] Barral, J.P. (1993): Urogenital Manipulation. Seattle : Eastland Press
[35] Stone, C. (1996): Die inneren Organe aus der Sicht der Osteopathie. Kötzing :Verlag für Osteopathie
[36] Drenckhahn, D. Zenker, W. (1994): Benninghoff Anatomie I,II. München: Urban & Schwarzenberg
[37] Lason, G. Peeters, L.(1993): Handbuch für Osteopathie: Das Becken OSTEO 2000 b.v.b.a

Ovary is associated with the following structures:

- Pelvic cavity
- Infundibulum and fimbriae
- Mesosalpinx and peritoneum
- Vascular system of the pelvis

Position of the ovary depends on the woman's age and activities and may often be linked to the symptomatology.

Very often ovaries lie in the rectouterine excavation behind the broad ligament to which it is attached by a peritoneal fold termed mesovarium.

The ovary is therefore situated posteroinferiorly to the tube and anteriorly of the rectum.

Ovarian tube is connected to the following structures:

- Broad ligament via mesosalpinx
- It is positioned between posterior ovary and anterior round ligament
- Medially it is related to the small intestine, bladder and rectum
- Laterally to iliac vessels, urethra, enteric sigmoid

Due to the above mentioned close links mobility and motility of uterus, ovary and tube may be negatively influenced by restrictions of other pelvic structures.

Particularly because pelvic organs share **a system of tension based on reciprocity** e.g. any one structure suffering too much or too little tension may affect all the other structures as well.

3.3.2. Bladder

Proper functioning of uterus and vagina depend to a large extent on the bladder. Normally, the uterus is anteverted and lies on the bladder. Each change of position of the bladder and each change of mobility is automatically transmitted to the uterus.

Restricted mobility between uterus and bladder may be brought about by adhesions which means that lubrication between these two organs is lowered. Another reason may be **restrictions** because the bladder has, partly or completely, lost the ability to move which influences the uterus.

Important structures in this context are:

The median and medial umbilical ligaments because their lateral parts end in the sacro-recto-genito-pubic lamina. Typically, a restriction in this area leads to an anterior fixation of the uterus.

The pubovesical ligaments, since they are connected to the vesicouterine ligaments (Ligamentum uterovesicale) which help to connect the bladder to the cervix.

The anterior part of sacro-recto-genito-pubic lamina contains the vesicouterine ligaments which blend into the vesicovaginal fascia and the pubovesical ligament.

Obturator foramen and *obturator internus muscle* are closely linked to the bladder. The lateral fascia of the bladder continues as the pubovesical and the uterovesical ligaments. Laterally they melt into the pelvic aponeurosis which also connects obturator internus muscle, levator ani and their aponeuroses. Thus, restrictions of the obturator internus muscle not only influence the bladder – as a result of their specific links – they also affect the suspensory apparatus of the uterus. Fibrous tissue is always found in and around the obturator foramen when patients suffer from bladder problems, particularly bacterial infections.

Restrictions in the area of the obturator muscle always result in anterior restrictions of the uterus and the perineum by way of the bladder.

3.3.3. Colon:

The colon is closely linked to the genital tract. Coecum or appendix is located very close to the right ovary. It could be interpreted as leaning on each other, or else as having a ligamentous link between coecum and ovary.

Adhesions or scarring which result from **appendectomies** play a major part because they may lead to mechanic restrictions in the right ovary or uterus.

Rectum or sigmoid colon are also in close contact with the uterus.

- The posterior side of the uterus is linked to the rectouterine excavation. The uterosacral ligament which is the posterior part of sacro-recto-genito-pubic lamina connects the uterus with rectum and sacrum. It is a relative point of fixation for anteversial and rotational motion of the uterus.
- Uterosacral ligament is fixed to the lateral wall of the rectouterine excavation.
- One very important link to the diaphragm is the rectovaginal septum. This septum goes off at the posterior part of the urogenital diaphragm and is fixed superiorly to the rectouterine excavation (Douglas's space).
- Sacro-recto-genito-pubic lamina and retrorectal fascia can both be traced backward to the presacral fascia. These links may both influence the sacral plexus.

Bad mobility of colic flexures are very common and may affect other regions of the colon through Told's fascia and in this way it may also influence the genital tract.

Moreover, the ribs connect colic flexures to thorax and diaphragm, but to the kidneys and the liver as well. The latter two being very important for proper functioning of uterus and ovary.

Reduced mobility of the colon may be caused by spasm, heightened muscle tone of smooth muscles or by torsion of the peritoneal ligaments.
The smooth muscles may be disturbed by an irritation of the intestines themselves eg. infection, disorder or malnutrition or else by an irritation of the autonomic nerve supply of the colon.
If restriction is caused by the peritoneal ligaments, we have to find out why. The cause may be found in the musculoskeletal system or in links to other organs.
It is an established fact that intestinal problems exacerbate dysmenorrhea, because the intestines are influenced by hormones as well as by the muscular activity of the uterus and vice versa.
Many women are *premenstrually congested* and this heightens the feeling of being bloated and heavy which is one of the symptoms of congestive dysmenorrhea.
Irritable bowel syndrome is classified as a functional disorder. It is characterised by pain in the colon, constipation or diarrhea or nausea and a sensation of fullness all of which may heighten menstrual pain. This, in turn, can increase symptoms of the colon because both have a similar nerve supply. If one organ has spasms the other reacts with spasms as well. With this knowledge uterine contractions can be stimulated via intestines.

3.3.4. Small intestine:

The small intestine is a very flexible structure with the exception of its peritoneal fixation, the root of mesentery.

Due to this flexibility intestinal interloops may slide between bladder and uterus and may cause irritations.
Ptosis is a very common problem. It is caused by postural problems (anterior pattern) and often goes along with an increased pressure of the small intestine on the lesser pelvis. The uterus may react to the increased pressure but peritoneal restrictions may also impair uterine mobility.
The *duodenojejunal flexure* is fixed to the crura of the diaphragm by the *Treitz' arch*. Restrictions in this ligament may influence the tension of the diaphragm which, due to its point of attachment in the thoracolumbar region down to L3 may in turn irritate the vegetative spinal segments responsible for the genital tract.
Another large area of influence goes back to the root of mesentery. It crosses from the duodenojejunal angle to the centre where it assumes a vertical position. Passing in front of L3 it continues diagonally on to the ileocecal angle.
The root contains the supplying vessels, essential for the performance of the small intestine. Tensions in the *ileocecal valve*, a common enough feature, impair the right ileosacral joint altering mobility of sacrum and ilium. Disorders of the small intestine mainly affect the region of T10 to L2.
Problems of the first part of the small intestine, the *duodenum*, always restrict mobility of the upper lumbar spine, mostly between T12 and L1.
The close functional and local link of duodenum and liver and the release of estrogen may also indicate that there is a connection to the *hormonal system*.

It is important to examine and treat the small intestine before examining uterus, ovary and tube, because restrictions in the lower area of the small intestine have an immediate effect on the uterus. Transmission of force on the uterus changes, pressure is transmitted via an abnormal axis.

Parasympathetic supply of colon and small intestine via vagus nerve presents a very important link to the upper cervical spine. Vagus nerve is closely linked to the upper cervical spine and the cranial base whose functioning is vital for the genital tract.

3.3.5. Liver

The liver has a *key function* in processing and discharging of estrogen as has been stated before.

In order to function properly the liver must have good mobility and motility.

Quite a number of structures and organs are connected to the liver and they all may have a negative influence on its mobility.

The liver is in contact with the hepatic colic flexure, the right part of the transverse colon, the right kidney, the superior part of the duodenum, the gastroesophageal junction and the stomach.

There is also a close connection to the diaphragm, peritoneum, pleura, mediastinum, pericardium, bladder and all the vessels passing through diaphragm.

Liver functions are manifold and are essential for the proper functioning of the whole organism.

Thus, *metabolic activities are optimised*, blood flow, lymphatic drainage and bile production and discharge are improved.

The whole digestive tract depends on liver function.

For women in particular, it is necessary to work on the liver because it metabolises hormones which are released to the small intestine via bile.

Work load of the liver is especially high after ovulation because it has to process a high estrogen level.

It is absolutely imperative that liver, gall bladder and duodenum interact well. The liver has its part in the circulation as well and that depends on how well its connection to the diaphragm works.
Pumping action by the diaphragm may be disturbed, if the ligaments linking it to the liver are restricted, therefore changing their position or else by *congestion* in the area of the liver.
If the liver is heavier, due to congestion, or if fixation to neighbouring structures lower the position of the liver, diaphragmal attraction is not working properly. The weight of the liver disturbs the balance and liver and diaphragm separate, thus disrupting pumping action.
A disruption of the venous flow through the liver invariably affects general venous circulation since the backflow of venous blood to *vena cava* is reduced. At the same time venous flow via *vena porta* is impaired which results in a venous congestion in the afferent venous systems.
Portal hypertension can lead to sciatica due to congestion of the venous circulation in the area of rectum / sigmoid which leads to dilatation of hemorrhoidal veins. They react with inflammation that leads to congestion of the sacral region.
At the same time Batson's plexus may be affected by congestion as well. In both cases sciatica develops on the left side.

Apart from this direct link between liver, estrogen level and good venous circulation there are other regions which are affected by a dysfunction and which may have an influence on PMS although indirectly.
Liver problems almost always cause restrictions of the cervical spine at C4-5 on the right side or on both sides. Problems of the gall bladder are invariably found at C4 on the left side.

These dysfunctions may be transmitted to the cervical spine by way of *pleura* and *cervical fascia* or through an irritation of the vagus nerve and phrenic nerve. Through this chain of lesions we may find shoulder problems which have their origin in liver dysfunction.
The more the liver is restricted in its mobility and motility the more the lower cervical vertebrae, the first thoracic vertebra and the first rib are restricted.
That means there is a link to the cervicothoracic diaphragm, the cervical fascia and the cranial base. In this way, in context with liver problems, we always find restrictions on the *right cranial base*. They may influence the function of SBS and the hormonal situation of the body.
In case of liver problems we find restrictions in the thoracic spine at T7-10 and the respective ribs.
In this way not only an increased weight of the liver brought about by changes in the relation between liver and diaphragm but also the affected areas of the spine may change the complete postural pattern.

3.3.6. Kidneys

The kidneys are, in the same way as the liver, *key organs* in visceral-osteopathic manipulations because their functioning is vital the whole organism. Liver and kidneys should therefore always be treated together.
From an osteopathic point of view the *right kidney* corresponds to the digestive tract thanks to its close connection to liver, hepatic colic flexure, the ascending colon and the second part of duodenum.
This is the reason why changes of mobility, motility and position of the kidneys (such as ptosis) may be transmitted to the digestive tract.
Depending on how the digestive tract is influenced the genital tract may be affected as well.

The *left kidney* corresponds to the genital tract. It is significant that restrictions of the left kidney are often linked to restrictions of the left ovary. It is not clear, however, how exactly this mechanism works, but we do know that in some way the blood vessel systems has its part in this.

Venous situation is very important because it is responsible for the venous backflow from the genital tract.
Blood from the left ovarian vein flows into the left renal vein, whereas the right ovarian vein drains directly into the inferior vena cava.
This, and ptosis of the left kidney may change the angle of the afferent ovarian vein rendering venous drainage more difficult and encouraging functional problems. *Cervix* and *ovary* are more often restricted on the left side and, very probably, the cause may be found in this venous asymmetry.
Since backflow does not work properly women feel congested in the area of the pelvis and may even feel pain which is more pronounced during the premenstrual phase of the cycle.
A phenomenon accompanying kidney problems are restrictions at the coccygeal bone. Here again, we do not know exactly how this mechanism works, but it is an established fact that treatment of the kidney releases the coccygeal bone. This being the point of attachment for the muscles of the pelvic floor, there is a clear link to the pelvic floor.
In my opinion, the close contact of diaphragm and psoas muscle with the kidneys is significant.
The two structures are closely related to the thoracolumbar junction.
Tension of the diaphragm may be strongly changed by the kidneys which prevents a balancing of pressure between abdomen and lesser pelvis. The result is an increased pressure on the organs of the lesser pelvis.

Psoas muscle serves as a gliding surface for the movement of the kidneys on the one hand and as a link between pelvis and hip joint on the other hand. Both these functions may have an irritating effect on the organs of the lesser pelvis.

The diaphragm is part of transverse fasciae which are linked by the longitudinal fascial systems. In this way, changes of diaphragmatic tension may be transmitted to other transverse fasciae.

3.4. Possible causes in the craniosacral system:[38] [39] [40] [41]

The close connection between the hormonal regulation and the craniosacral system means that, in order for the hormonal system to fulfil its role properly,a good function of the primary respiratory mechanism is necessary.
The possible causes for dysfunctions are: sphenobasilar synchondrosis, diaphragm of sella turcica, circulation of cerebrospinal fluid and the connection of the cranium with the sacrum via the dura mater spinalis.

3.4.1. Primary Respiratory Mechanism

The craniosacral mechanism is a functional, anatomical system existing in all living beings which have a central nervous system. The craniosacral system emphasises the functional unity of cranium and sacrum in the primary respiratory mechanism.

PRM (primary respiratory mechanism) consists of the following factors:

1. Motility (inherent movement) of the brain and the spinal cord
2. Fluctuation of the cerebral spinal fluid (CSF)

[38] Stone, C. (1996): Die inneren Organe aus der Sicht der Osteopathie. Kötzing :Verlag für Osteopathie
[39] Liem,T. (1998): Kraniosakrale Osteopathie. Stuttgart: Hippokrates
[40] Drenckhahn, D. Zenker, W. (1994): Benninghoff Anatomie II. München: Urban & Schwarzenberg
[41] Paoletti, S. (2001): Faszien. München: Urban & Fischer

3. Mobility of the intracranial and intraspinal membranes
4. Mobility of the cranial bones
5. Involuntary movement of the sacrum between the ilia

The autonomous motion of cerebral tissue and production and absorption of CSF generate a rhythm, feed it and draw from it at the same time. Membranes receive these rhythmic impulses and transmit them to the cranial bones and via dura mater of spinal cord to the sacral bone as well.

Proper functioning of the whole organism is brought about by:

- Continuous motion of cranium and sacrum
- Constant and continuous changes of tension from intracranial and intraspinal membranes to the fasciae and connective tissue of the remaining body
- Transmission of rhythmic pressure changes of the intracranial and intraspinal CFS on extracellular CFS pressure and from this to each single body cell

PRM influences the following:

- Metabolism of central nervous system
- Nuclei of cerebral nerves and vital centres of third and fourth ventricle
- Transport of neurohypophysial substances
- Venous drainage of the brain
- Biochemical and bioelectric balance of the body
- Exchange of body fluids
- Structural and dynamic balance in the body

The craniosacral system is, due to the above mentioned factors, very likely to contribute to lesion mechanisms. Firstly, lesions may take their origin directly in the craniosacral system, secondly, it presents an adaptational mechanism for the whole body which, in turn, may affect the whole organism.

Proper functioning of hormonal regulation depends largely on structures like the sphenobasilar synchondrosis (SBS), diaphragm of sella turcica, circulation of CSF and the connection between the cranium and the sacrum via dura mater of spinal cord as described below.

3.4.2. Sphenobasilar synchondrosis:

SBS is the articulation between occiput and sphenoid. Disturbances in this area affect all the other cranial bones and the mobility of the cranium. They lower craniosacral mobility and block the whole craniosacral system to a higher or lesser extent. And they reflect peripheral, extra- or intracranial disturbances in the form of adaptational mechanisms of the sphenobasilar synchondrosis.

Possible causes of disturbances of the sphenobasilar synchondrosis may be found in:

- Head traumata including birth traumata
- Strong tension of neck muscles beginning at occiput and fascial tension
- Intracranial tension of the dura at its point of attachment at the sphenoid and occipital bone
- Sutural restriction
- Falls on the sacrococcyx
- Visceral dysfunction via fascial chains
- Musculoskeletal dysfunction via myofascial links to the cranial base

All these factors may affect sphenobasilar synchondrosis which always leads to changes in *CSF circulation* and to changes of tension of *intracranial membranes*. Thus, hormonal balance may be disturbed on several levels.

3.4.3. Diaphragm of sella turcica – venous sinus

The hypophysis is imbedded in the sella turcica of the sphenoid. It is covered by the diaphragm of sella turcica presenting an aperture for the *hypophysial stalk* which is the link to the third ventricle. Together with the other ventricles it plays a major role in initiating the craniosacral rhythm (CSR) and fluctuation of CSF. Another significant factor is tension of the *diaphragm of sella turcica*.
The aperture in the diaphragm of sella turcica for the hypophysial stalk dilates in inhalation and shrinks in exhalation. Abnormal tension of the diaphragm of sella turcica is present in all lesions of sphenobasilar synchondrosis and may thus influence hypophysis.
In my opinion this influence is based on the fact that arterial blood supply of hypophysis, a portal system, partly runs along the hypophysial stalk.

This system of portal vessels is the basis for the functional linkage of hypothalamus and hypophysis. Arteries are accompanied by small veins joining larger veins and finally emptying into cavernous sinus.
I am convinced that, if tension of diaphragm of sella turcica changes, it can directly influence the vessel system or else it can indirectly influence the vessel system through the close link of diaphragm of sella turcica and cavernous sinus. The cavernous sinus serves as a connection to all venous sinuses whose drainage is essential for a functioning balance of fluid circulation.
Tension of dural membranes has a great impact on venous sinuses which is not surprising since they are built by dural layers.

3.4.4. Circulation of CSF

Quality of PRM depends largely on production, circulation and absorption of CSF.

The influence of PRM on different systems and links has already been described at length.

3.4.5. Connection via dura mater of spinal cord to sacrum

Cranium and sacrum build a unit which is linked by the dura mater of spinal cord.

Therefore all lesions in the area of the cranium are reflected in the sacrum which influences mobility and motility of the genital tract thus contributing to PMS and dysmenorrhea. The ovaries contribute to the impact on the hormonal system.

A mechanism of lesions may also be brought about in reverse order.

This chapter shows that lesions in all regions of the body may be the origin of a lesion chain ultimately leading to PMS or dysmenorrhea.

The hormonal system and its regulatory mechanism play a superior role in the development of dysmenorrhea and PMS.

4. Methods

4.1. Presentation of the Problem

The patients included in my study were women suffering from PMS and congestive dysmenorrhea.

The problem seems to be affecting younger women who have not given birth yet, more often than others. Therefore it made sense to cover a certain age group.

The *question* is whether osteopathic treatment, within a time period of 4 cycles (4 therapy sessions), can improve symptoms and whether there are symptoms which react more favourably to osteopathic manipulations.

Verification of treatment success is done by means of a questionnaire.

