Our Blue Wonder

Sex hormones and varicose veins

M. Oswald

Ins Innere der Natur
dringt kein erschaffner Geist.
Glückselig!
Wem sich nur die äuß're Schale weist.

To Nature's heart
No living soul can reach.
Thrice happy he
To whom she shows
Even her outer shell.

Albrecht von Haller
1708 – 1777

Our blue wonder

Translation from the German original
“Unser blaues Wunder”

Photo: in the *Museo la Specola Firenze*

Our blue wonder 1.0

My father often warned me that I would experience my blue wonder if I did not follow his well-intentioned rules. Fear of blue wonders, which I imagined to be terrible, made me do what I had been taught for the first half of my life.

As a result, I also spent the first 10 years of my phlebological work in fighting varicosis and the apparently nonsensical thrombotic occlusions of veins. For several years now, I have changed my focus and view veins in a manner that deviates from the goal-oriented approach I was trained to use. Since then, I have experienced what my father threatened me with, but with increasing enthusiasm. I experience my blue wonder with something that concerns us all and that we all have: a true wonder – it always has been and it always will be – **the blue wonder, our veins.**

Outside of the usual publication possibilities, I would like to inform you here of a wondrous observation and present a few thoughts of my own, whose correctness I cannot prove, but which are possibly of general interest.

I cannot explain wonders.
Being inquisitive as any living thing, I made a discovery that goaded me to continue thinking about how such understood, wonderful, natural health could suffer such uncomprehensibly strange, wondrous, denaturating changes, the symptoms of the illness – the illness itself.

Augsburg, September 2005
Martin Oswald M.D.

Sex hormones and varicose veins

What is clear at this time – what we know

We have all learned that varicose veins are related to hormones. It is known that more women are affected than men. Pregnancy, with its hormonal changes, is especially known to cause changes in the veins of the legs.

How hormones get into the leg

It is clear, the hormones move downwards by means of the arteries and then ..... they create a vein dilation there. --------Like in the arm—? **Or?** Well – ***s o m e h o w*** ..... I mean, the arterial hormone-saturated blood flows along the leg and, of course, also into the arm and so on – in pregnancy, there are no varices in the face, on the abdomen, or otherwise, but ..... that's just the way it is ..... ***well*** ... we just know that's the way it is.

The hormones of the pelvic region create isolated dilations in the leg, which are then varices .... because weight increases and the pressure of the fetus on the blood vessels and the **upright posture** of humans – nature wasn't able to keep up in the evolution of the veins ..... and so ..... Well, –

I guess that's just how it must be!

What do hormones do to the veins?

I have little concept of everything that hormones can do to the veins. I have read that sex hormones have an expanding effect on the wall of the blood vessel[1]. The concentration of hormones in the blood of women and men changes in daily and monthly cycles - as well as over the course of one's life. The body can maintain functional balance through regulation of the number of receptors for these hormones in the wall of the veins. This balance is necessary in order to ensure the stability and function of the veins during hormone fluctuations.

Put simply, the number of hormone receptors in the walls of the blood vessel increases with a lower hormone concentration in the blood. Vice versa, there are fewer receptors when a large amount of hormones are in the blood. I do not know the location of sensor for the hormone concentration. However, I suspect some kind of central region. The body will hardly have sensor control units in every centimeter of vein, when it can assume a uniformly high or uniformly low concentration everywhere in the body. It is currently presumed that the receptor density is controlled by means of the arterioles.

Hormone concentrations rise during pregnancy, as well as during the monthly cycle. Nature is prepared for this and protects the blood vessels by reducing the number of receptors for the hormones. This happens with all of the blood vessels in the body, meaning in the veins of the legs as well.

[1] **Göretzlehner G.,** Simon E., Auswirkungen der Sexualsteroide auf die Gefäßwand, Phlebologie 1999; 28: 40-4

In nature, there is no derailment of this system, which has worked so well for millions of years; not only with people, but with a multitude of other living creatures with hormones as well. Pregnancy is a natural, physiological process without punishment of the mother for the creation of new life.

Dear Reader,

Before my writing becomes too dubiously unscientific; I am now coming to the scientific part:

It contains observations from and thoughts on special varicose veins in men and women.
The core of the work deals with a study performed on men. However, I constantly come back to the women when it seems pertinent. I hope that this does not confuse you. We find the analoque veins in both sexes and, as you will see, also an identical pathology.

In the second part, I deal with additional aspects, which can result from a phlebo-logical consideration with hormones.

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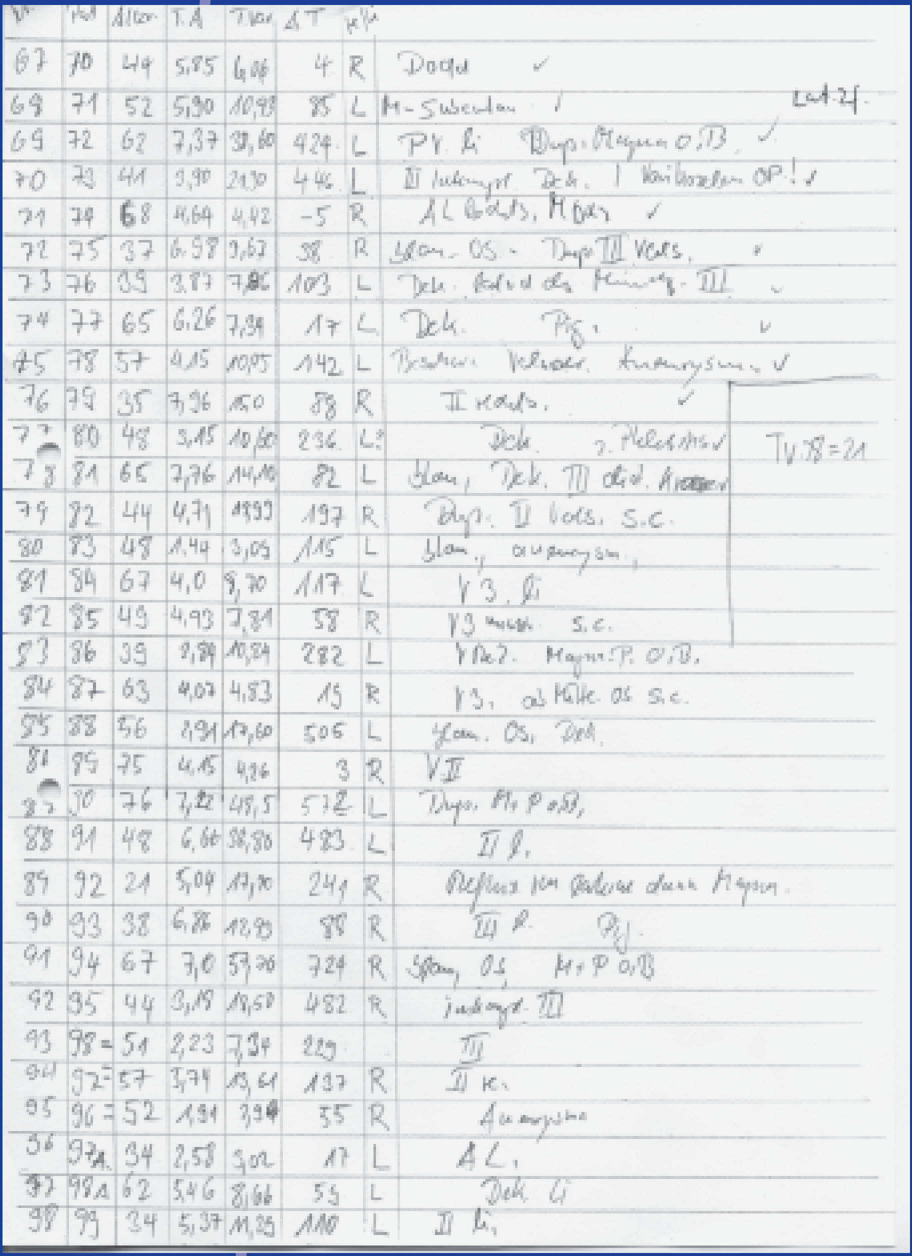
Study
01/19/05 to 07/27/05

2.0



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71	74	68	4,64	4,42	-5	R	[illegible] ✓
72	75	37	6,98	9,62	38	R	[illegible] ✓
73	76	39	3,87	7,86	103	L	[illegible] ✓
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83	86	39	2,89	10,84	282	L	[illegible]
84	87	63	4,07	4,83	19	R	V3, [illegible] S.C.
85	88	56	2,91	17,60	506	L	[illegible]
86	89	75	4,15	4,26	3	R	VII
87	90	76	7,22	48,5	572	L	[illegible]
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90	93	38	6,86	12,93	88	R	[illegible]
91	94	67	7,0	57,70	724	R	[illegible]
92	95	44	3,19	18,50	482	R	[illegible]
93	98 =	51	2,23	7,34	229		[illegible]
94	92 =	57	5,74	13,64	137	R	[illegible]
95	96 =	52	1,91	3,9[illegible]	55	R	[illegible]
96	97A	34	2,58	3,02	17	L	AL,
97	98A	62	5,46	8,66	59	L	Dek [illegible]
98	99	34	5,37	11,25	110	L	II [illegible]

Tv.78 = 21

Excerpt from the handwritten data recording:
TA: Testosterone arm, TVar: Testosterone Varices, ΔT: Testosterone difference in %

Amazingly high sex hormone concentrations in varicose veins of the leg

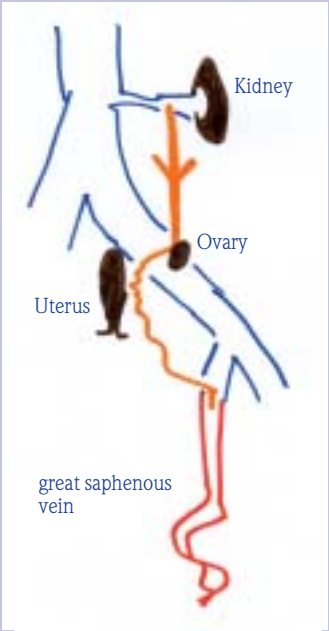
Not all varicose veins are the same. In spite of all of the complicated, even international attempts at classification, the character of the respective type of varicosis can often only be recognized with insufficient clarity.

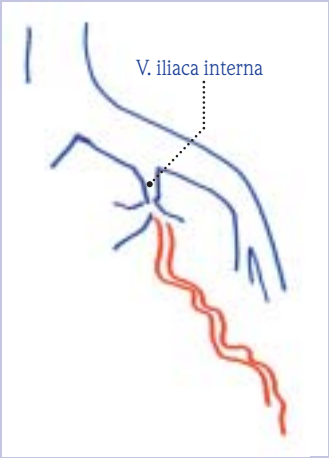
As a result, we frequently see varices, which do not fit the common cliché. They are bluer than others, are more prone to bleeding, create more localized overheating, period-related complaints, and can often not be assigned to the classical reflux concept with duplex sonography. *The incomplete insufficiencies of the great saphenous vein with an intact saphenofemoral valve function are a typical example for reflux forms, which cannot be explained with the common varice model.* For years, I have been dealing with such varices that probably »come« from the pelvis.

Here, the works of – and personal contact with – John Hobbs[2], J. Weber[3], A. Lechter[4], R. Schobinger[5], A. Fieri[6] and G. D. Richardson[7] were particularly helpful.

[2] **Hobbs J. T.,** The pelvic congestion syndrome, British Journal of Hospital Medicine, Vol. 43, 1990, S. 200-206
[3] **Weber J.,** Pelvines Stauungssyndrom in Diagnostik und Therapie, Vasomed 1999,11:202-7
[4] **Lechter A.,** Phlebology (1987) 2, 181-188, Pelvic varices and gonadal veins
[5] **Schobinger R.,** Das ileofemorale Dumping-Syndrom, Aktuelle Probleme in der Angiologie, 43 Der Oberschenkel, Bern:180-185
[6] **Pieri A.,** trans-vaginal colour doppler ultrasound in the diagnosis of pelvic reflux and of female varicocele, Phlebologie 1999, 52, Nr1: 45-51
[7] **Richardson G. D.,** Beck T. C., pelvic congestion syndrome: diagnosis and treatment, Aus NZJ Phleb 1999 3: 51-56

Recently I observe an increasing number of papers regarding the connection of the pelvic vein system from the V. ovarica and branches of the V. iliaca interna to the epifascial veins of the leg. In spite of this, – it seems to me – this venous pathology is given little scientific consideration – and even less in everyday practice. This is probably because we are not familiar with clinical and duplex-sonographic access and also because these forms of varicosis often cause less dramatic symptoms. But if they are operated on in a classical manner and clinically eliminated for the most part, they are more likely to recur.





▲ reflux from a branch of the V. iliaca interna

◀ reflux from the insufficient V. ovarica

The phlebological scientists are still working on questions, such as regarding the recurrence in the groin, by varying unsuccessful answers and taking such paths again and again and demonstrating their superior prowess to one another, but in the end they are all faced with therapeutic defeat in the face of the power of the cause of the recurrence. This force of varicogenesis or variconeogenesis is still unknown.

This force of varicogenesis or variconeogenesis is still unknown.

The concept of excessive pressure or excessive volume for a part of the otherwise perfectly developed life form, man, is uncertain and, yet, is spread – for lack of any other explanation – in its unchanged form.