4.2. Questionnaire

The questionnaire I set up is based on the grouping of PMS according to Abraham[42] which I copied from Trickey.[43]

Guy Abraham divides PMS into 5 subgroups to which he assigned main symptoms. I only changed the subgroup "Pain" in order to obtain a more precise characterization of pain and thus improving the statistical assessment (see questionnaire in appendix).

It seemed logical to follow this classification that serves as a basis for the theoretical part of my theses and helps to reduce the numerous symptoms related to PMS to a manageable number.

The questionnaire starts with a short anamnesis of menstruation since this seemed to be sufficient for my purposes.

[42] Abraham, G.E.(1980): Premenstrual Tension. Curr Probs Obstet Gynecol 3 (12) S.1-39

[43] Trickey, R.(1998): Women, Hormones &The Menstrual Cycle. St.Leonhards/Australia: Allen&Unwin

The general anamnesis concerning traumata, birth and so on is done with the anamnesis sheet I usually use in my office (see appendix)

4.3. Choosing the subjects

In my study I included 20 women suffering from PMS and congestive dysmenorrhea.
They were assigned haphazardly to a trial group and a control group. Each group consists of 10 women.
The subjects had to fulfil the following criteria:

- PMS and congestive dysmenorrhoea and their different degrees of severity
- Age group of 18 - 30
- No previous childbirth
- No known current pregnancy
- No contraceptives or intrauterine contraceptive device
- No known organic diseases of the urogenital tract, hormonal disturbances or endometriosis

The women were referred to me by my gynaecologist, the general practitioner I share my surgery with or by word of mouth. I made sure I was not interfering in any other kind of treatment.
All the subjects/patients who were not referred to me by my gynaecologist or who had not had any medical check-up had to undergo a gynaecological examination and, if necessary, had to have a hormonal status done in order to make sure their complaints were of the functional type.
Most patients suffering from pronounced pain symptomatology take painkillers as a preventive measure in order to “function” properly in their work, studies or

family life. They were asked to take painkillers only if absolutely necessary so that changes could be noticed.

4.4. Procedure

All the subjects were asked to come to my office according to the schedule of their cycle. My advisor, Sarah Wallace and I decided that it would be most useful to treat patients in their premenstrual phase which is about 5 days before the onset of bleeding, because this is definitely the time when symptoms are most severe. It does contradict the opinion of experienced osteopaths who prefer to treat women in the first phase of their cycle, right after bleeding has stopped. However, we thought it more advantageous in order to establish the immediate effect on symptoms and to evaluate the long term effect by treatment during 4 cycles.
The trial group consisting of 10 women were treated by general osteopathic methods whereas the control group received no treatment at all.

All the women filled in the questionnaire.

The trial group was treated four times in their premenstrual phase, according to each patients individual cycle and asked to answer the questionnaire once more. The control group was asked to answer a questionnaire a second time after four menstrual cycles.

4.4.1 Procedure – trial group

The patients are asked to come for an interview, they are given all the necessary information and answer the questionnaire before the first treatment.

There is a very thorough and comprehensive medical anamnesis taking into account birth, traumata, accidents, diseases and existing troubles in order to establish a context to existing problems.

Osteopathic examination and treatment

For me it was very important to follow the osteopathic principle of perceiving and treating a person in a holistic way and not just using special techniques and examine their effects. In view of the complex clinical picture of PMS it seemed particularly fitting to do so.

Treatment proper was done according to the results of the osteopathic examination and was therefore "customised" to the individual needs of each patient.

I focused especially on so-called key parts which are in close contact with the hormonal system and the genital tract.

Osteopathic examination comprised the following:

Assessment of erect posture:
- ⇒ Postural type
- ⇒ Asymmetries
- ⇒ Conclusion drawn from an existing problem

Listening in erect posture:
- ⇒ Conclusion to possible visceral, structural or cranio - sacral dominant lesion

Mobility test of pelvis:

Global mobility test of spine:

Assessment in sitting position:
- ⇒ Listening, comparison to erect position
- ⇒ Mobility test of pelvis
- ⇒ Mobility test of spine, ribs
- ⇒ Mobility test of upper limbs, if necessary
- ⇒ Diaphragm
- ⇒ Liver

Comparison of the results in erect and sitting position	
Prone / supine position:	⇒ Mobility test for lower limbs, pelvis, spine
	⇒ Mobility of all structures being linked to the uro-genital tract
	⇒ Mobility, motility of organs
	⇒ Assessment of the craniosacral system

Osteopathic treatment:

Treatment was done individually and according to the results of examinations.

It seemed very important to me to try again and again to link the existing restrictions to the hormonal system and the urogenital tract.

Four treatment sessions were held in the *premenstrual phase* of each patient's cycle.

At the end of the fourth treatment the questionnaire was answered again.

4.4.2 Procedure of control group

The ten women belonging to the control group were informed about the course of action, were given two questionnaires and asked to answer one of them immediately and the second one after 4 cycles.

In addition, a complete case history is carried out to determine any noticeable or striking features.

5. Results and statistical evaluation

My study was based on the hypothesis that four sessions of osteopathic treatment would improve pain symptomatology and premenstrual symptoms. Additionally, a general survey of each patient's menstruation history was conducted, but served as background information only.

5.1. Results of the questionnaire

5.1.1. Part A of the questionnaire – Menstruation history

This part of the questionnaire captures information on the development of menstruation from the first period to the beginning of this study.
The results include all the women participating in the study.

The interviews yielded the following results:

- The time of the first menstruation period for almost all the women was between ages 10 and 14 with a cycle length of 28 to 30 days, very often involving moderate or heavy bleeding.
- Concerning the first menstrual period and cycle length, most women are within a normal range, women with a tendency to light bleeding do not suffer from dysmenorhhea or PMS as often as others.
- Surprisingly, their mothers' attitude was not generally perceived by the patients as an example, which is the opposite of what I expected.
- Half of the women surveyed take pain killers, but there is a distinct tendency towards taking them only as a last resort. If there is a long day at work to withstand, women take pain killers more readily than at week-ends, when they can rest and keep warm.

- There are considerably more women who are affected by PMS in each cycle giving rise to the assumption that exogenous influences (stress, lack of sleep, etc) are not playing such an important role after all. However, 80% of my patients complain of stress in their jobs or in the family.
- Concerning pain, there have been distinct changes in the cycle of all women surveyed, whereas 10 out of 20 women said bleeding and cycle length had not changed.

Talking again to my patients I found out that increasing pain may be encouraged by some kind of behavioural pattern: “I get my period – it will be painful again”. But, in the course of the years, there might also be a change in the hormonal system and the circumstances of life which have an influence.

- 19 out of 20 women complained of discharging blood clots. This may indicate a disproportion between the amount of menstrual blood and the amount of coagulation factors therein. The amount of passing blood does not allow for maintaining liquidity. This in turn, may explain the fact that all the 20 patients suffer from moderate to heavy bleeding in most cycles.

This information was very interesting for me, as I had suspected to find more deviation regarding menarche, cycle length and bleeding intensity. Moreover, I had attributed more significance to the model role of the mother.

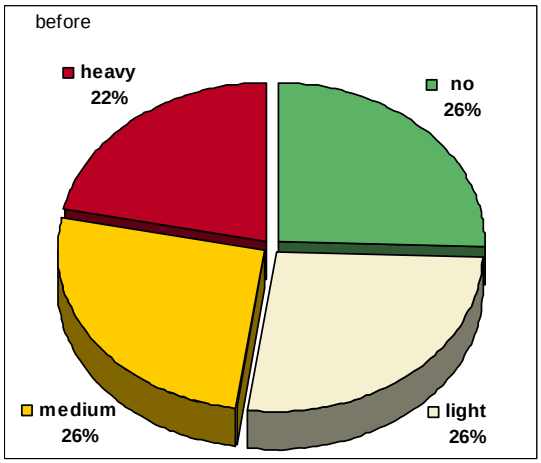
We have to bear in mind, though, that anamnesis of menstruation grants a rough overview only. Looking back with a critical eye, I think this part of the questionnaire should have included more questions in order to obtain more detailed and precise answers.

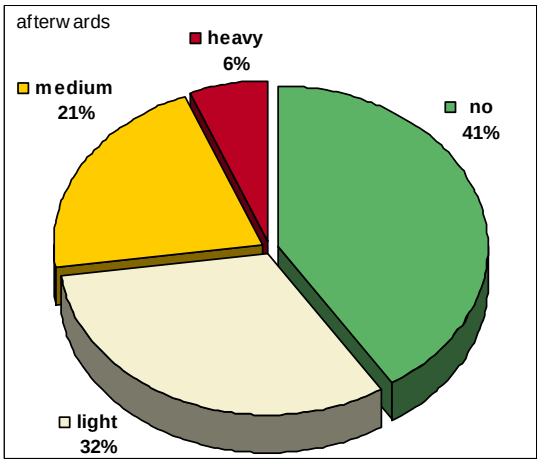
5.1.2. Part B of the questionnaire – Symptom groups

The following diagram clearly illustrates the changes.

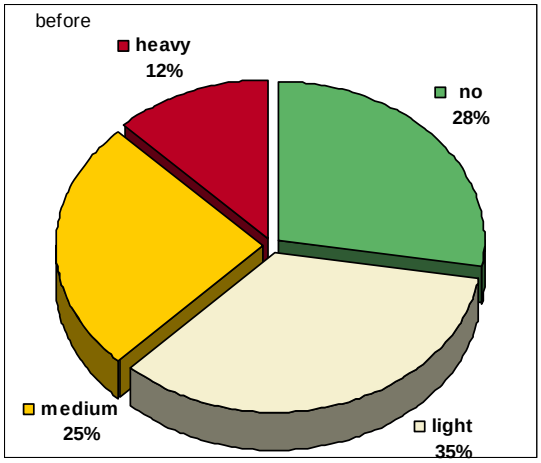
5.1.3. Graphic representation of the results in an overview

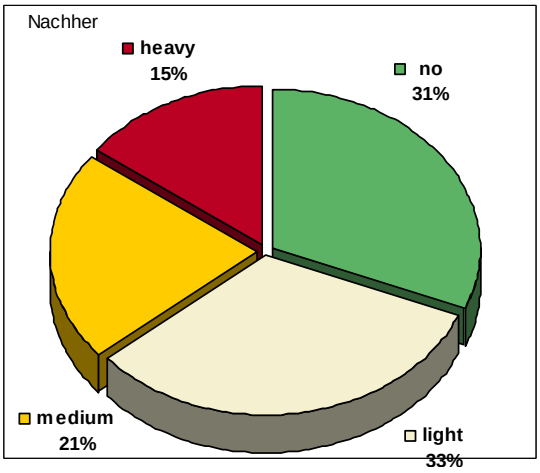
Test subjects





Control group





The graphic shows that there is a significant release in severe symptoms (-16%), at the same time the number of patients indicating no symptoms at all increased by 15% , whereas the control group remains nearly unchanged.

The overview of the statistics gives a first impression of the results which will be detailed in the following paragraph.

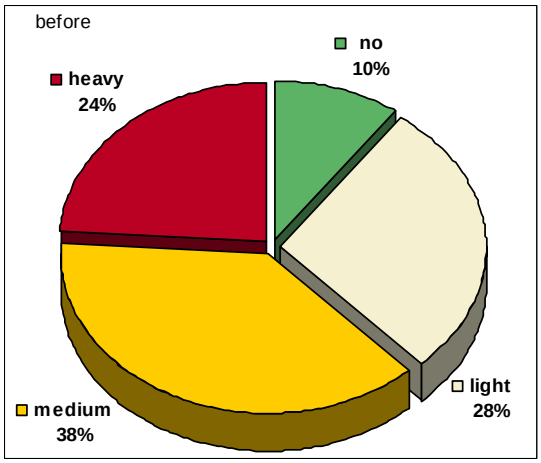
5.1.4. Graphic representation of the results of the individual groups of symptoms

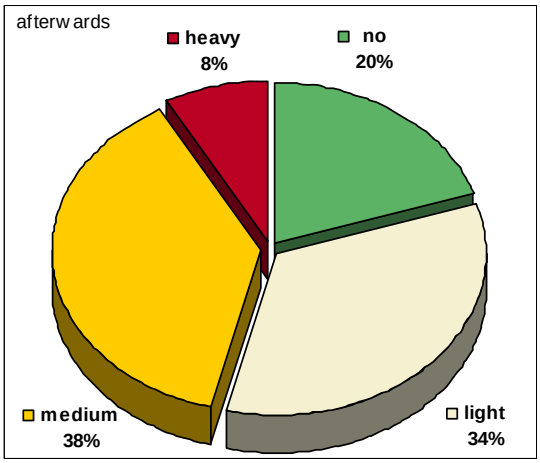
In order to keep the statistical evaluation clear and concise the questions of the individual symptom groups can be looked up in the appendix.

Graphic representation of the symptom groups – comparison of test group with control group:

In the symptom group PMS-A-anxiety, the main symptoms of which are nervous tension, irritability, aggressiveness, mood swings, fear and anxiety, there is a distinct improvement of the test group versus the control group.

Test group PMS-A-anxiety

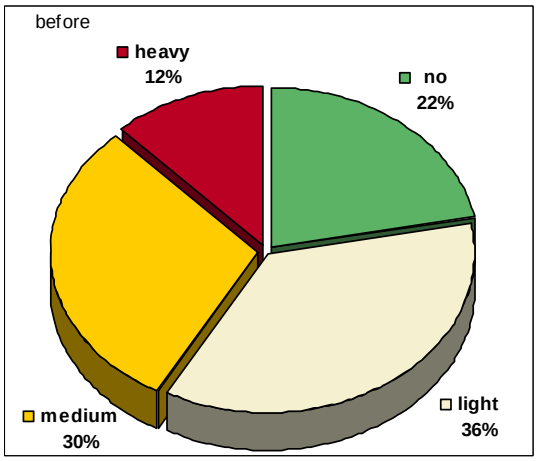


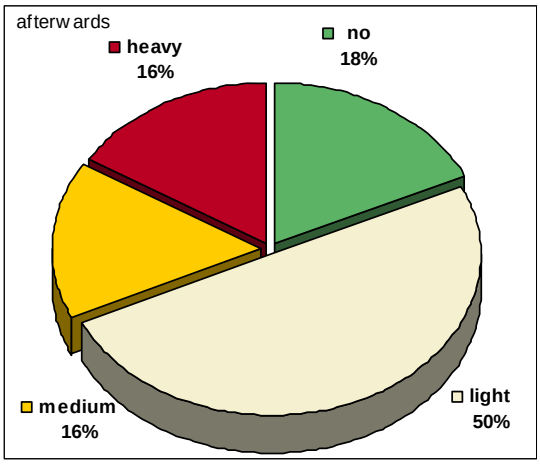


The following changes have been observed by comparing the test group with the control group.

The test group showed a significant change of severe symptoms decreasing from 24% to 8%, whereas the number of persons indicating no symptoms at all nearly doubled.

Control group PMS-A-anxiety

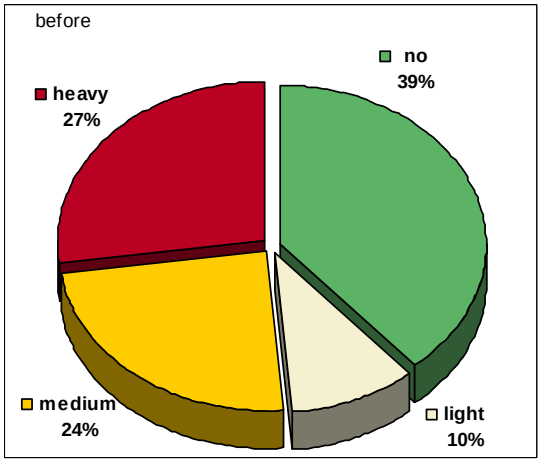


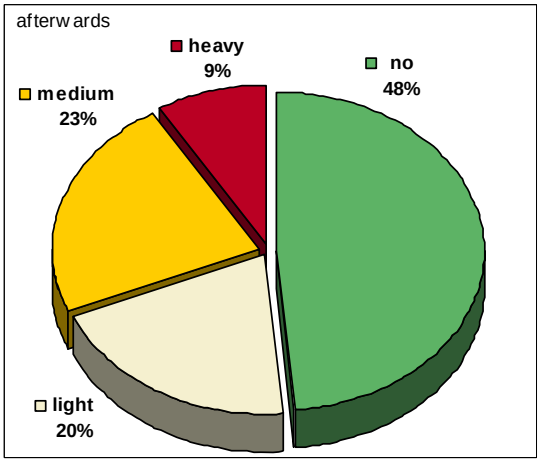


In the control group a change in intensity of the symptoms from middle to slight symptoms was described in 14% of the cases.

In the symptom group PMS-C-cravings, the main symptoms of which are headache, craving for sweets, increased appetite, heart pounding, tiredness, dizziness, and fainting, there is also a distinct improvement in the test group.

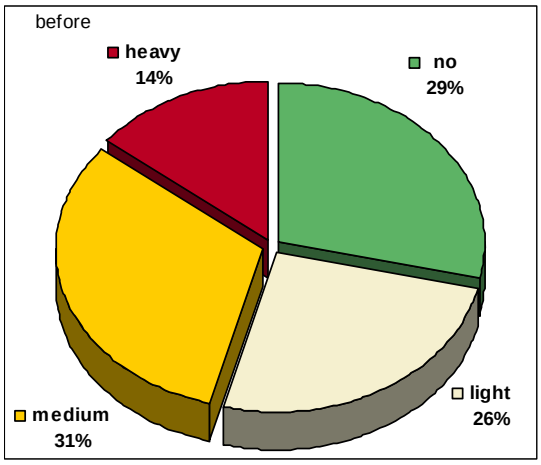
Test group PMS-C-cravings

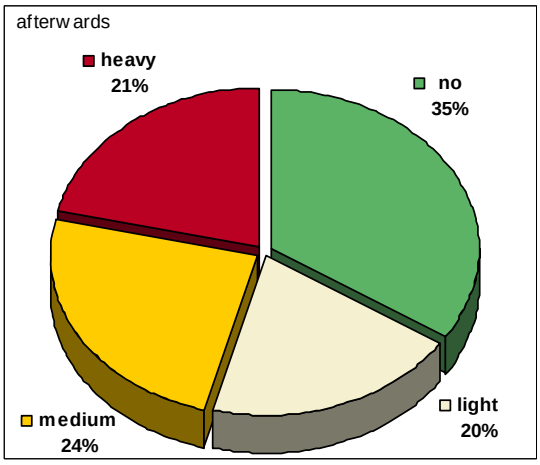




The test group showed a significant relief of the server symptoms from 27% to 9%, the number of persons indicating only light symptoms doubled.