As I recognized the old definition as unsuitable, I am delighted and refreshed by a new train of thought, regardless of whether or not its validity can be proven. In the worst case, it is just as wrong as the previous unsuitable explanations. However, it may possibly lead to solving the riddle of groin recurrence or even varicogenesis.

My Discovery

After noting from the descriptions of some female patients that they had pain in legs with varices shortly before and during the first two days of their period, the thought occurred to me that some substance from the pelvis is flushed downwards into the varice. There it triggers an irritation on the walls of the vein during this time.

In 1999, I held a lecture at the European Phlebologists's Congress in Bremen regarding my first experiment. I had determined estradiol in the varicose vein blood of women and compared the concentration to that of blood from an arm vein. The examinations were always performed patients standing up, as those women reported that the symptoms had disappeared while lying down. The result was sensational – at least for me.

I was *able to record much higher concentrations of estradiol in the varicose vein blood in several female patients.* The increases were up to 800 percent. In a control group with classical great saphenous vein trunk varices, for example, the hormone content was identical in arm and leg, as expected.

The publication of this observation in a small group of 10 female patients earned me the honor of subsequently being invited to several top-class scientific meetings and lectures. A study on possible histological differences between »my« hormone varices and the classical form caused me to suspect that such a varicose vein originating from the pelvis could also be present in men with »unusual« duplex sonography findings.

I therefore also started to examine sex hormones in the blood of men. After I had discovered hormone differences – testosterone in this case – in a small group also here, I planned a prospective study.

I wanted to clarify:

▶ how often hormone discrepancies exists among patients with varicose veins,

▶ what the ratio is regarding the concentration of testosterone in the varicose vein blood in comparison to the blood from a vein in the arm.

Unfortunately, I only became interested during the course of the examinations in whether there are indications in clinical, duplex sonographic or intra-operative findings for such hormone discrepancies. This is why data about these aspects are incomplete.

100 male patients – in order to keep the statistical numbers simple – with varicose veins of all types were accepted into the prospective study. Only small saphenous vein varicosis forms were not considered, as I initially assumed no correlation to the pelvic vein system here.

Age	Minimum	17
	Maximum	79
	Average	51

Method

The concentration of testosterone in the varicose vein blood was compared to the blood from the crook of the arm. Testosterone was determined as total testosterone in the blood.

Dependability of the test, deviation possibilities, collection procedures

Coefficient of variation of the test 2–8 % (laboratory test runs maximum of 5 %), intra-individual deviation possibilities up to 5 %. Overall, these parameter result in a deviation within the intra-individual serums of up to a maximum of 10 %.
In my evaluations, I allowed for a difference of up to 20 % as "the same".

The concentration in the varice blood may also depend upon the location of the aspiration in the leg. For this reason, I then documented each of these in the course of the study.
I attempted to aspirate as far proximally in the varice area as possible. Sometimes also duplex-controlled in the great saphenous vein of the upper thigh. Proximally because I had assumed a dilution of the testosterone level in the distal varice area and was excited to see large differences.

The blood samples were taken standing following the duplex-sonographic examination without a provocation maneuver. This means that the patient had been standing for already several minutes.
On the arm, I aspirated blood out of the V. cubitalis or V. cephalica, depending upon the individual situation in the crook of the arm.

The collection time was the first examination – sometime during office hours – thus completely regardless of the time of day. The testosterone level varies throughout the course of the day. As I was originally only interested in a possible percentage difference between the blood samples, the time of the blood collection was irrelevant. The ratio remains the same regardless of the level of the concentrations.

For the blood collection, I used a needle with a diameter of 0.8 mm for the leg, a cannula with a diameter of 0.55 mm for the arm. According to the laboratory technicians working with me, there are no differences in the measured values of testosterone based on the size of the cannula. As long at the blood is not hemolytic, the diameter of the aspiration cannula plays no role.

Results

From 100 male patients with varicose veins:

Testosterone concentration

The normal level is between 2.8 and 8.0 ng/ml.

Testosterone concentration		Number of patients
> 8,0 ng/ml	testosterone in the arm	0 %
< 2,8 ng/ml	testosterone in the arm	18 %
< 2,8 ng/ml	testosterone in the leg	3 %
> 8,0 ng/ml	**testosterone in the leg**	**52 %**

52%

Maximum level in varicose vein 120 ng/ml.

120 ng/ml

No patient had a value that was too high in the arm, whereas 18 % showed a testosterone deficiency in the arm. Only 3 patients had a below normal value in the varicose vein. In more than half of the patients, the hormone value in the varicose vein was above the normal range. The maximum value was 120 ng/ml. This is 15 times the normal amount!

Difference of the samples intra-individual between varice and arm

Difference leg / arm	Number of patients
up to and including 0%	6%
0% – 20%	16%
20% –100%	27%
100% – 200%	18%
200% – 1000%	30%
more than 1000%	3%
∑ Patients with difference over 100%	**51%**

Class division: "more than … up to and including"

51%

Minimum difference	– 11 %
Maximum difference	**2870 %**
Average of the increase when over 20 %	294 %

2870%

In 6 patients, the concentration in the leg was the same as or less than that in the arm. 22 patients had a difference of up to 20%, which is in the range of possible error sources.
78 patients had differences.
51 % more than 100 % with a maximum of 2870 %.

Difference > 100 % and conc. > 8 ng/ml 43 %:
43 patients, whose difference was more than 100%, also had a concentration in the varice that was above normal.

Conc. leg > 8 ng/ml – Minimum difference value 38 %:
If a value over 8 ng/ml was found in the varice, the difference in relation to the arm was always increased, by at least 38 %.

Information regarding the aspiration side

Right leg	39 %
Left leg	45 %
No information	16 %
Maximum concentration right	98 ng/ml.
Maximum difference right	1710 %
Right leg + difference > 100 %	33 % of the patients aspirated on the right side

Since we know from the varicocele that the left V. spermatica is more often insufficient, I wanted to portray or exclude a similar situation by means of numbers. According to the results, the right leg seems to be less affected than the left.

Clinical notes:

It is noticeable that many varices appear particularly blue and cause swelling or other symptoms, all the way up to varice bleeding. I did not address this matter consistently enough in this study. There seems to be a tendency, however, for the varices with hormone increases to cause increased propensity for swelling and symptoms during heat.

Duplex findings:

I did not always document the precise differentiation of the types of refluxes, as I still described my duplex findings in practice according to the x-ray-orientated reflux gradation by Hach only.
As a result, I am lacking many more precise differentiation criteria for many patients, which I would like to know more about now:
Reflux with Valsalva, only with Valsalva, only with decompression, aneurysmatic dilation of the great saphenous vein, or subcutaneous path in the thigh are criteria, which can play a role and are visible in color-duplex-eximination only.