Control group PMS-C-cravings

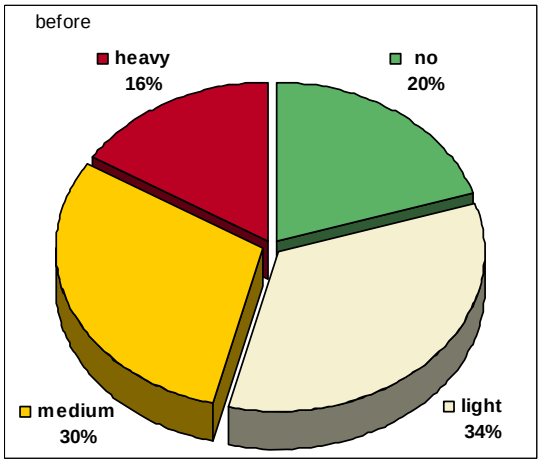


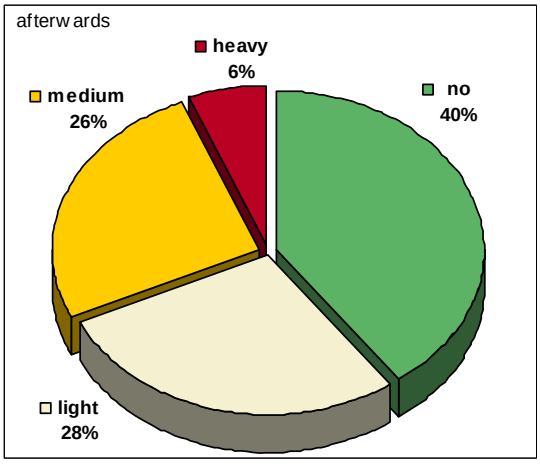


The control group does not show any significant changes.

In the symptom group PMS-H-hyperhydration, the main symptoms of which are breast tenderness, bloated abdomen, weight gain, swelling of the lower extremities and the eyelids, there too, was a distinct improvement in the test group.

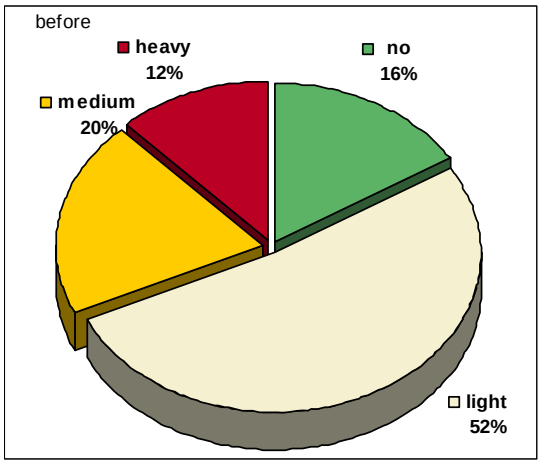
Test group PMS-H-hyperhydration

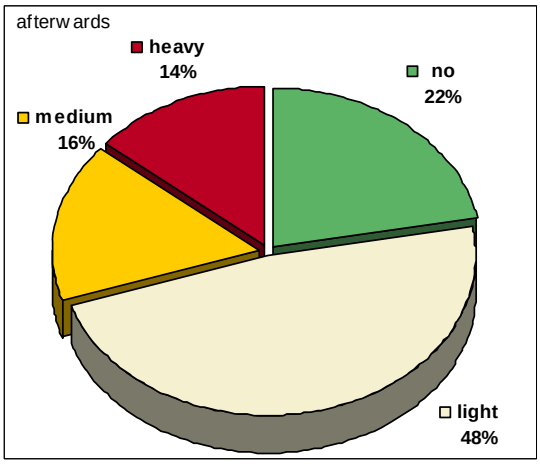




The test group indicated a significant relief of the severe symptoms from 16% to 6%, the number of persons indicating no symptoms at all doubled.

Control group PMS-H-hyperhydration

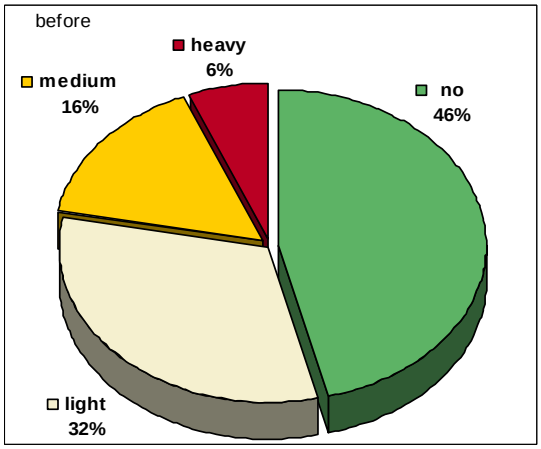


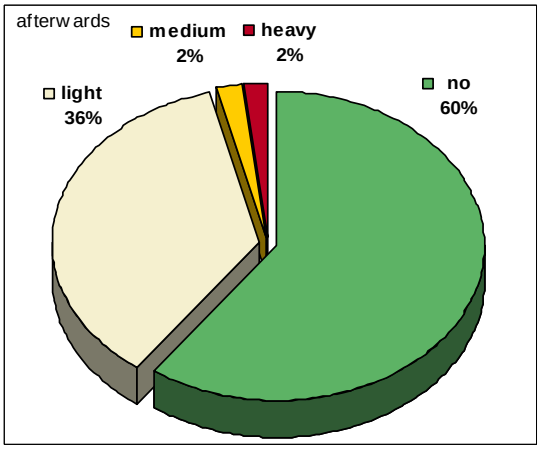


The control group does not show any significant changes.

Also the symptom group PMS-D-depression, the main symptoms of which are depression, forgetfulness, weeping, disorientation, and insomnia, showed positive changes in the test group.

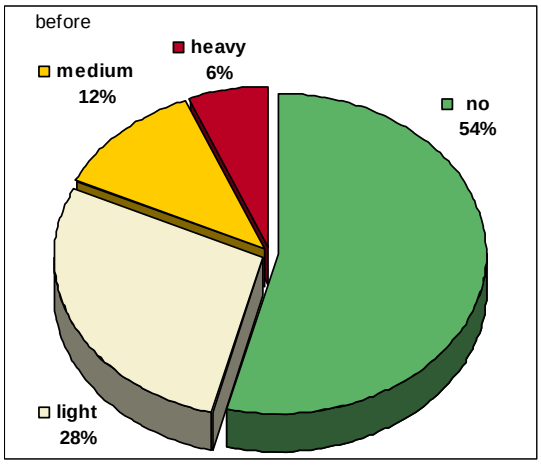
Test group PMS-D-depression

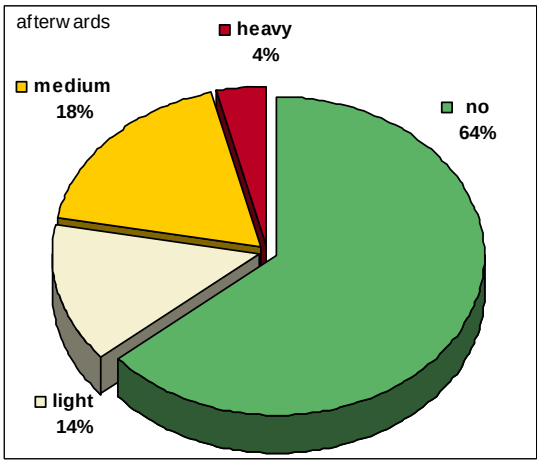




The test group indicates a significant relief of the middle range symptoms from 16% to 2%, the number of persons indicating severe symptoms increased by 14%.

Control group PMS-D-depression

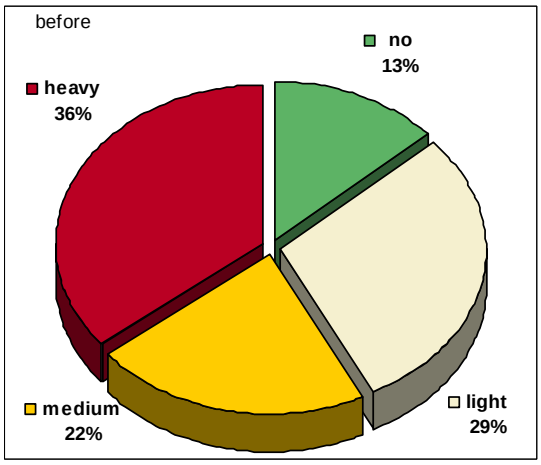


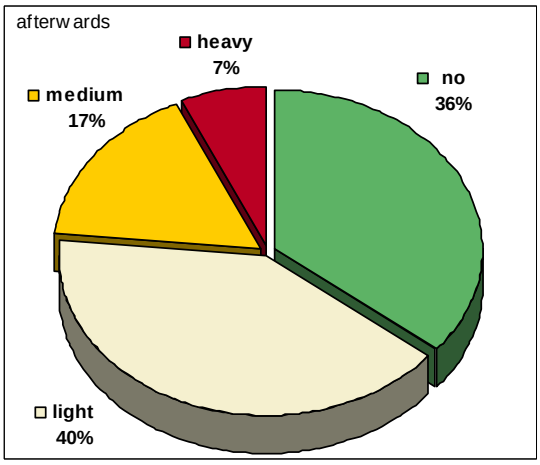


In the control group the number of persons indicating no symptoms at all increased by 10%.

The most pronounced changes took place in the test group PMS-P-pain, the main symptom of which is pain.

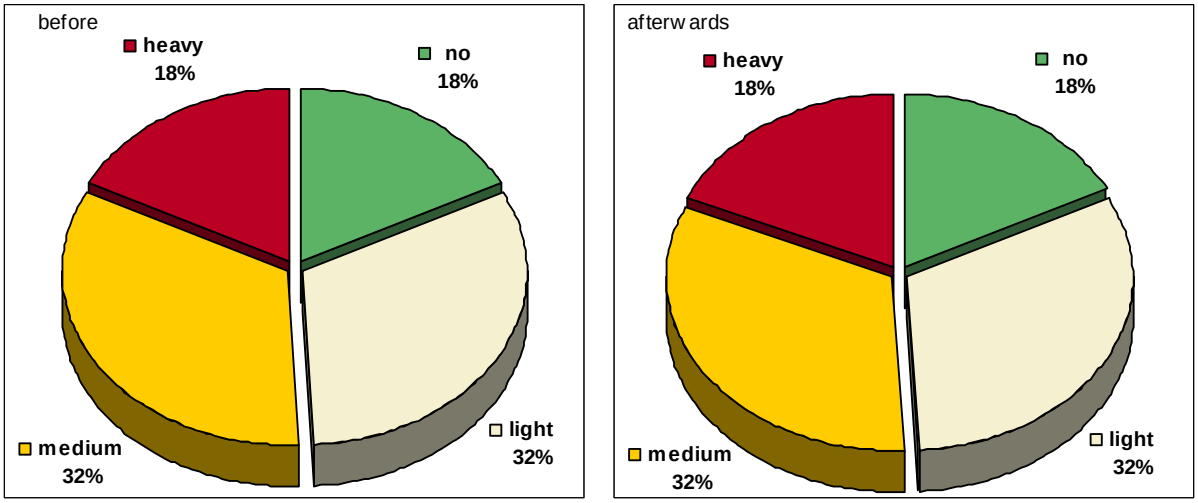
Test group PMS-P-pain





A significant change was observed in patients with severe symptoms, namely a decrease from 36% to 7%, the number of persons indicating no symptoms at all augmented from 13% to 36%.

Control group PMS-P- pain



The control group does not show any significant changes.

6. Discussion

The starting point for my study was the hypothesis that holistic, osteopathic treatment could bring about an improvement of congestive menstrual disorders and premenstrual syndrome.

It took very intensive and critical analysis of the problem to make me aware of its complexity. Ruth Trickey's book on Women, Hormones & Menstrual Cycle was of invaluable help to me.

Medical and scientific quarters put forth numerous theories which all boil down to one thing: hormones.

Hormones, of course, but why do these unmeasurable changes of hormones cause some women to suffer symptoms, yet others are not affected?

In my opinion, this is where osteopathy comes into play. Osteopathic treatment can help the body to deal more easily with the tricky hormonal situation.

Dysfunctions on all levels of the body can be reduced, allowing the body to better adapt itself to the hormonal situation

Within the framework of an osteopathic treatment we have to distinguish between two types of techniques. Those that influence directly the hormonal balance (e.g. work on SBS, third ventricle), and those that improve loss of mobility and motility in other body parts and so indirectly influence the hormonal situation by giving more flexibility.

Item 3 "Considerations and approach to osteopathic treatment" deals with these questions in detail.

Besides the hormonal factors a number of other aspects are worth considering, too.

- Psychological factors: in my work I am able to deal with psychological aspects inasmuch as I am convinced that by touching a persons body I also touch his/her soul.

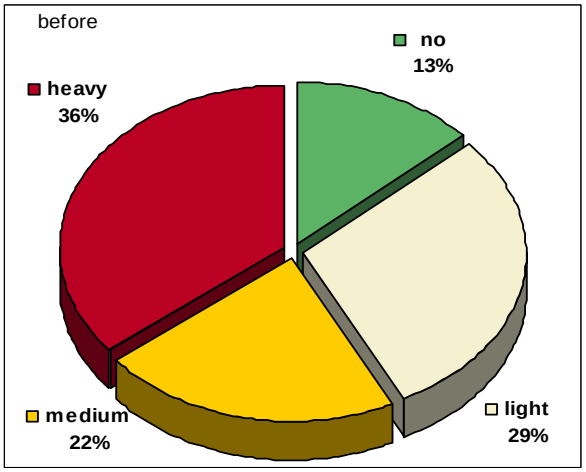
- Stress
- Nutrition-related factors

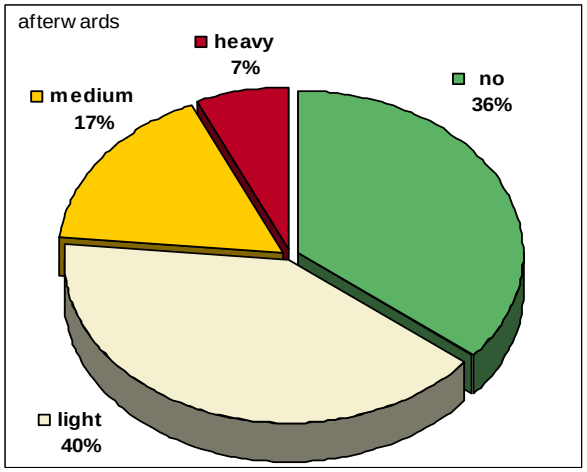
Liver function, in my opinion, is a vital aspect. With all my test subjects I could detect a more or less pronounced loss of mobility or motility of the liver. Given the fact that the liver plays a crucial role in processing estrogens on the one hand, and detoxifying the whole body on the other hand, I cannot support the administration of hormones, a common treatment for dysmenorrhea and PMS, because **hormone preparations** present an additional strain on the liver.

Now, I would like to go into more detail about the questions stated in the introduction.

1. Is it possible to improve pain symptomatology of PMS with osteopathic treatment or is there no difference in comparison with the control group? This question can definitely be answered with **yes** (see statistics).

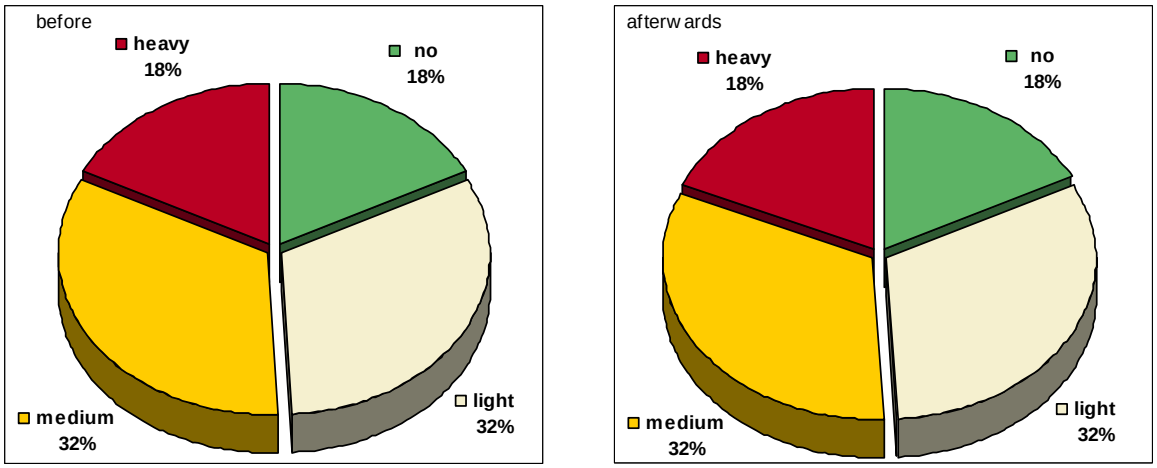
Test group





The test group indicated a significant betterment of the severe symptoms from 36% down to 7%, the number of persons indicating no symptoms increased from 13% to 36%.

Control group

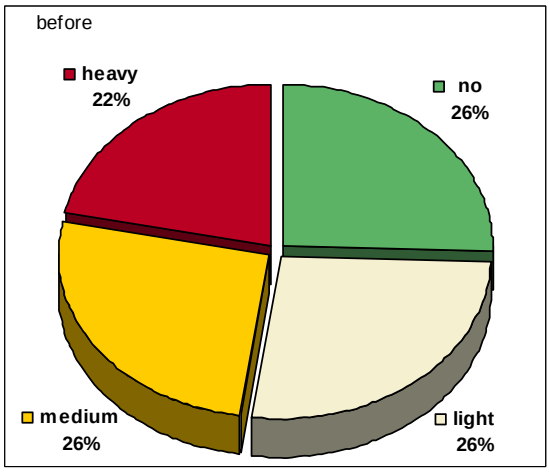


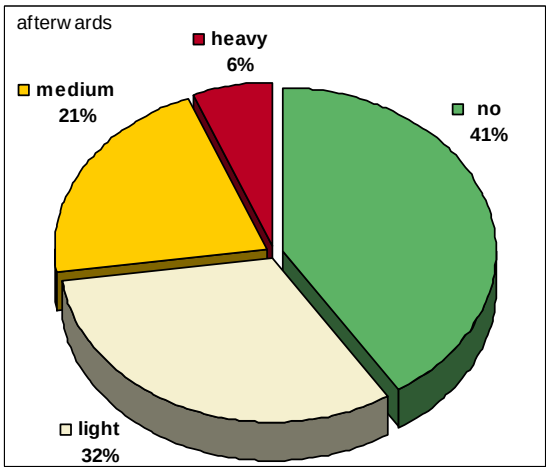
The control group does not show any change.

2. Do the positive changes affect all symptom groups? Again the answer is **yes**, but the most significant changes are seen in the area of pain.

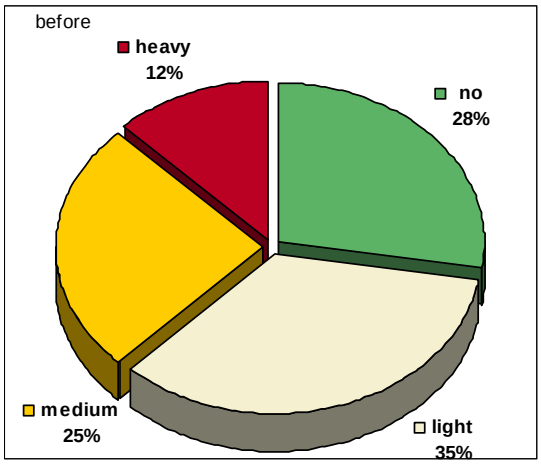
The graphic shows that there is a significant release in the severe symptoms (- 16%), at the same time the number of patients indicating no symptoms at all increased by 15%.

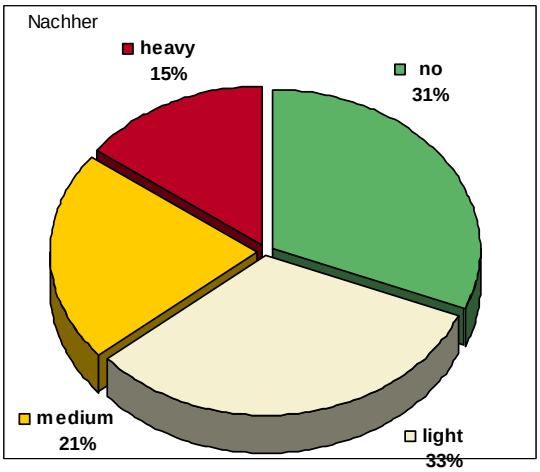
Test group





Control group





The control group does not show any change.

3. Is it possible to recognise preferential mechanisms of lesion? In general, I found it very difficult to put the lesion patterns of the individual patient in a system and so create a kind of recipe that would be useful to those who read my study. Unfortunately, there is no such recipe.

   The only thing I can do is give an account of my personal experience with my patients and comment on the resulting assumptions.

Due to the anamnestic details my patients disclosed to me, I neglected traumatic or operative influences, because very few patients mentioned them.
While doing the necessary tests for osteopathic data collection I came across numerous lesions.

The relevant lesions

- concerning posture were more often found in patients of the anterior type
- in the pelvic area were mainly restrictions of primary sacroiliac mobility in context with changes of tension and function in the lesser pelvis
- in the thoracolumbar junction became evident as difference of tension in the area of the diaphragm
- were found in T7/8/9, cervicothoracal junction, C0/C1/C2
- were changes of tension in the area of the fascia of the middle axis
- always included a restriction of the SBS which is a direct link to the hormonal system (pituitary gland, third ventricle)
- always included reduced fluctuation of CSF between cranium and sacrum.

On the basis of the data I collected and the findings which clearly revealed these central points of problems I set up the following hypothesis:

From the osteopath's view, patients suffering from congestive dysmenorrhea and PMS need to be given a lot of attention to their craniosacral system and the structures which are causal for the proper functioning of the hormonal system (liver, small intestine, lesser pelvis …). Besides the hormonal factors, however, the mobility of the pelvic area plays a major role, from a structural as well as a visceral point of view, as it ensures the best possible conditions for the proper functioning of the organs.

While working with the patients I became aware of how significant proper functioning of the entire fascia of the middle axis is and how stabilising the effect on the whole body is, if this vital area functions properly.
The anatomic background confirms the importance of these connections, particularly considering the close links to the cranial basis.
Seen on a wider scale other factors have to be included as well.

- Stress reducing factors (80% of the subjects claimed to be exposed to heavy stress in their job or their private life)
- Nutritional factors, often induced by stress factors. A confirmation of this assumption is the fact that at week-ends, with a usually lower stress level, patients use fewer pain killers
- Psychogenic factors

The success I achieved with osteopathic treatment leads me to the conclusion that with my point of view I have good possibility of giving therapeutic support to at least some of the women suffering from dysmenorrhea and PMS. Being a woman myself, it is particularly rewarding to be able to contribute to such a considerable extent to **the improvement of life quality**.

The small number of test persons included in this study does not allow a statistically relevant result; nevertheless, the results obtained are a clear indication of the validity of osteopathic treatment.

The design of the questionnaire was a difficult task. In hind view, I would include more details for the different groups of symptoms, especially the PMS Pain Group. The case history sheet concerning menstruation would also be more relevant if more details were contained.

As a closing remark I would like to direct attention to the study of Heike Karl-Schindler, who concluded her study during the time it took me to conclude mine.

Very encouragingly, her study Osteopathic treatment of primary dysmenorrhea – possible effects revealed similar results, thus supporting the findings of my study.

In the course of my work I became more and more interested in the specific function of the liver as regards the processing of hormones. It would be very motivating for me, if somebody decided to write his/her thesis on that topic.

7. Summary

At the beginning of my study I put forward the hypothesis that it should be possible to improve complaints of congestive dysmenorrhea and the symptoms of PMS by giving the patients osteopathic treatment. The hypothesis was confirmed by the findings of the questionnaire.
It has been proven beyond any doubt that hormones, or rather the limited flexibility of the body to deal properly with the current hormonal situation, play a significant role. Therefore, any osteopathic treatment has to aim at supporting the body individually so that this situation can be dealt with in a more favourable way.
In my experience, it is important to combine structural, cranial and visceral treatment in order to do justice to the individual clinical picture.

In order to support osteopathic work and increase concerned patients' responsibility for themselves it is necessary to dig a little deeper and find out about the circumstances of these women's lives. In this way, the significance of stress and other psychological or nutritional causes becomes apparent.

As a concluding word I would like to emphasise that in the course of this study I attached more and more importance to the knowledge that with my osteopathic abilities I literally hold in my hands the possibility to contribute to the quality of life of so many women.

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9. List of illustrations

Abb.15 Feedback regulation between ovaries, hypophysis and hypothalamus
Drenkhahn, D. and Zenker, W. (1994): Benninghoff Anatomie II. München: Urban & Schwarzenberg. Page 147.

Abb.16 Enterohepatic circulation of estrogens
Trickey, R. (1998): Women, Hormones & The The Menstrual Cycle: St. Leonhards/Australia: Allen & Unwin. Page 61.

Abb.17 Portal system between neurohypophysis and adenohypophysis
Drenkhahn, D. and Zenker, W. (1994): Benninghoff Anatomie II. München: Urban & Schwarzenberg. Page 187.

Abb.18 Ventricle of the brain
Putz, R. and Pabst, R. (1993): Sobotta I. München: Urban & Schwarzenberg. Page 296.

Abb.19 Postural profiles
Richard, J.P. (1994): Die Wirbelsäule aus der Sicht der Osteopathie. Kötzing: Verlag für Osteopathie. Page 67.

10. Appendix

10.1. Questionnaire

Questionnaire: ***to be filled in before the first and after the last treatment***

Patient Nr. Age: Date:
Height: Weight.

A. History of menstruation				
1. First menstruation	10-12 □	12-14 □	earlier □	later □
2. Cycle length	28-30 □	35-40 □	less □	more □
3. Intensity of bleeding	heavy □	moderate □	light □	scant □
4. Did your mother suffer from similar problems?		no □		
5. Do you take any drugs / pain killers?	yes □	no □		
6. Do you get the pain / problems in every cycle?	yes □	no □		
7. Has your cycle changed since the first menstruation?	yes □	no □		
8. What does this change include?				
	• change in pain	yes □	no □	
	• change in bleeding	yes □	no □	
	• change in cycle length	yes □	no □	
9. Do you discharge blood clots during your period?	yes □	no □		

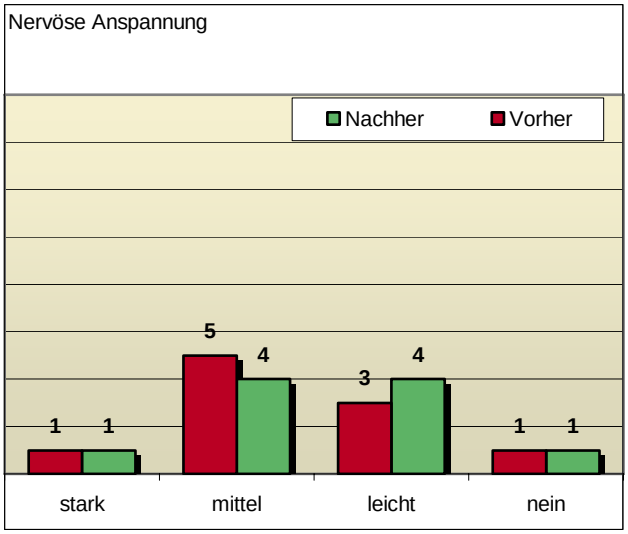
B. Symptoms				
• **PMS-A-anxiety**				
1. Nervous tension	no □	light □	medium □	heavy □
2. Irritability	no □	light □	medium □	heavy □
3. Aggressiveness	no □	light □	medium □	heavy □
4. Mood swings	no □	light □	medium □	heavy □
5. Fear, anxiety	no □	light □	medium □	heavy □
• **PMS-C-cravings**				
1. Headache	no □	light □	medium □	heavy □
2. Craving for sweets	no □	light □	medium □	heavy □
3. Increased appetite	no □	light □	medium □	heavy □

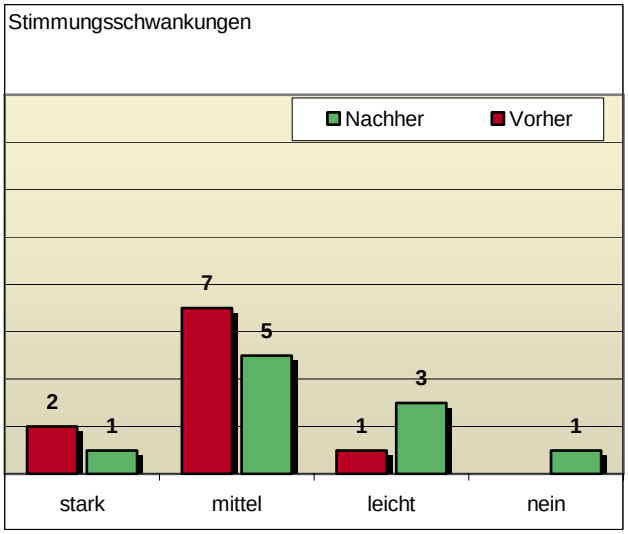
4. Heart pounding	no □	light □	medium □	heavy □
5. Fatigue	no □	light □	medium □	heavy □
6. Dizziness	no □	light □	medium □	heavy □
7. Fainting	no □	light □	medium □	heavy □
● **PMS-H-hyperhydration**				
1. Breast tenderness	no □	light □	medium □	heavy □
2. Bloated abdomen	no □	light □	medium □	heavy □
3. Weight gain	no □	light □	medium □	heavy □
4. Swelling of lower extremities	no □	light □	medium □	heavy □
5. Swelling of eyelids	no □	light □	medium □	heavy □
• **PMS-D-depression**				
1. Depression	no □	light □	medium □	heavy □
2. Forgetfulness	no □	light □	medium □	heavy □
3. Weeping	no □	light □	medium □	heavy □
4. Disorientation	no □	light □	medium □	heavy □
5. Insomnia	no □	light □	medium □	heavy □
• **PMS-P-pain**				
1. Pain before and at onset of bleeding	no □	yes □	slightly reduced □	distinctly reduced □
2. Pain considerably reduced by increased bleeding or discharge of blood clots	no □	yes □		
3. Continuous, dull pain in the background	no □	light □	medium □	heavy □
4. Masked by spastic, episodic pain	no □	light □	medium □	heavy □
5. Pain feels like hard work for the body	no □	light □	medium □	heavy □
6. Pain centred in lower abdomen	no □	light □	medium □	heavy □
7. Radiating pain to				
● inguinal region	no □	light □	medium □	heavy □
• back	no □	light □	medium □	heavy □
• thigh	no □	light □	medium □	heavy □
8. Sensation of fullness and heaviness in upper and lower abdomen	no □	light □	medium □	heavy □
9. Digestive complaints	no □	light □	medium □	heavy □
10. Reduced pain tolerance	no □	light □	medium □	heavy □

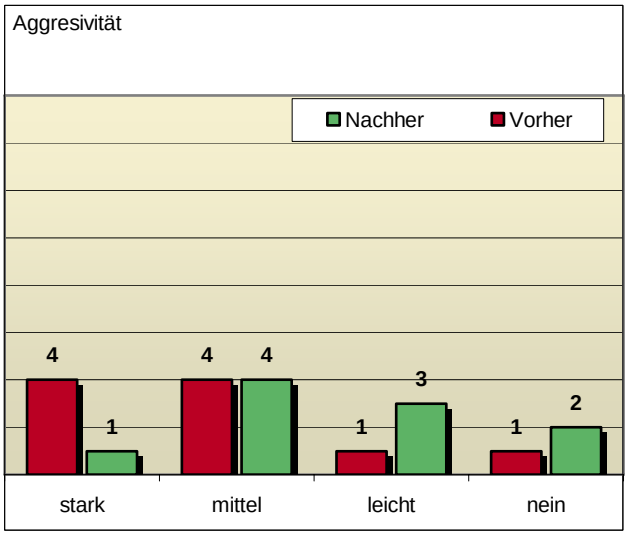
10.2. Statstical evaluation of the individual questions