I found no hormone increase with varices of the type
▶ Acc. lateralis
▶ Dodd
▶ Isolated calf varicosis
In addition, the cases of recurring varicosis from an isolated great saphenous vein stump insufficiency sometimes had a remarkable difference in the concentrations, but were in the normal range. (max. conc: 7.37 ng/ml)

Hormone increases
▶ With described negative reflux in straining maneuver, but clear reflux with decompression in 33 cases – only 3 of such patients without clear hormone difference.
▶ Also patients with Valsalva-positive reflux paths of the great saphenous vein. Only mentioned with 9 patients, with 3 patients no major testosterone difference.
▶ All varices of thigh type, without positive findings in the saphenofemoral junction, which did not belong to the group of Acc. lateralis, had a clear hormone increase.

Intra-operative notes

I have now operated on 45 patients. This does not correspond to the frequency of indication. It could be more. The reason for this statement is that the examinations are very current, meaning that they took place in the first half of 2005. The waiting time for an operation date can currently be up to 6 months, however. Thus, some patients have definitely not been operated on. Unfortunately, I also only began to document peculiarities intra-operative in the course of the study.

With 6 patients, I describe clearly ectatic cranial tributaries to the great saphenous vein crosse. In all of these cases, the hormone differences were > 100 % or the concentration > 10 ng/ml.

If I mentioned a clear increase in diameter of the varicose vein running from proximal to distal, a massive hormone increase could be measured (2 cases).

I find the description of the varicose vein as heavily aneurysmatic in 8 cases. 6 times, there is a clear hormone difference. In 2 cases, however, a Dodd-Perforans and another hormone-identical varicose types are mentioned as heavily dilated.

Discussion

There are apparently two forms of varicose veins.
▶ varicose veins without and
▶ varicose veins with sex hormone increase.

Varicose veins without testosterone difference

(up to 20% deviation range)

Physiologically, we expect that testosterone is present in all extremity veins in the same concentration. It is produced in the testicles and reaches the left renal vein or right V. cava directly by means of the Vv. testiculares. By means of the lungs and heart, it is then uniformly distributed throughout the body. In venous blood there should be an equal concentration in the arm and the leg. However, I know of no higher testosterone consumption in the arm when compared to the leg. The varice blood is probably blood that is pressed downward out of the V. iliaca externa – as it is refluxing –, but here too, we initially expect no hormonal change.

This consideration corresponds with the first group of varice-patients. 22 percent of the patients were in this group. These were cases, in which a varicose forms outside of the venous branches of the pelvis. Isolated perforans varices of the thigh and calf type therefore only carry blood from the deep vein system of the leg. The same is true for Acc. lateralis-insufficiencies. The reflux in the varice path does not absorb any hormone-saturated blood.

When dealing with branches to the great saphenous vein that were already isolated by means of prior operation and recurring varicosis, in which reflux only comes from the junction stump, no more hormone flows into the varice path in the distal direction.

Some cases of trunk varicosis of the great saphenous vein with positive Valsalva reflux also exhibited no hormone differences.

Varicose veins with testosterone increase – gonadal varicosis

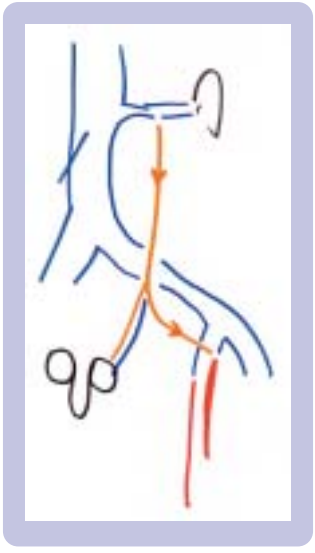
78 percent of patients

How the hormone entered the varicose vein

The increase of testosterone or estrogen is probably caused by a pathological return flow or supply flow of hormone-saturated blood into the varice from the organ of production.
In men, these are the testicles; in women, the ovaries.

The connection to the gonads leads me to call this change a gonadal varicosis.

We phlebologists know little of the region above our groin horizon. As a result, it was a difficult journey for me at first when I ventured into this unknown landscape of veins. Especially difficult because this anatomy was not a part of my everyday business. Other colleagues, such as gynecologists and urologists, were the keepers of this part of human anatomy, I thought. But it is these colleagues who define the groin as their own horizon and do not deal with varicose veins of the legs. And so – I crossed my boundary.

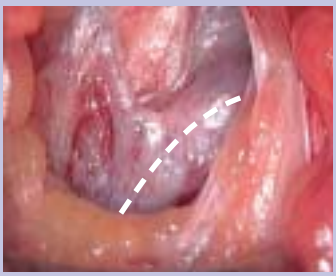


There are connecting veins between the male genitals and the great saphenous vein. As there is a radiologically proven connection vein between the V. ovarica and the area of the thigh veins in women, these will surely also exist in men from the V. spermatica to the penis, scrotal skin, and prostate with veins starting there going to connection veins in the soft tissue of the groin and thigh.

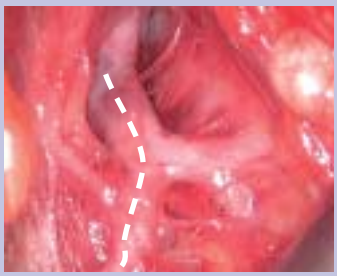
Tributaries from cranial and entering into the great saphenous vein junction are of particular importance. Blood from the cranial lateral and medial vein region can flow into them. The V. epigastrica superficialis, the V. circumflexa ileum superficialis, and the V. pudenda externa with its variations are structures with which we surgeons are familiar.
Colleagues who do not perform surgery or who use intravenous therapies, often miss these noticeably dilated indications with their thickened walls.

Intra-operative site (right groin)