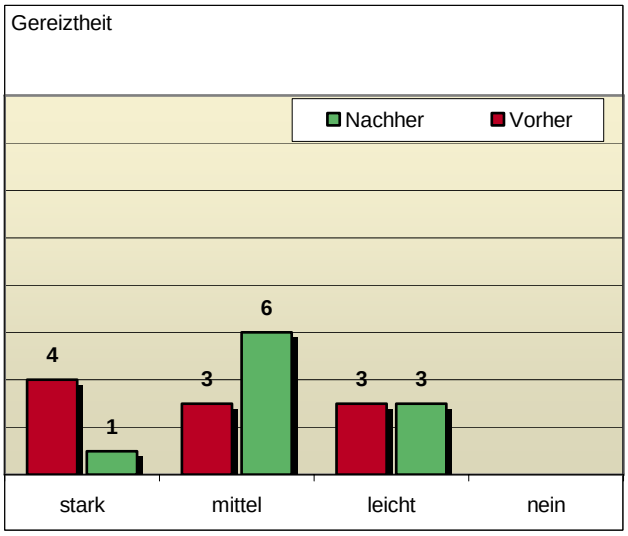
10.2.1. Test group

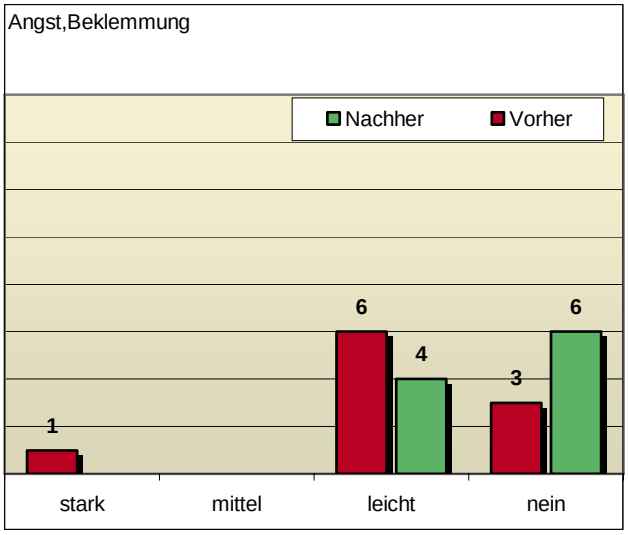
PMS-A- Anxiety



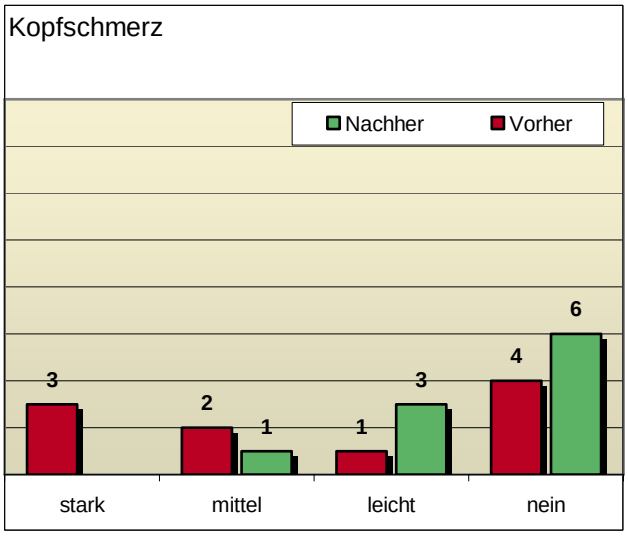


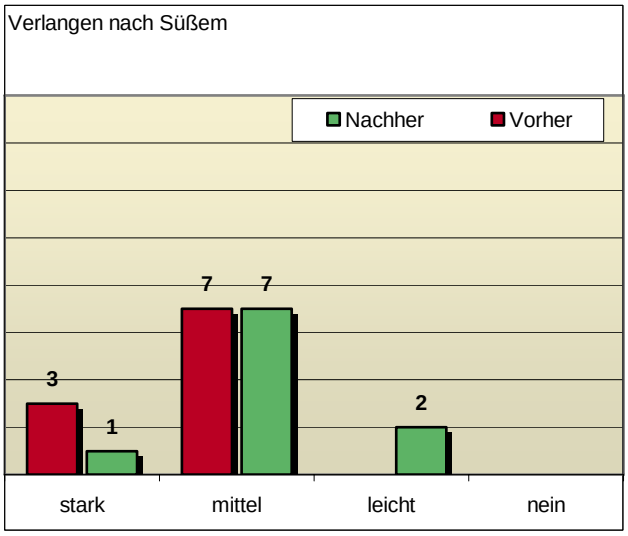


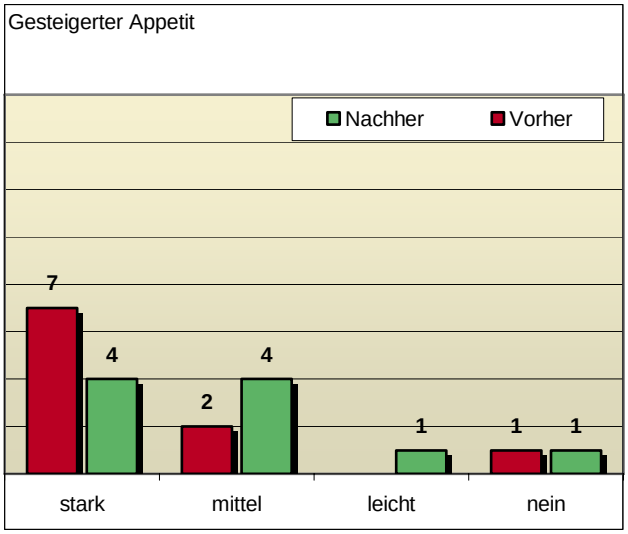


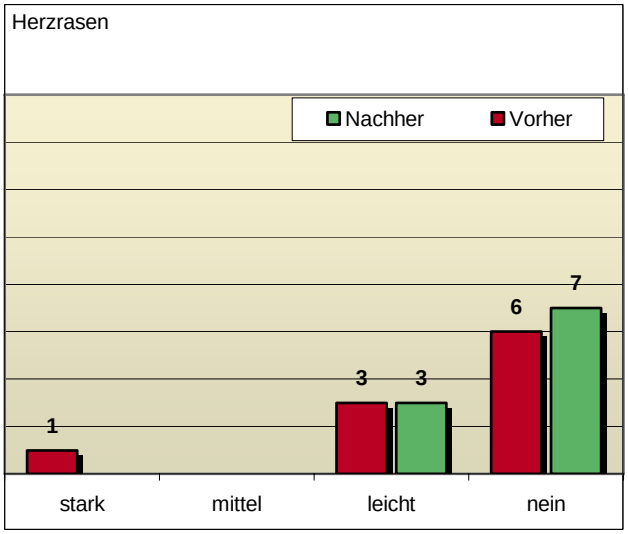


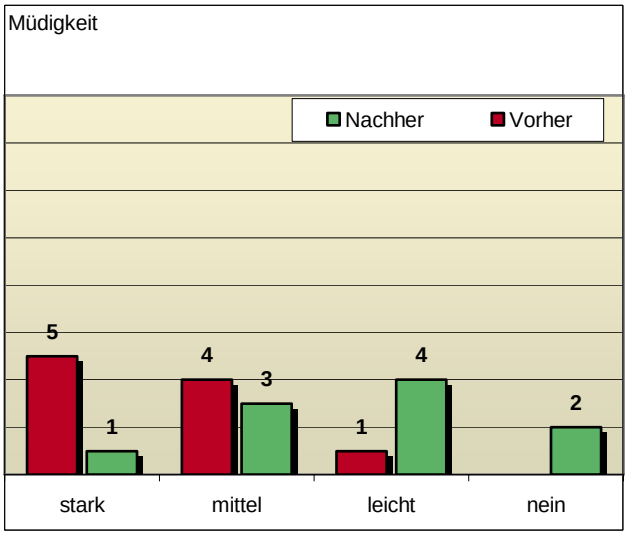
PMS-C- Cravings

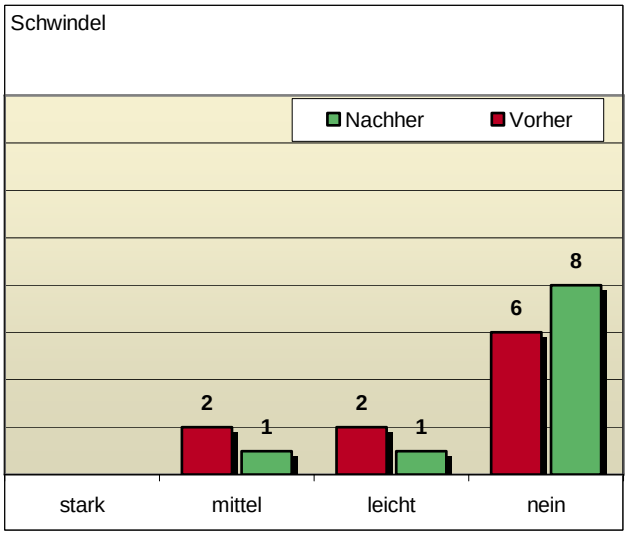


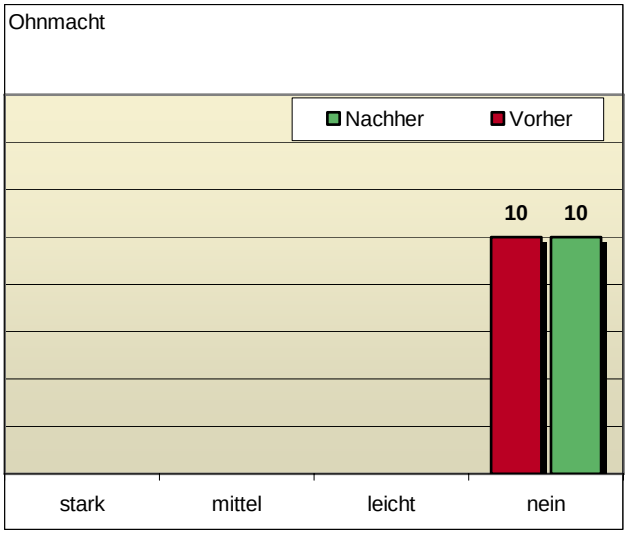



Gesteigerter Appetit
Nachher
Vorher
7
4
2
4
1
1
1
stark
mittel
leicht
nein

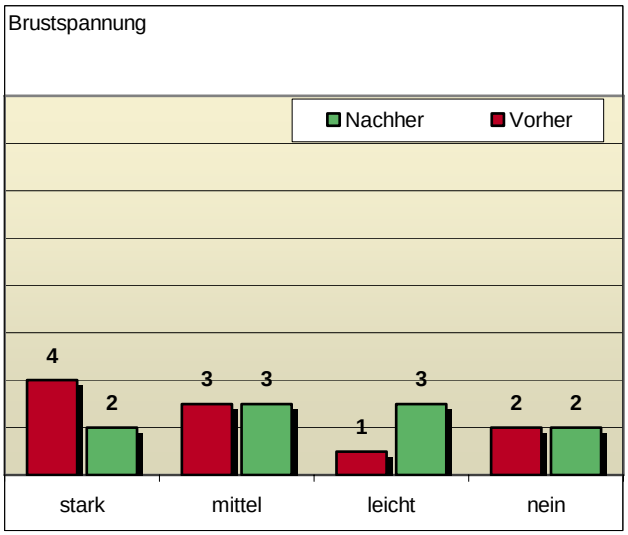

Herzrasen
Nachher
Vorher
1
3
3
6
7
stark
mittel
leicht
nein

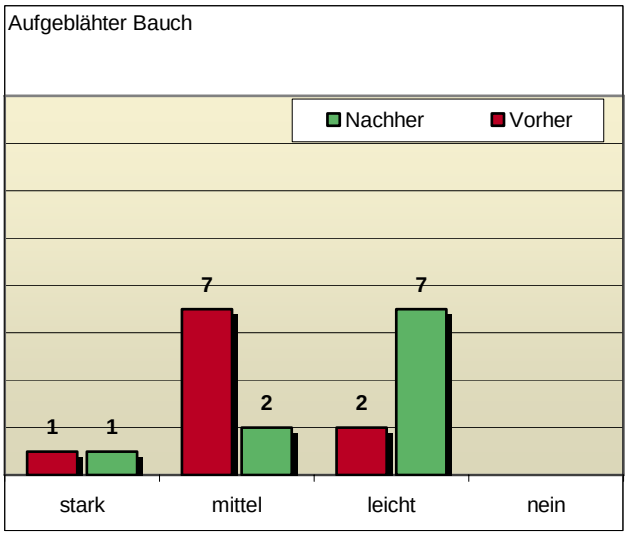

Müdigkeit
Nachher
Vorher
5
1
4
3
1
4
2
stark
mittel
leicht
nein

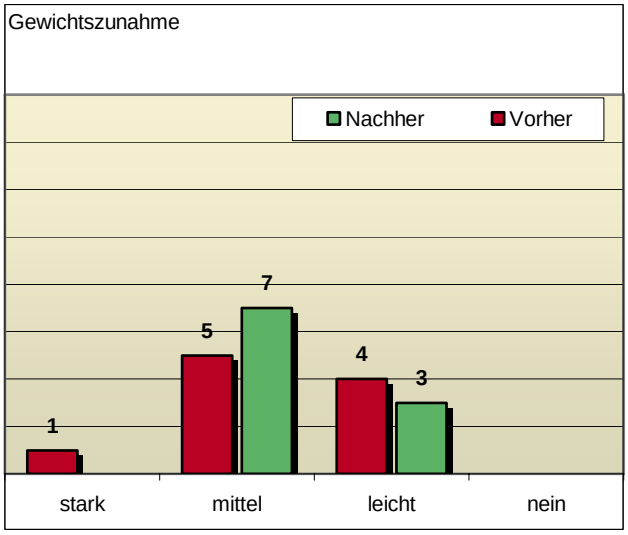

Schwindel
Nachher
Vorher
2
1
2
1
6
8
stark
mittel
leicht
nein

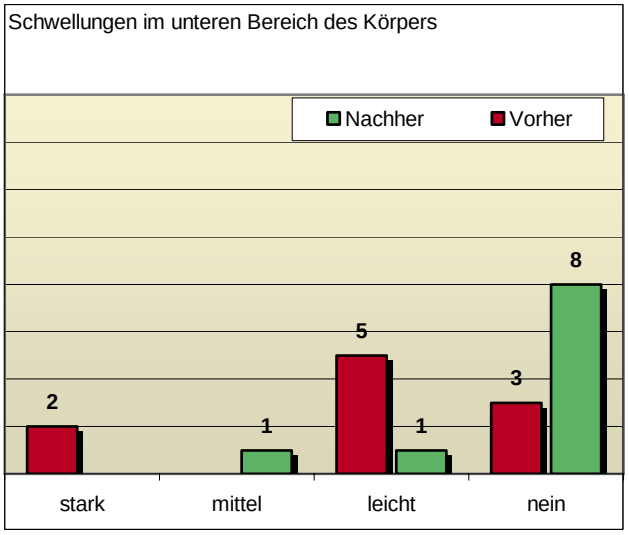


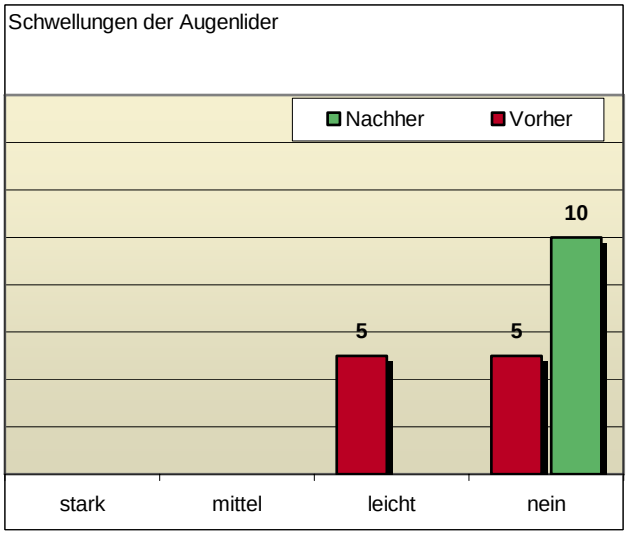
PMS-H-Hyperhydration



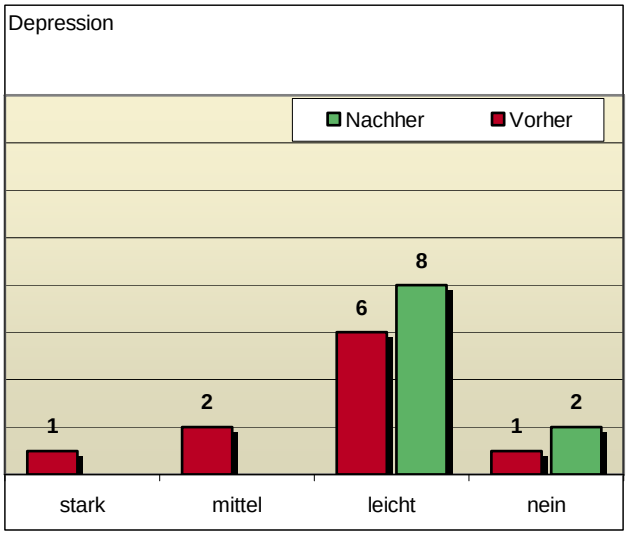


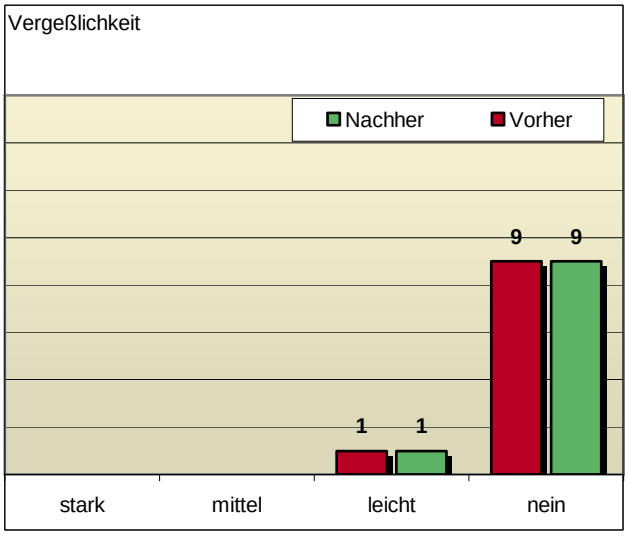

Gewichtszunahme
Nachher
Vorher
1
5
7
4
3
stark
mittel
leicht
nein

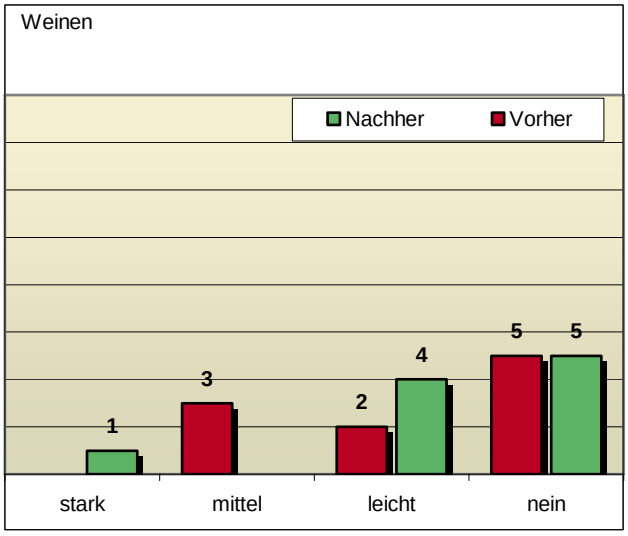

Schwellungen im unteren Bereich des Körpers
Nachher
Vorher
2
1
5
1
3
8
stark
mittel
leicht
nein

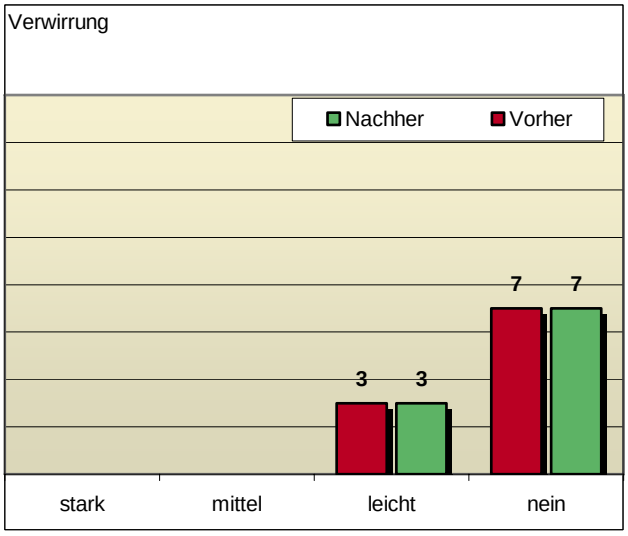

Schwellungen der Augenlider
Nachher
Vorher
5
5
10
stark
mittel
leicht
nein

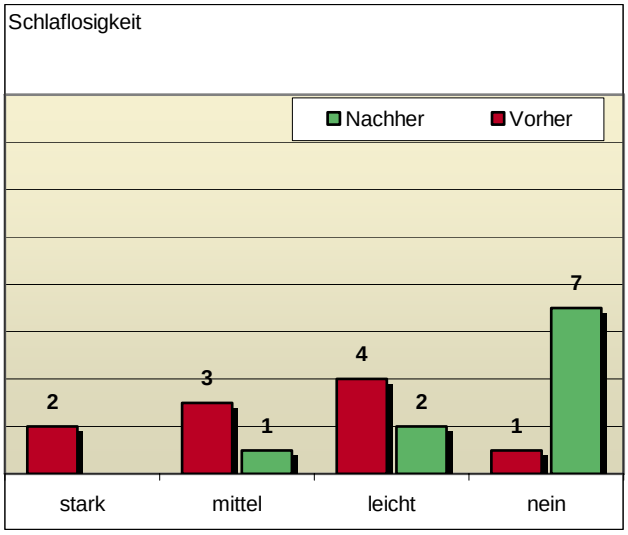
PMS-D-Depression



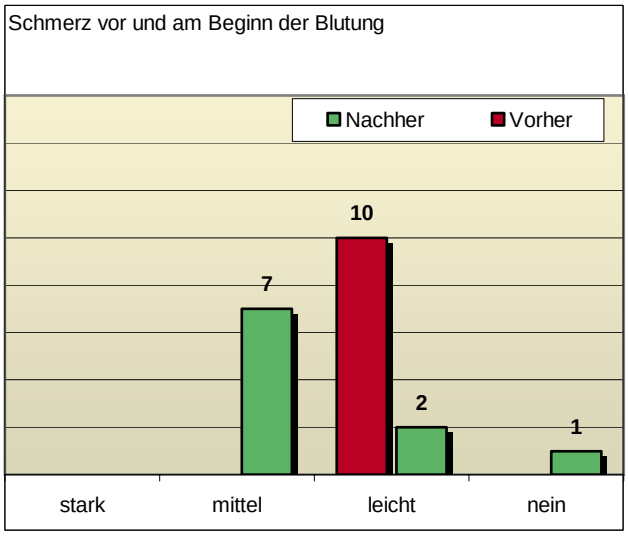


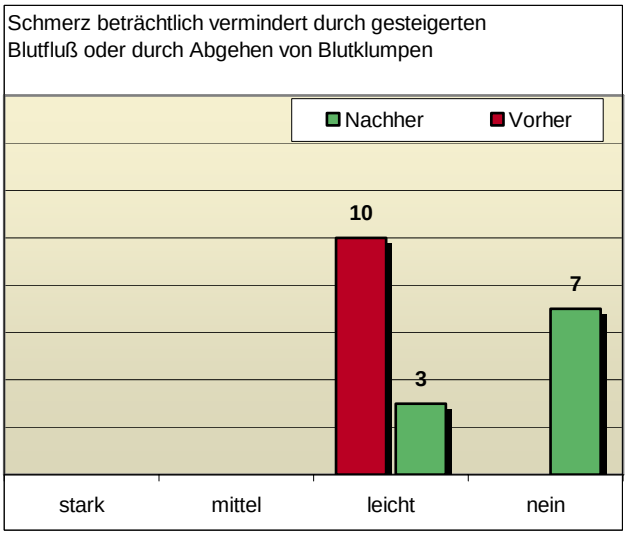


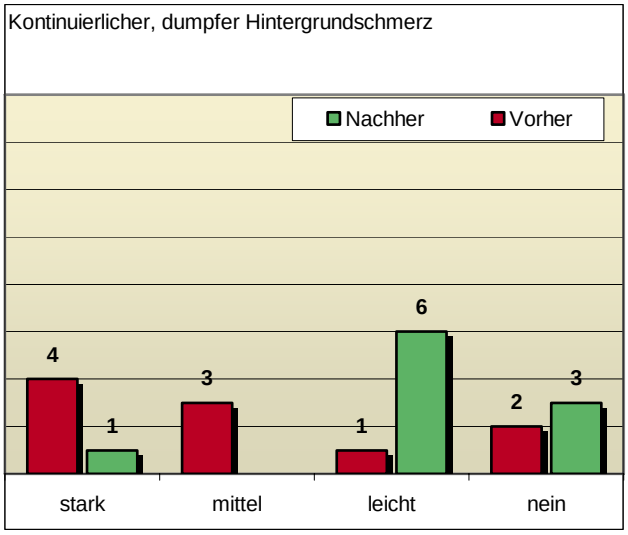


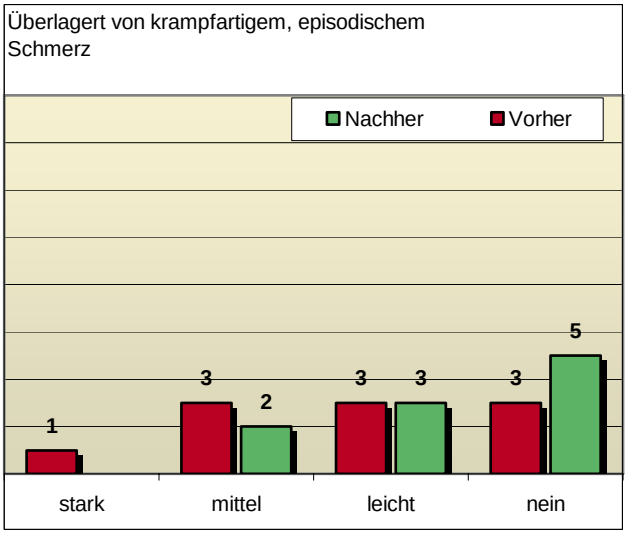

Schlaflosigkeit
Nachher
Vorher
2
3
1
4
2
1
7
stark
mittel
leicht
nein

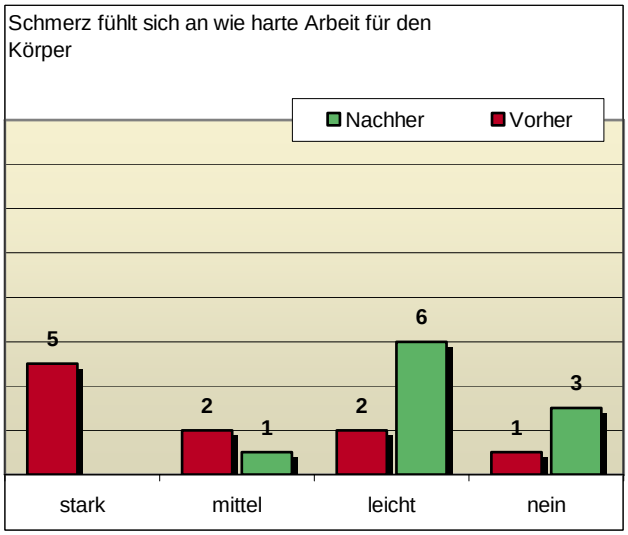
PMS-P-Pain

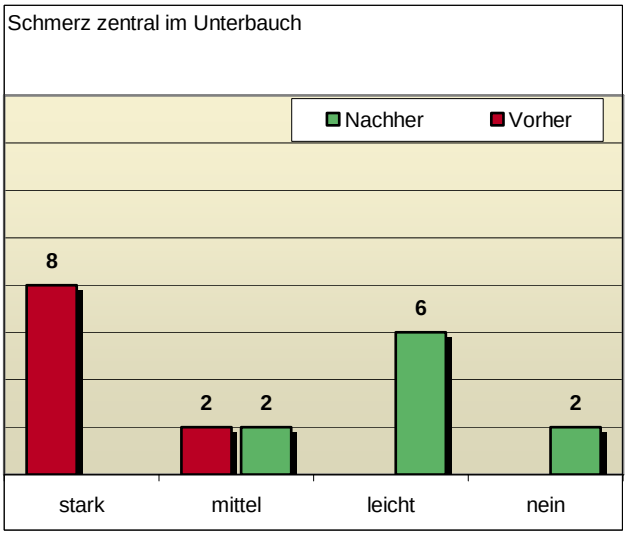

Schmerz vor und am Beginn der Blutung
Nachher
Vorher
7
10
2
1
stark
mittel
leicht
nein

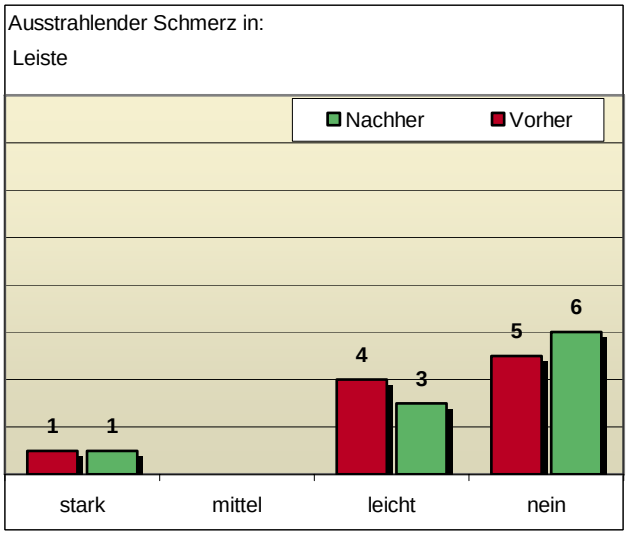

Schmerz beträchtlich vermindert durch gesteigerten Blutfluß oder durch Abgehen von Blutklumpen
Nachher
Vorher
10
3
7
stark
mittel
leicht
nein

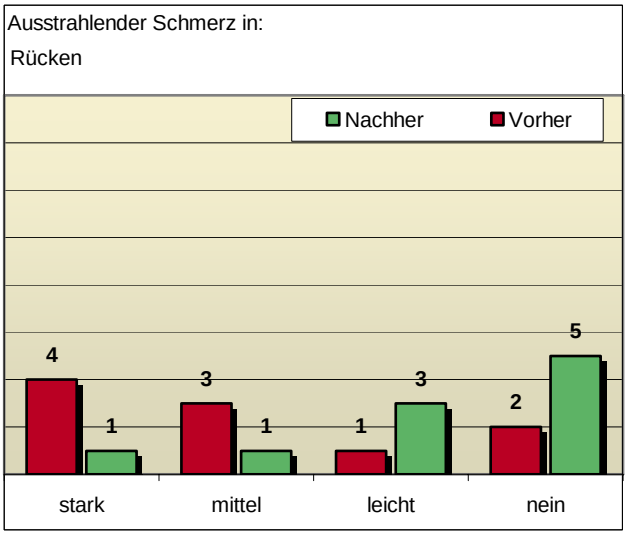

Kontinuierlicher, dumpfer Hintergrundschmerz
Nachher
Vorher
4
1
3
1
6
2
3
stark
mittel
leicht
nein

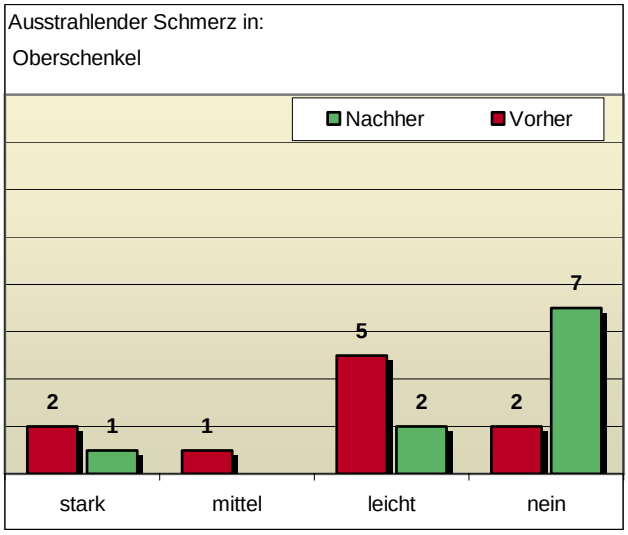

Überlagert von krampfartigem, episodischem Schmerz
Nachher
Vorher
1
3
2
3
3
3
5
stark
mittel
leicht
nein

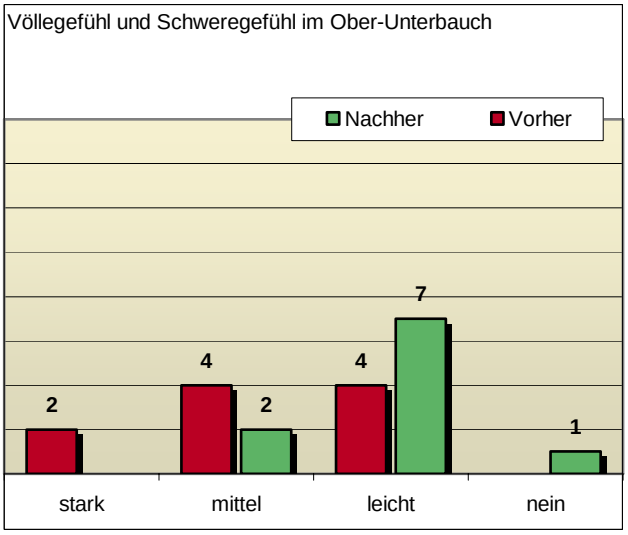

Schmerz fühlt sich an wie harte Arbeit für den Körper
Nachher
Vorher
5
2
1
2
6
1
3
stark
mittel
leicht
nein

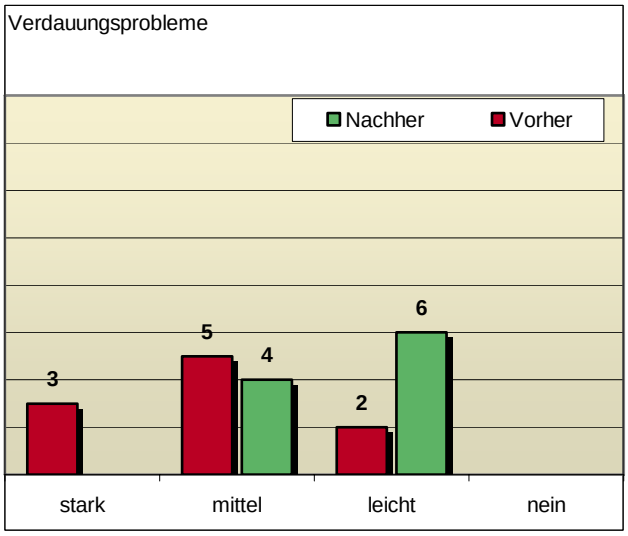

Schmerz zentral im Unterbauch
Nachher
Vorher
8
2
2
6
2
stark
mittel
leicht
nein

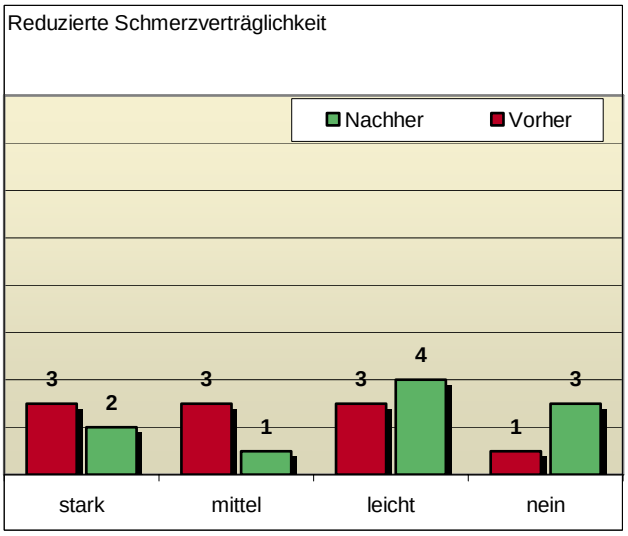

Ausstrahlender Schmerz in:
Leiste
Nachher
Vorher
1
1
4
3
5
6
stark
mittel
leicht
nein


Ausstrahlender Schmerz in:
Rücken
Nachher
Vorher
4
1
3
1
1
3
2
5
stark
mittel
leicht
nein


Ausstrahlender Schmerz in:
Oberschenkel
Nachher
Vorher
2
1
1
5
2
2
7
stark
mittel
leicht
nein

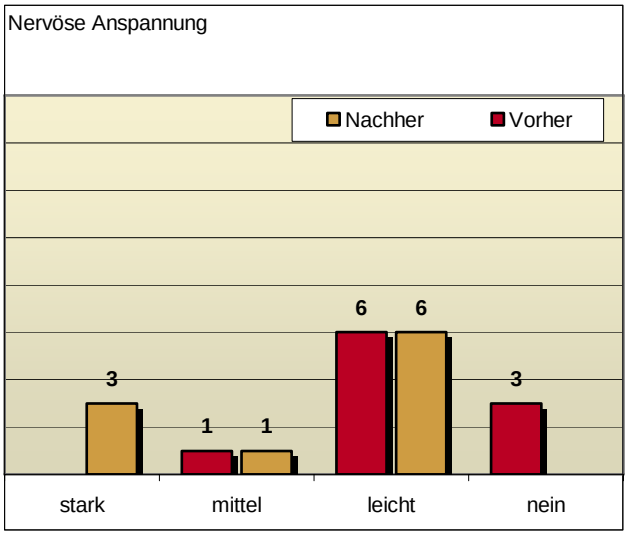

Völlegefühl und Schweregefühl im Ober-Unterbauch
Nachher
Vorher
2
4
2
4
7
1
stark
mittel
leicht
nein

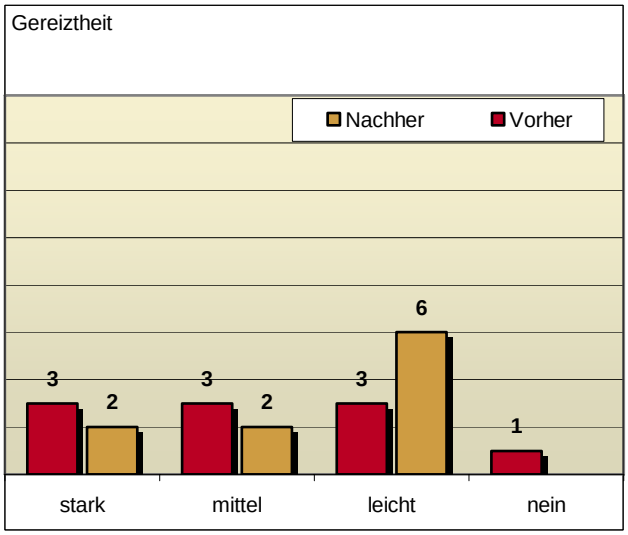

Verdauungsprobleme
Nachher
Vorher
3
5
4
2
6
stark
mittel
leicht
nein

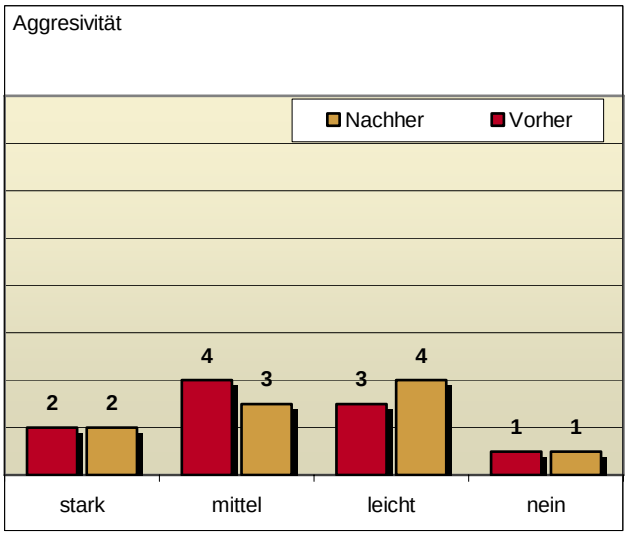

Reduzierte Schmerzverträglichkeit
Nachher
Vorher
3
2
3
1
3
4
1
3
stark
mittel
leicht
nein