great saphenous vein



▲ cranial, lateral supply flow to great saphenous vein without dilation

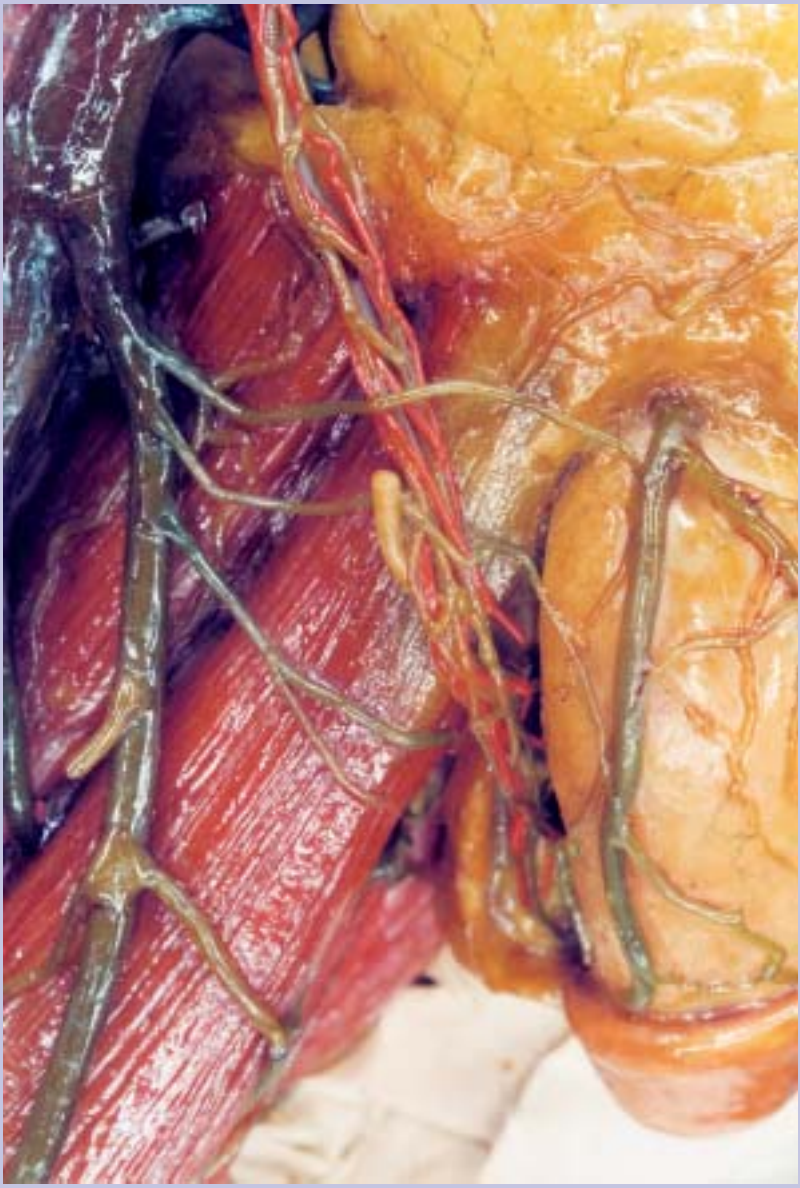


▲ dilated medial supply flow with incomplete great saphenous vein varicosis (same female patient)

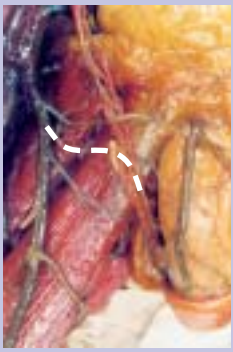
But surgeons have also not pointed out this condition sufficiently to date. For example, I have never consciously observed that a varicose continued from a dilated vein of these tributaries that branch from cranial, lateral, and medial directions. I know of no images of varicosis of the lower abdominal skin (except for after thrombotic occlusion of the V. iliaca as collateral circulation vein) or of the leg, nor of a varicocele, which started at an insufficiency of the saphenofemoral junction and ran upwards with reflux.

It is always the V.acc. lateralis or medialis or the great saphenous vein itself, meaning the veins that branch distally into the great saphenous vein junction, which change to become varicose. Dilated proximal tributary veins in the groin are thus suspect for me as a matter of principle. Thus, I assume that their dilation is not the start of a varicose vein, but rather a part of a varice path beginning further up, which simply extends further downwards into the leg by means of the great saphenous vein.

From the plexus pampiniformis, a vein branches over to the vein system of the leg, which I was able to photograph in an anatomy preparation – cast in wax – in the Museo La Specola in the Natural Science Museum of the University of Florence. This may serve as a visual aid for the processes. The individual anatomy in the groin has many different variants.



▲ wax preparation of veins (right groin), Museo la Specola Firenze



I still find the question of why this connection exists to be exciting. Does blood always flow there? If so: in which direction? Is it possible that it flows in different directions in different body positions or movements?
The connection vein fundamentally branches both into the Plexus pampiniformis and into the great saphenous vein.

Where is the beginning? Where is the end?

Is it physiologic that blood from the testicles flows into the great saphenous vein junction?

During a Crossektomy – meaning while lying down – bleeding occurs when we remove the clamp from tributary veins cranial to the junction. Intra-operatively, at least, blood flows to the spheno-femoral junction. As a result, a hormone increase would be expected with a reflux there into the great saphenous vein downward in all "classical" great saphenous vein varices.

In case of complete great saphenous vein insufficiency, the main reflux comes from the V. femoralis. Bleeding with opening of the simply small-diameter cranial branches in classical cases is minimal intra-operatively. Thus, only a small "physiological" supply of blood can occur from the connections to the v. spermatica. The varice hormone concentration in these cases will be only slightly raised – if at all.

However, perhaps it only bleeds here because the counter-pressure from the great saphenous vein is missing in such a non-physiological intraoperative maneuver.

With the incomplete great saphenous vein insufficiency, on the other hand, I often find cranial branches wide with thickened walls or dilated. Here, the saphenofemoral transition has no reflux. Thus, all blood that flows downward at decompression maneuvre must originate from a supplying side branch. Assuming that these side branches – branching into the saphenofemoral junction or beneath this into the great saphenous vein – have connection to the hormone producing organs of the pelvis, we should find much higher hormone concentrations here than in the arm. Intra-operative macroscopic results correlate with the respective laboratory results.

If a small amount of blood flows from the Plexus pampiniformis to the great saphenous vein, this may be physiological. If a large amount of blood flows in these communicating veins – if the hormone concentration is clearly increased in the varices – this must be a pathologically altered shunt connection in this direction.

Increased testosterone

- ▶ in the vein blood of the leg is non-physiological.
- ▶ is to be expected in varices of the great saphenous vein type.

A large difference in comparison with the venous blood of the arm and above normal values indicate a gonadal varicosis.

Importance of the side distribution

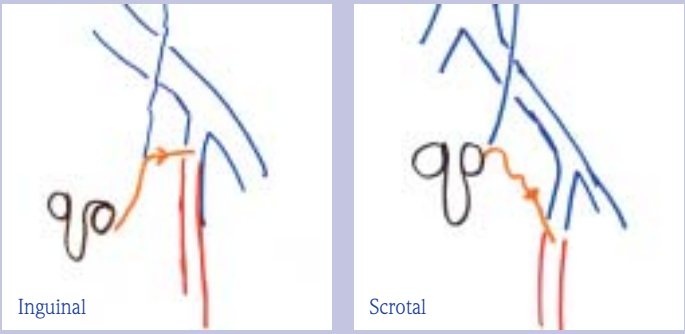
I was only able to determine a slight side preference for the left leg. Due to the fact that varicoceles form more often on the left side, this was expected. The pathology on both sides could be caused by contralateral insufficient inflows from left to right or by inherent right-side insufficiencies.

My previous further diagnostic procedure

In the heat of my joy at this discovery, I initially informed all patients of the result of their hormone examination. In a manner similar to the well-known return flows from the pelvis among women and their methods of treatment, I recommended the obliteration of the V. spermatica through radiological intervention. The first 3 diagnostic procedures were sobering. In spite of clear hormone differences between the varice and the arm: none of the patients had a demonstrable insufficiency of the V. spermatica in the retrograde radiological depiction. In order to avoid additional, senseless invasive examinations, I stopped the procedure. Here, I missed several patients, whose subsequent examinations then showed signs of insufficiency.
Unfortunately, it is not possible for me at this time to provide more information regarding the precise insufficiency pictures of the 100 patients. This is the main focus of therapists. Sonographically, I had attempted to find the V. spermatica or connection branches to the soft tissue cranially from the saphenofemoral junction, but did not make any progress and the invasive diagnostics did not seem to me to be sufficiently justified.

There are definitely complete insufficiencies with reflux in the proximal portion of the V. spermatica and others without.
If the V. spermatica – retrogradely – portrayed is sufficient, I can only imagine a flow into the system of the leg, when there is some kind of a gradient; so the blood flow escapes in the direction of the groin through a communicating vein, from where it then branches into the varice area.

Gonadal varicosis of the caudal, incomplete type



Mixed forms

Just as there are patients with great saphenous vein varices, those with small saphenous vein varices, other with both, on the left and the right, only on the left, nothing on the right, small saphenous vein on both sides, etc. – there will also be cases with isolated gonadal, isolated extragonadal reflux path, forms of transitional types up to those with contralateral reflux origin.

Are the hormone values pertinent?

We observe the fact that varicose veins fill at differing speeds after lying or sitting on a chair particularly when marking the varicose vein before a therapeutic operation. Some – perhaps those with small lumen pelvic supply flows – even require several minutes until they fill to their full dimensions.

This observation seems to indicate that – in accordance with the Trendelenburg test – a varicose vein, regardless of type, does not fill due to arterial feed through the venules from below, but rather always from above. This observation is decisive.

Thus, I initially had doubts about the pertinence of my values, as I sometimes aspirated a large varice and sometimes a small one. Thus, I had aspirated blood at different speeds. I thought that if I quickly pulled out a large volume with the plunger of the syringe, I might possibly have a great deal of blood from the varice leg filled by venules located distally from the aspiration point in the sample. However, if nearly all of the blood in a varice comes from above in standing, the aspiration speed during collection is of no importance.

The assumption is supported by the fact that all blood flows downwards and by the observation that the great saphenous vein often has a very small diameter in healthy persons, shows hardley a sign of flow – thus little blood is transported into this blood vessel from below.

Can the testosterone also come from somewhere else?

Estrogen can also be formed from androgen in the fatty tissue, I was told by a gynecologist. If there were a similar situation among men, where the testosterone also came from the leg, this could influence the hormone concentration in the varicose vein. However, after discovering that no hormone increase exists in cases of isolated calf varicosis – meaning cases, in which venous blood can apparently only come from the venous system of the leg –, I believe that I can refute this argument. In addition, when people are adipose in the lower extremities, the same is often true of the upper extremities as well.

Hormone increases would therefore also be expected in the vein blood of the arm.

Increased hormones in the varicose vein blood only comes from the regions of production, the gonads.

Importance of the differing levels of concentration

It can be assumed that a high pathologic concentration of hormones will have a correspondingly powerful specific effect on the blood vessel wall – meaning relaxation of the smooth blood vessel musculature with corresponding dilative effect. If the testosterone level is above the normal range, the regulation mechanisms are overtaxed. The greater this concentration, the more frequently pathological effects occur.
51 patients were above the normal value in the varices with a maximum value of 120 ng/ml.
Half of the patients with varices have a hormone increase above the normal value.

This massive dilative effect is able to dilate originally small veins into large hemodynamically relevant varice formations and thus make a varicose vein important, which possibly would have remained clinically concealed without the hormone content. This moment is assigned great importance in the **development** of varicose veins or recurrences.

Gonadal varicosis and recurrence

For as long as therapy has existed for varicosis, the propensity for recurrence has remained an unsolved problem. All manner of technical procedures have not led us to a definitive prevention for recurrence. Currently – as was the case 50 years ago, by the way[8] – the information regarding this ominous problem usually lists the recurrence rate as being between 20 and 40 percent. This information points out the phenomenon, but also contains the statement that the remaining 60 percent of the patients are permanently cured. These patients therefore did not suffer from the malady that we believed we were treating: a chronic degenerative disorder in the veins of the leg with a fateful propensity for recurrence – an incurable disease. After the treatment, they are free of varicose veins forever.

[8] **Jäger F.,** Krampfadern, Leipzig 1947 p. 89

The varicose vein itself is thus not incurable, but rather only a certain group, which has yet to be identified.

In the everyday work of surgery, there are no other veins in the body (except for the hemangioma), which lead to new formation after ligation. If a blood vessel is ligated, it remains "sealed" there. This is not the case with all of our varicose veins.

The explanation that it is simply the pressure on the wall of the vein, which is the cause of a recurrence, is not very plausible, as this condition is the same for all patients. But not all patients suffer a recurrence.

Therefore, there must be a circumstance, which plays a role with a group of people suffering from varicosis, which is simply lacking in others. We noted that a recurrence always occurs if the proximal insufficiency point is not closed. As is the case with insufficient operation. The same thing inevitably takes place when we remove the great saphenous vein, but the insufficiency point is located further up in the pelvis and we do not recognize it.

In addition to the possible remaining proximal reflux portion, I am attempting in the following to provide an explanatory model based on my study results.

All refluxive veins of the varice patient, who has no hormone increase in the varicose vein and thus no pathological connection to the venous system of the gonads, are removed by means of an operation. He remains free of recurrences.

Sex hormones are, as mentioned above, dilative substances, whose effect on the wall of the blood vessel is buffered by the presence of more or less of the respective receptors.

The density of receptors is defined centrally and controlled by means of the arterioles or a respective nerve impulse.

Nature assumes a uniform distribution of the hormones for the entire body by means of the arteries - except for the hormone transporting gonadal veins. Naturally, this control system is perfected as a prerequisite for a healthy circulatory system.

In the case of a gonadal varicosis, however, hormone-saturated blood flows from the gonadal venous system downward to the leg. Thus, it enters into a venous system, which is preset to the general, much lower hormone concentration. The resultant unprotected vein wall – far too many receptors for the actual amount of hormone present in the lumen – becomes a "victim" of the now unchecked dilative effect of the hormone. A pathological dilation of the veins occurs … Varicose veins are dilated veins.