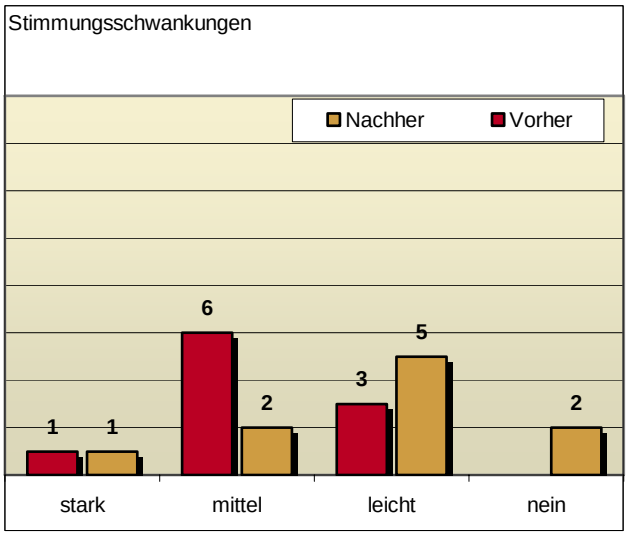
10.2.2. Control group

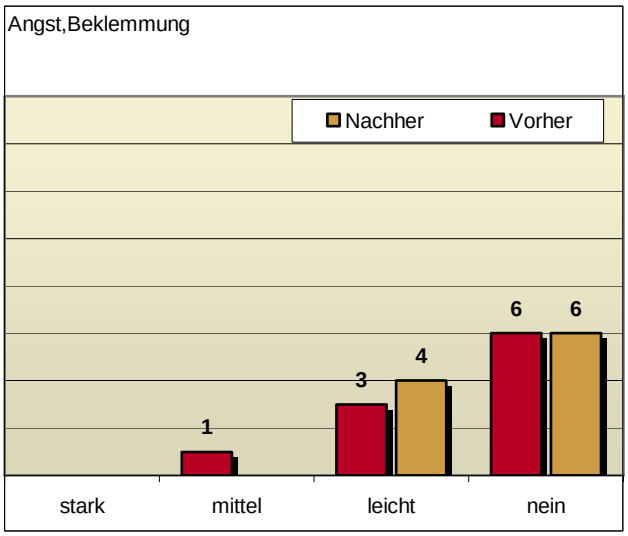
PMS-A-Anxiety



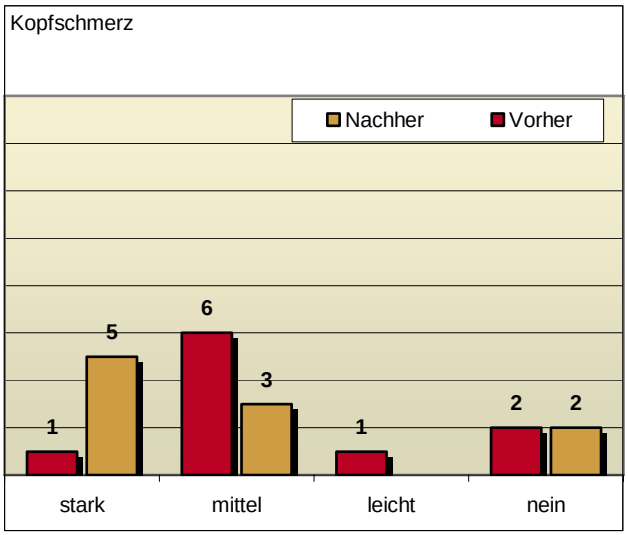


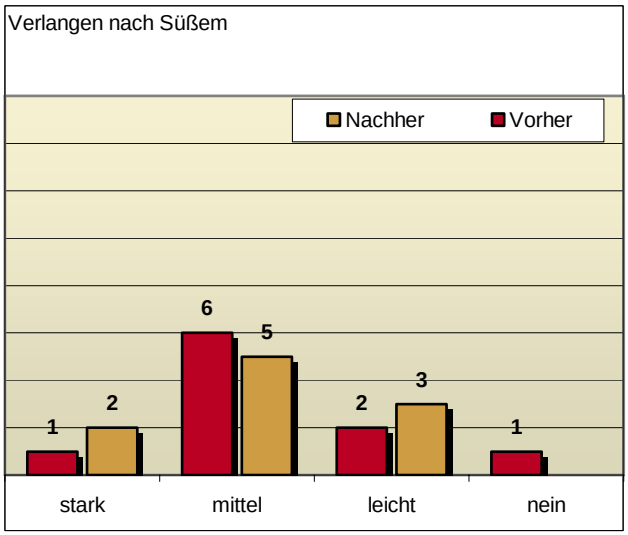


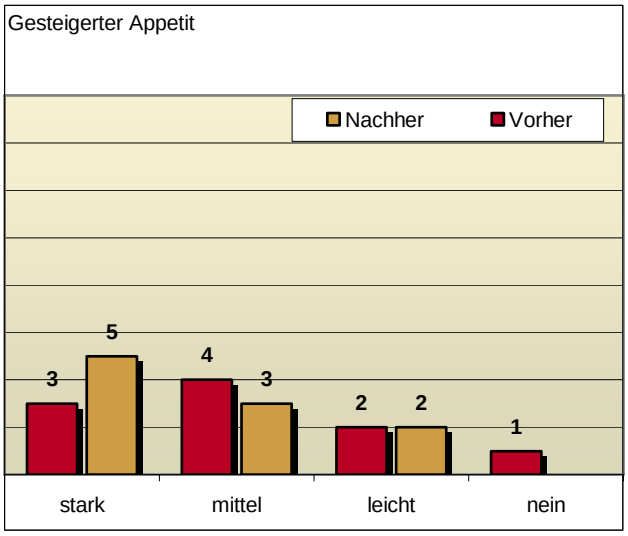


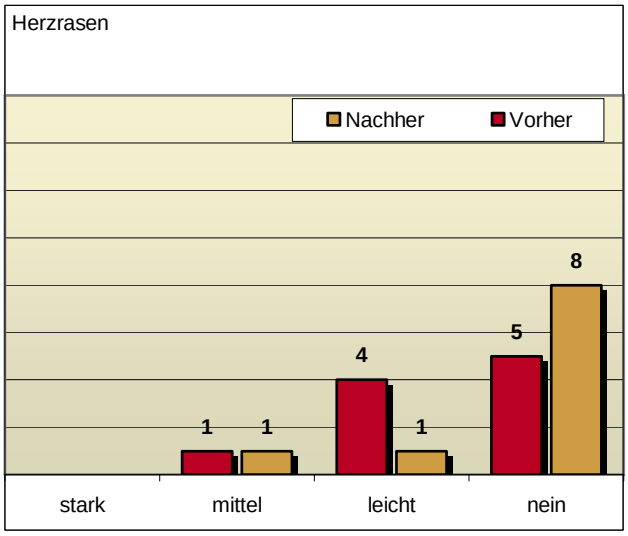


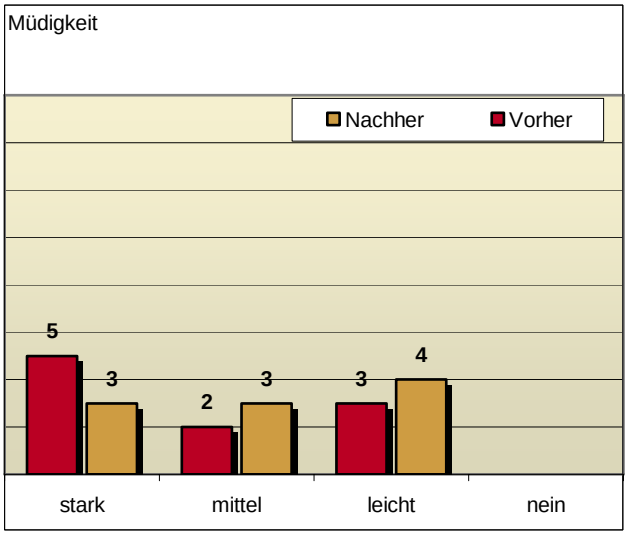
PMS-C-Cravings

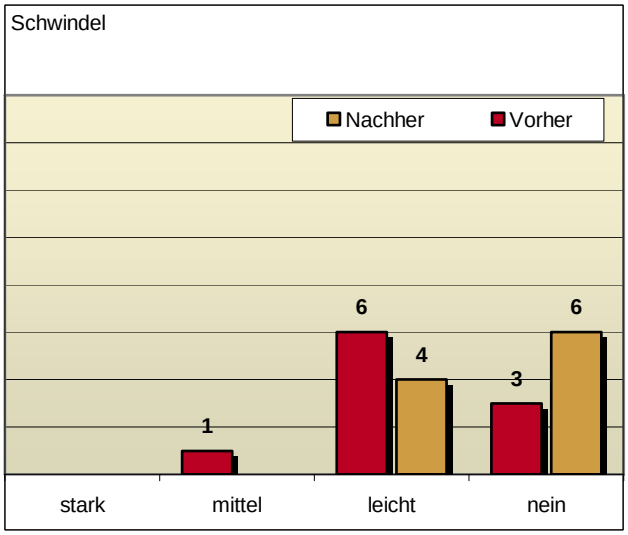


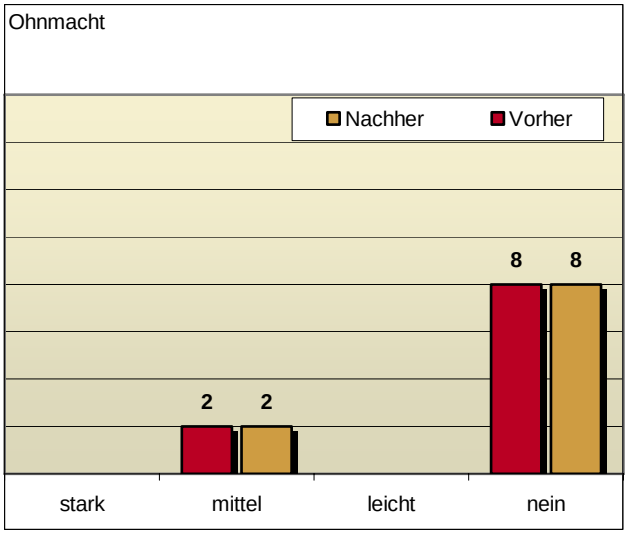




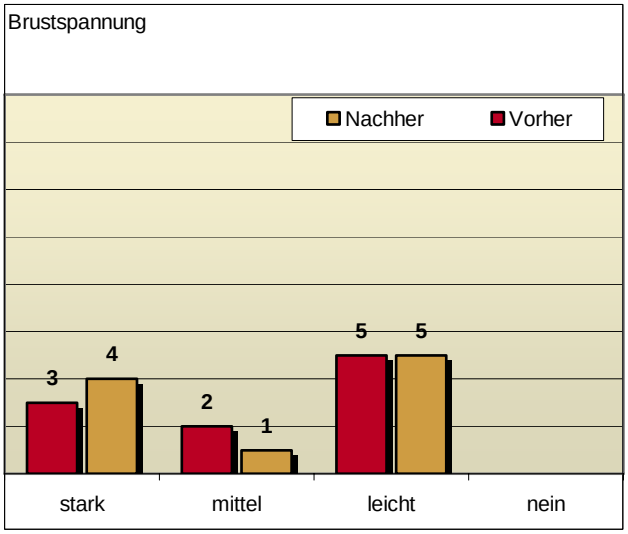


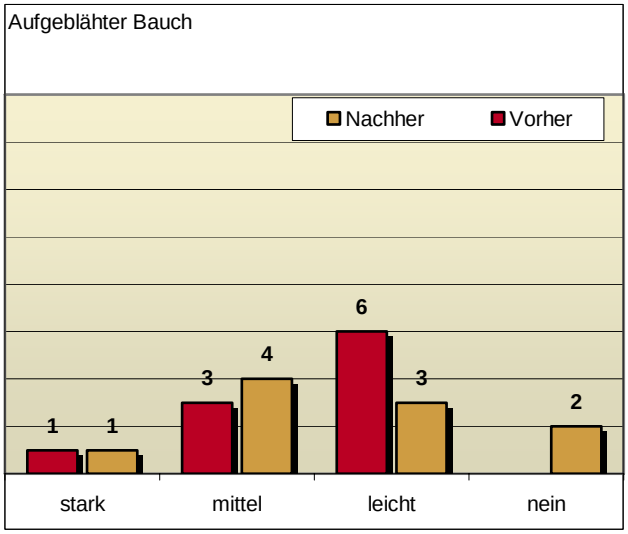


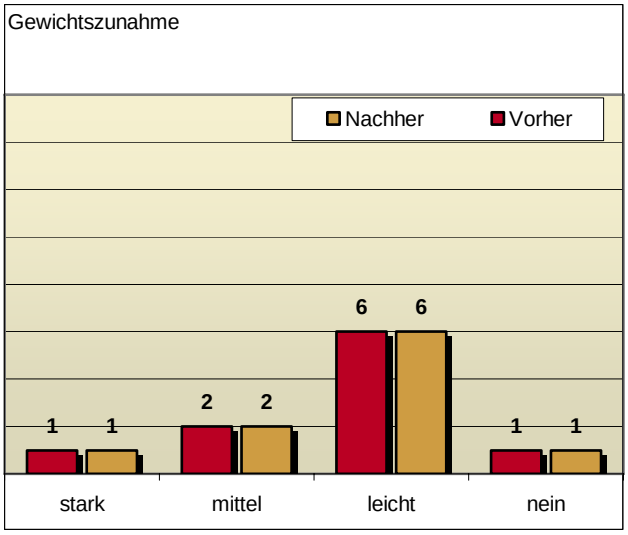


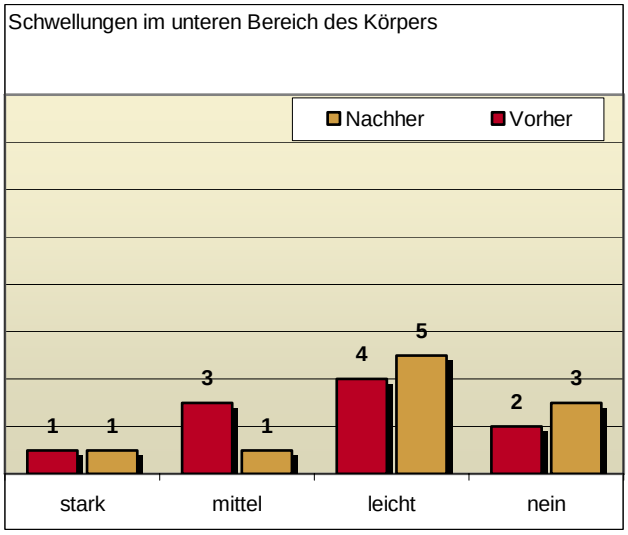


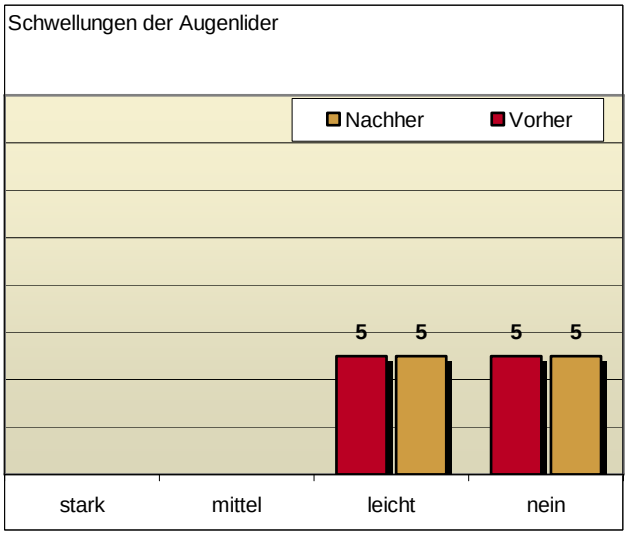
PMS-H-Hyperhydration



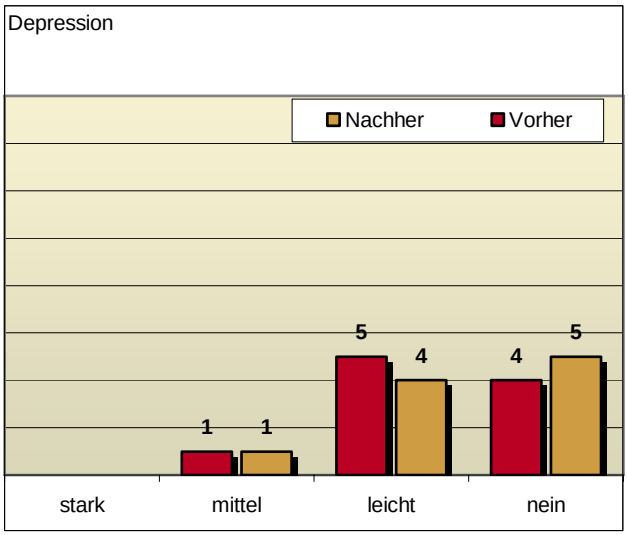


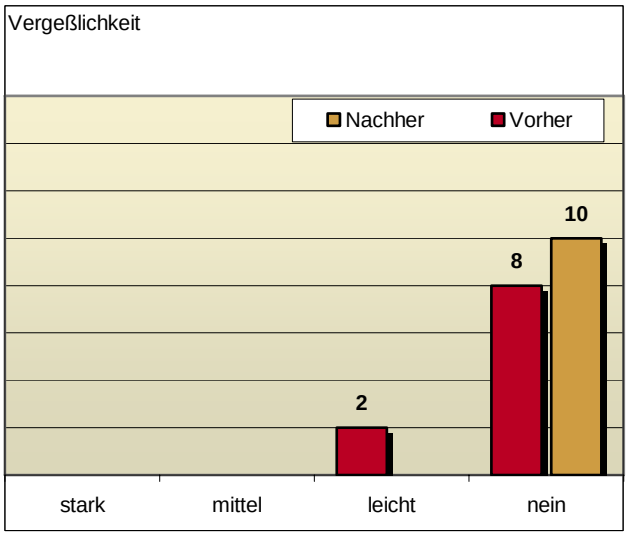

Gewichtszunahme
Nachher
Vorher
1
1
2
2
6
6
1
1
stark
mittel
leicht
nein

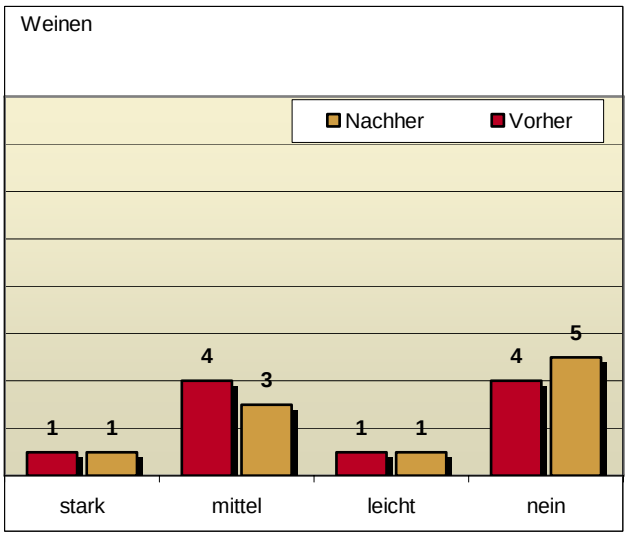

Schwellungen im unteren Bereich des Körpers
Nachher
Vorher
1
1
3
1
4
5
2
3
stark
mittel
leicht
nein