When such patients are operated upon in the classical manner, the communicating branch to the gonadal system remains and can accelerate a recurrence over time due to its dilative content. The remainder of the varicose vein regains clinical importance especially due to its high hormone level and the varicose veins "grow again", as our patients so aptly describe.

The appearance of recurrences after a thorough operation in the leg may always have been a case of gonadal varicosis.

Conclusion:

There are pathologically high concentrations of sex hormones in varicose veins of the leg among women and, surprisingly, also among men.

The percentage of these patients is very high.

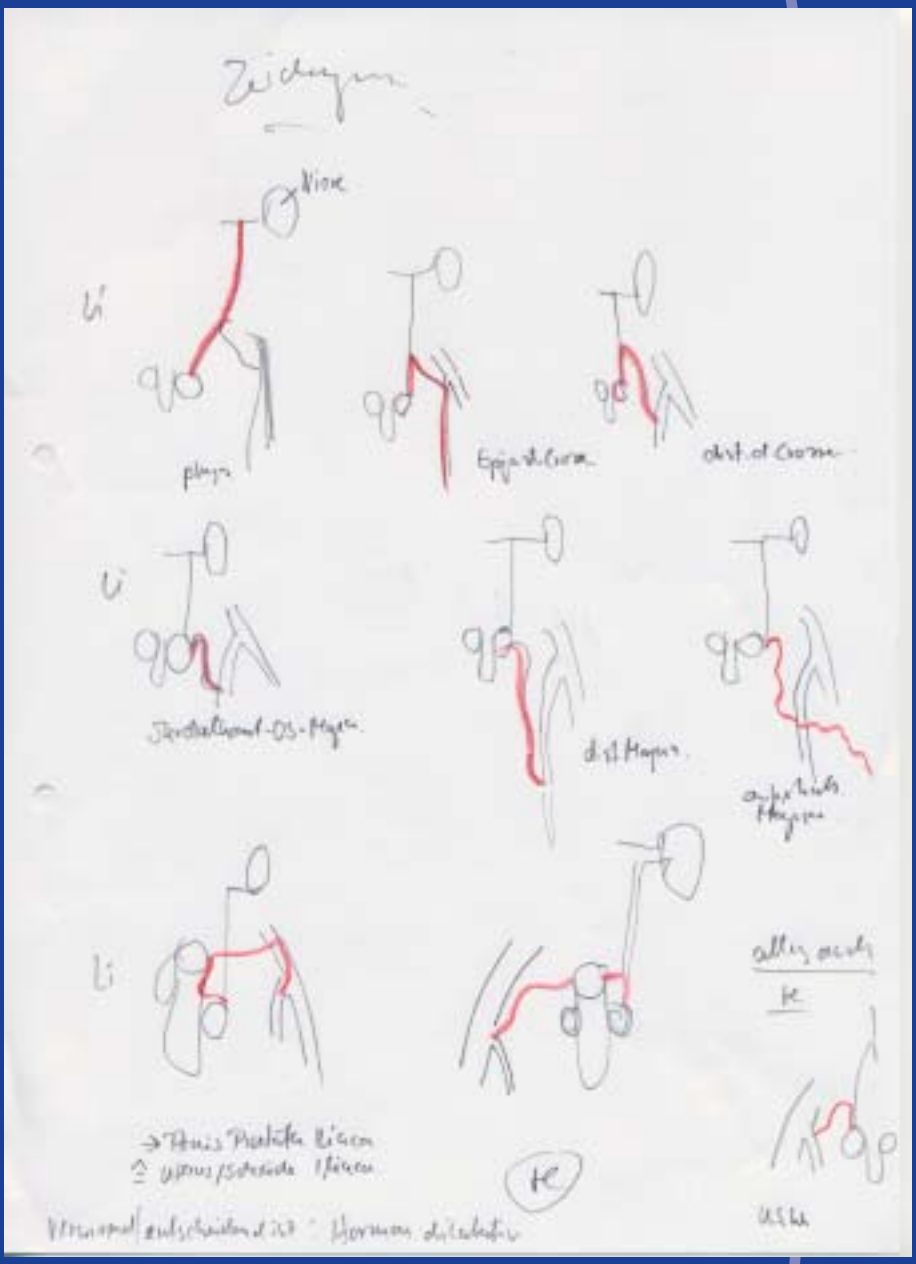
The abnormal hormone supersaturation in the varicose vein blood is often amazingly high.

Large hormone differences between varicose vein and arm blood in conjunction with a duplex sonographically demonstrated intact junction region in the Valsalva test and reflux with decompression are criteria, that together indicate a flow of blood from the gonadal venous system.

The determination of the difference alone does not yet prove the course of the reflux of the varicose vein.

Pathological high concentrations of dilative substances cause pathological vein dilations.

The cause of the recurrence in the groin after operations or other medical forms of destruction is still unclear.
The determination of hormones may be a pathway toward solving the riddle.



Thumbsketch of possible types of reflux

While pondering my discovery, additional questions and aspects came to my mind.

Additional Aspects 3.0

Nutcracker syndrome blood from the renal vein?

The sonographically and radiologically portrayable phenomenon of compression of the left renal vein by the superior mesenteric artery is suspected by many authors as the cause of varicocele (male and female), and possibly also for gonadal varicosis. It is referred to as the nutcracker syndrome.

The authors assume that a constriction of the V. renalis leads to a prestenotic accumulation and thus to a collateral formation by means of V. ovarica or spermatica. Thus, an accumulation is supposed to cause the varicocele in men, the gonadal intrapelvic varicosis in women, and also the pelvic originated varices in the leg. Actually, sonographic images allow us to see a clear, constant flow moving downwards in the gonadal veins, with radiology indicating a connection to the varicose vein in the pelvis or also in the leg. Although the model that describes pressure as the cause seems plausible, several observations contradict this pressure theory.

First of all, I know of no prestenotic dilations in the circulatory system, whether arterial or venous – but the vein is dilated here. In addition, nature never makes a detour through an entire extremity during a typical obstacle-induced collateralization. It always seeks the shortest or best possible escape blood vessels. I cannot find this practical approach in the varicose vein, even in the male varicocele.

Shorter venous collaterals to the heart would be possible through the V. suprarenalis, high under the diaphragm into the V. cava. Here, collateralization does not occur, however, and definitely not the formation of a varicose vein in this direction.

In the nutcracker syndrome, the blood flows with a high volume from the V. renalis in the caudal direction.

With gonadal varicosis of the leg, however, just as with male or female varicocele, the blood seems – once it has reached the varice area – in a kind of venous cul de sac, brought into movement only as a result of movement, whether artificially due to provocation maneuvers during the examination or in everyday life as a result of changing positions. In any case, in patients who are standing still, I can scarcely see a downward movement in the varicose vein by means of duplex sonography – in contrast to the reflux in the parauterine veins during a transvaginal duplex sonography with an Ovarica insufficiency.