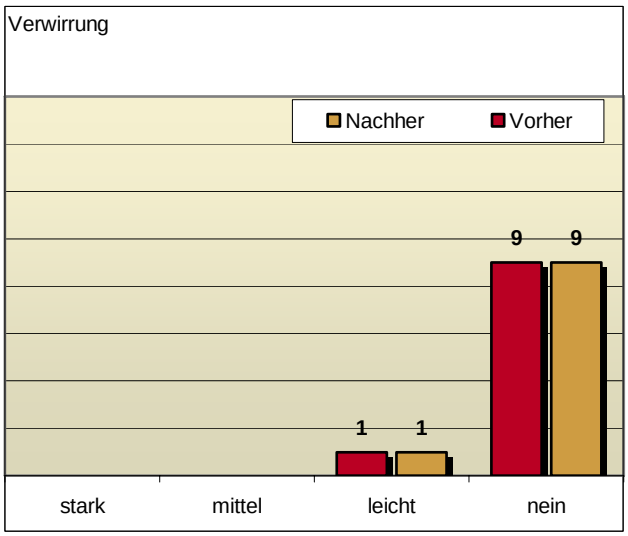

Schwellungen der Augenlider
Nachher
Vorher
5
5
5
5
stark
mittel
leicht
nein

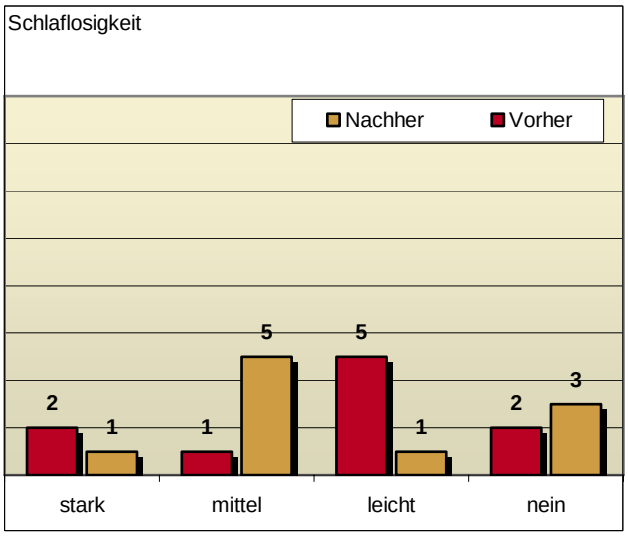
PMS-D-Depression



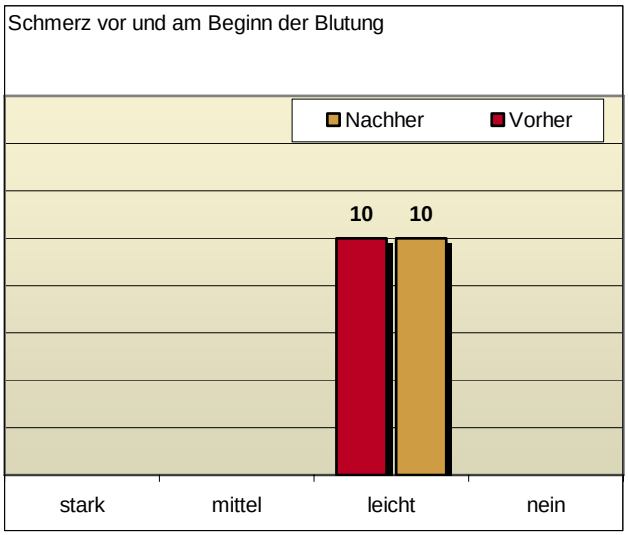


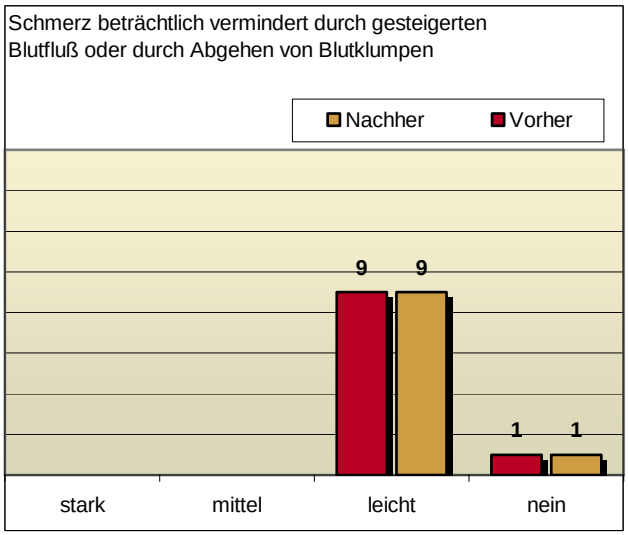


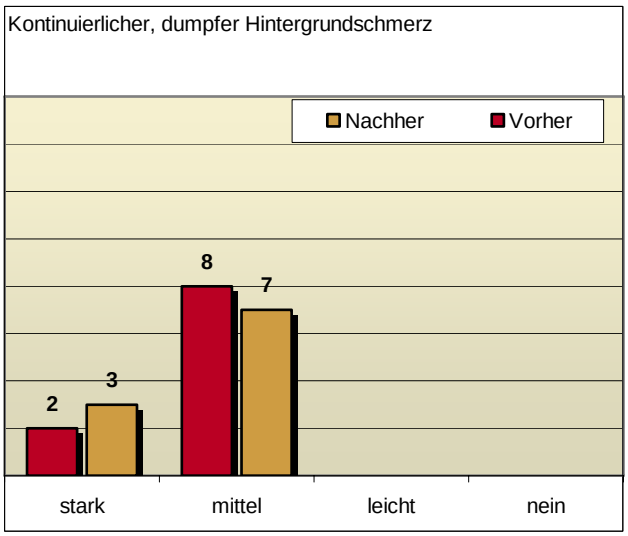


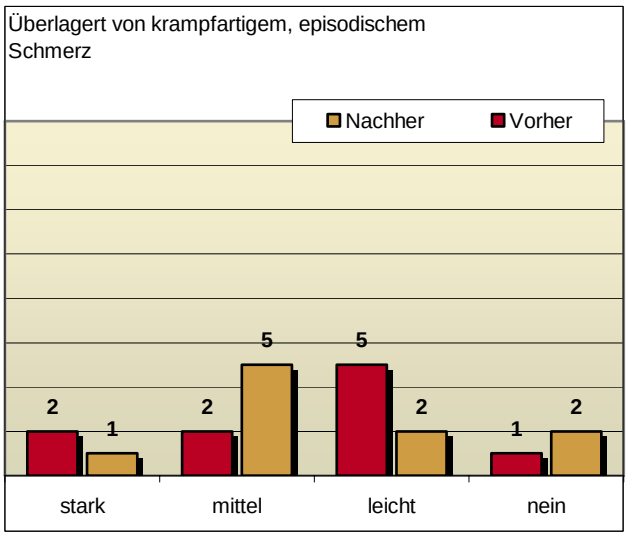


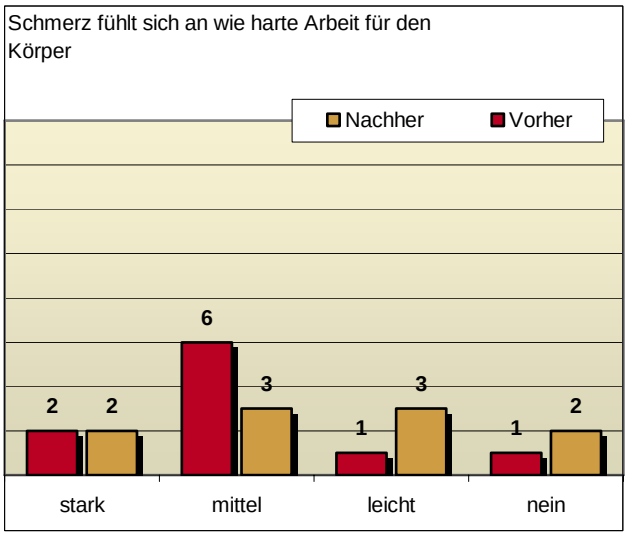
PMS-P-Pain

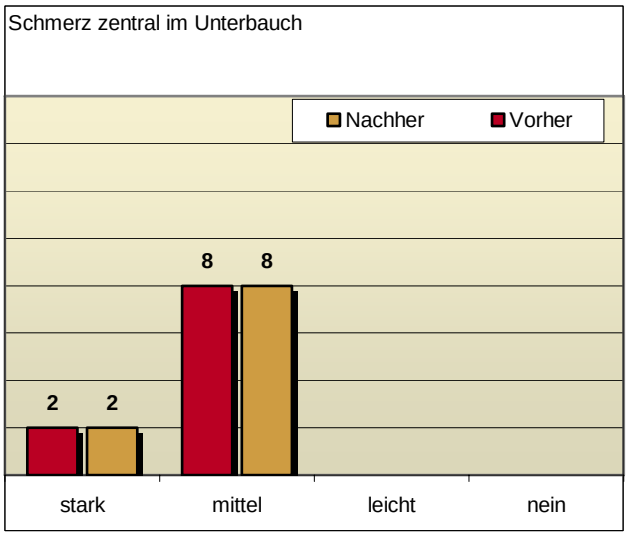

Schmerz vor und am Beginn der Blutung
Nachher
Vorher
10
10
stark
mittel
leicht
nein

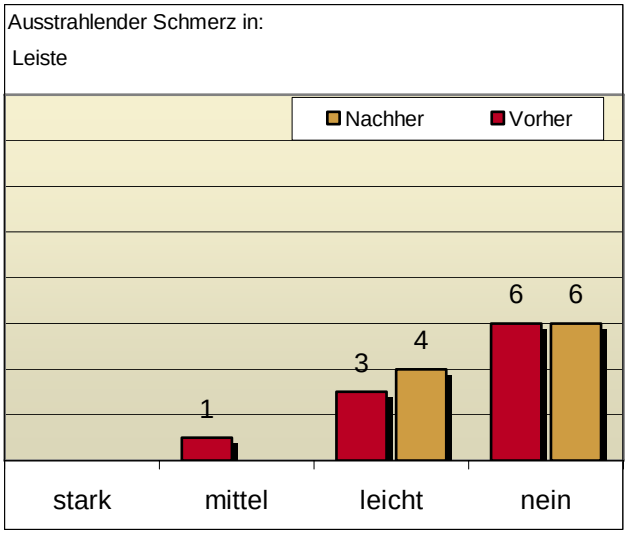

Schmerz beträchtlich vermindert durch gesteigerten Blutfluß oder durch Abgehen von Blutklumpen
Nachher
Vorher
9
9
1
1
stark
mittel
leicht
nein

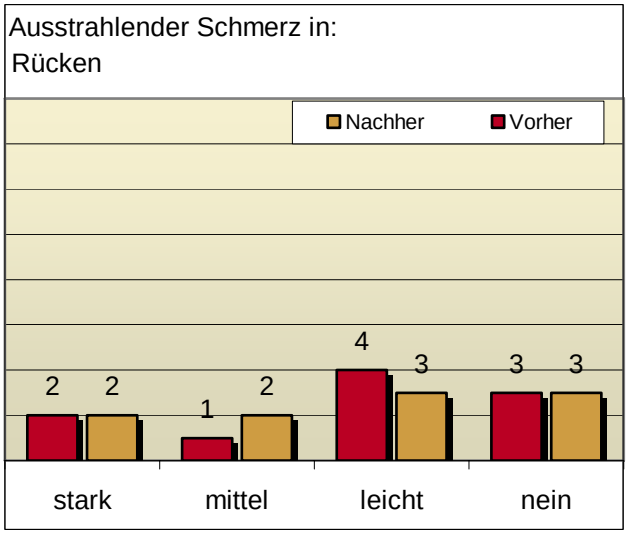

Kontinuierlicher, dumpfer Hintergrundschmerz
Nachher
Vorher
2
3
8
7
stark
mittel
leicht
nein

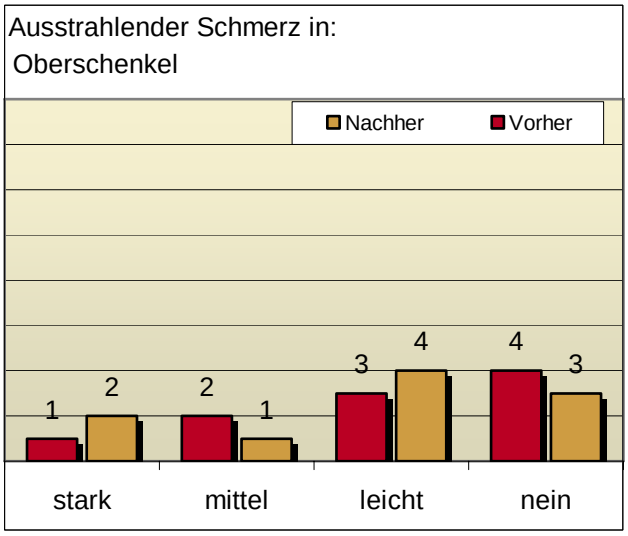

Überlagert von krampfartigem, episodischem Schmerz
Nachher
Vorher
2
1
2
5
5
2
1
2
stark
mittel
leicht
nein

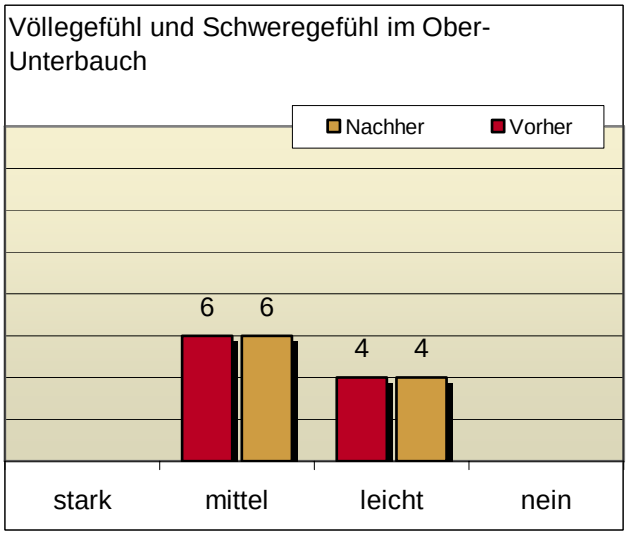

Schmerz fühlt sich an wie harte Arbeit für den Körper
Nachher
Vorher
2
2
6
3
1
3
1
2
stark
mittel
leicht
nein

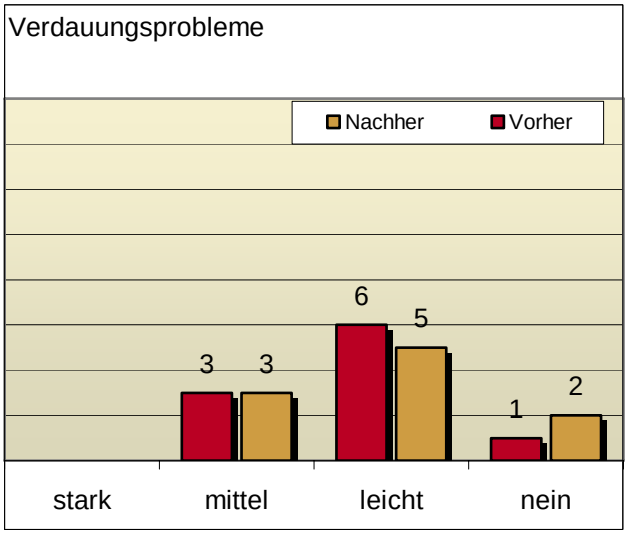

Schmerz zentral im Unterbauch
Nachher
Vorher
2
2
8
8
stark
mittel
leicht
nein

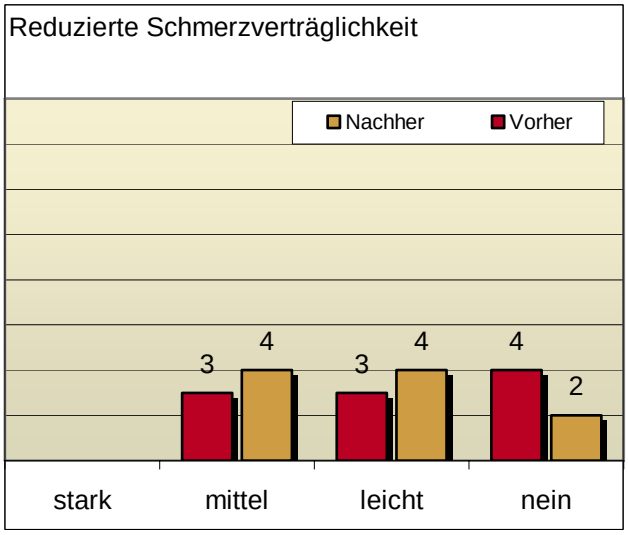

Ausstrahlender Schmerz in:
Leiste
Nachher
Vorher
1
3
4
6
6
stark
mittel
leicht
nein


Ausstrahlender Schmerz in:
Rücken
Nachher
Vorher
2
2
1
2
4
3
3
3
stark
mittel
leicht
nein


Ausstrahlender Schmerz in:
Oberschenkel
Nachher
Vorher
1
2
2
1
3
4
4
3
stark
mittel
leicht
nein


Völlegefühl und Schweregefühl im Ober-Unterbauch
Nachher
Vorher
6
6
4
4
stark
mittel
leicht
nein


Verdauungsprobleme
Nachher
Vorher
3
3
6
5
1
2
stark
mittel
leicht
nein


Reduzierte Schmerzverträglichkeit
Nachher
Vorher
3
4
3
4
4
2
stark
mittel
leicht
nein

11. Abstract

The Influence of Osteopathic Treatment on Congestive Menstrual Disorders and Premenstrual Syndrome

by Ingrid Riepler-Reisecker

Subject: this study aims at finding out, if it is possible to exert a positive influence on congestive menstrual disorders and premenstrual syndrome by using osteopathic treatment and thus improve quality of life for the women concerned.

Study design: comparative, clinical study plus statistical evaluation.

Method: 10 patients of the test group are given osteopathic treatment in their premenstrual phase for a period of 4 menstrual cycles. Before the first and after the last treatment patients fill in a questionnaire.

The results of the test group are compared with a control group who also fills in two questionnaires at an interval of 4 cycles.

Results: the results of the study are statistically evaluated and represented in diagrams. On the basis of this evaluation we can clearly see a statistical tendency towards iomprovement for both congestive menstrual pain and premenstrual syndrome. However, due to the low number of participants the results cannot be called statistically significant.

Conclusion: on the basis of the statistical results it has been proven beyond any doubt that it is possible to influence hormonal dysbalances of women by giving them osteopathic treatment. Function of organs in the lesser pelvis is improved by a harmonisation on the structural, cranial and visceral levels and congestive pain symptomatology is considerably reduced.

Key words: congestive menstrual pain, PMS, quality of life, hormonal dysbalance from an osteopath's view.