Here, a great deal of blood constantly flows toward the probe next to the uterus in clearly dilated veins. This blood must have an outlet somewhere. The already filled, non-flowing varice of the leg or testicle cannot take in any more blood. A venous shunt through the gonadal veins in the caudal direction and varicosis seem to have their own pathology, existing simultaneously and also independent of one another.

Does blood flow from the renal vein through the gonadal veins to the leg?

In part of the patients in the study, I also determined the concentration of DHEAS in addition to testosterone. Dehydroepiandrosteronsulfate is a hormone of the suprarenal gland and is mixed with the blood of the kidney and possibly carried to the junction point of the V. spermatica. I was curious as to whether I would also find increased levels of this substance in varices in the leg and thus be able to prove a reflux from the renal vein to the leg. In 5 cases, there was never an increase of this substance in the leg. In all of these cases, testosterone was clearly increased by up to 750%. Thus, I was not able to prove a reflux from the renal vein to the leg in this manner. In order to check this further, I would have to test for a kidney-specific substance in the blood.

Gonadal varicosis and varicocele

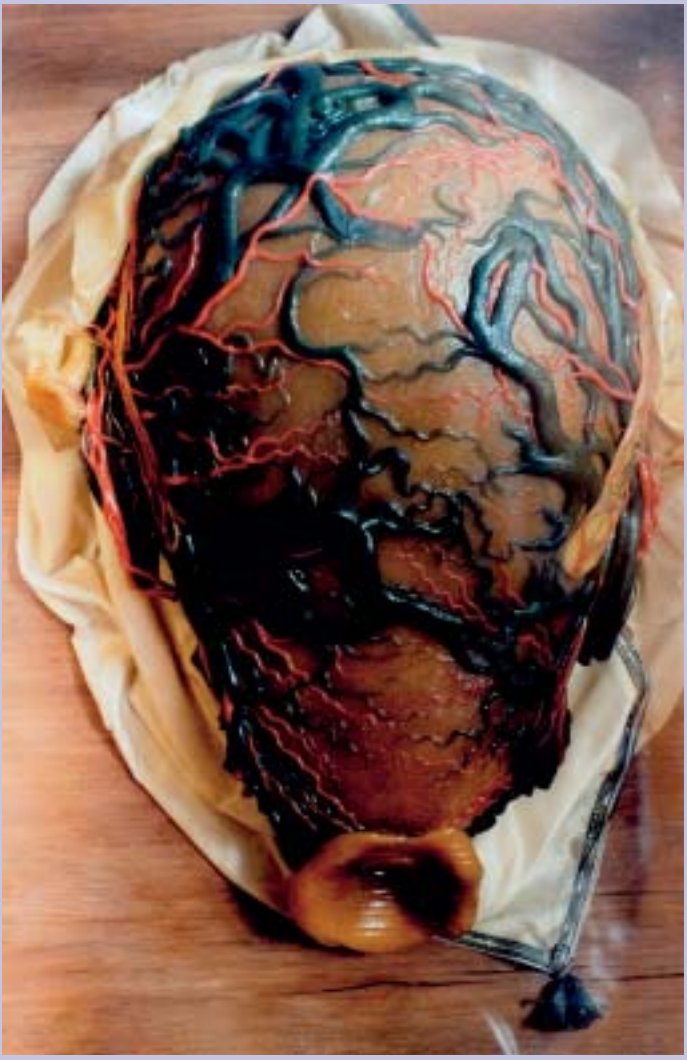
Do men with a testosterone increase in the leg also have a varicocele? I pursued this matter with only lukewarm enthusiasm. Central Europeans all wear underwear. Even if this article of clothing has only been covering the human body since the 19th century, it has now become a self-evident, almost “natural” part of the individual. I still hesitated to have the patients strip completely for my medical curiosity. So I simply questioned the patients. Only 2 patients had already been treated due to a varicocele. There is a varicosis of the v. spermatica, which develops in the scrotum and apparently a form, which leads to a leg varicosis. Possibly also both forms in combination.
In the future, I will pay more attention to the clinical and sonographic findings of the varicocele.

No thrombosis in the varicocele

Urologists never see a spontaneous thrombosis in the varicocele. They also see no flow of the venous blood once it has sank into the scrotum; no thrombosis in spite of a standing column of blood – amazing!
Stasis – even in a pathologically dilated vein – alone does not yet seem to trigger a thrombosis.

Pregnancy varicosis – different hormones from the pelvis? Different pathways?

As I was able to detect sex hormone increases in men and women in varices, the connections to the pelvic venous system are insufficient for both sexes, completely independent of the uniquely female phenomenon of pregnancy.



Uterus veins during pregnancy

The fact that many women who are pregnant in civilized industrial countries get blue legs even in the first weeks of pregnancy I explain as follows. Already before the pregnancy- similar to men - the pathological communication pathways between the pelvis and leg exist caused by a factor that I will address later.

However, during this period, there is not only a generalized hormonal change, which makes women appear fuller, prettier, and having better circulation. In the area of the fetus – the pelvis – there is fundamentally greater dilation of the blood vessels than in the rest of the body. (The Vena ovarica is initially only a few millimeters thick, but then becomes thicker than your thumb) I suspect that a substance – let us call it dilatin –, which takes effect in the blood of these blood vessels, makes extreme dilation processes possible here.
It could also be some nerve controlled system. However, such an approach creates no sensible explanation for the pathology in the leg, because the nerves only have an effect on the anatomically predetermined regions. Intrasanguinal hormones or agents that work in a similar manner can be washed away by the bloodstream and take effect in another location.
The asumed dilative substance dilatin, whose form is unknown to me is most likely carried into the leg by means of the predetermined pelvic originated – possibly gonadal – reflux pathways in the same concentration as in the pelvis and, similar to its physiological effect there, leads here to a massive dilation of the affected veins and blue coloration of the leg. (A particular effect on the intracutaneous veins is noticeable. This can also be an aethiological indication for the appearance of some mysterious spider veins … here, too, the veins turn blue, the bundle of veins coming from the leg, but are usually sufficient – what about in the pelvis?)

During the subsequent course of the pregnancy, in addition to the various blue discolorations a macrovaricosis of the pudendal varicosis type or a vulvavaricosis often occurs. They point towards the source of reflux in the pelvis.

The massive venous dilation in the leg usually subsides noticeably after delivery. I assume that whatever substance caused the blood vessels around the uterus to dilate so much stops and that the blood vessels in the leg, similar to those in the pelvis, return to their pre-pregnancy state – but only in a similar manner. Because varicosis persists in many women. I suspect that there is a difference between the ability of the veins in the pelvis to react to dilatin and that of the veins of the leg. Whereas the latter are designed by nature to have no contact with this substance, the veins of the female genitals are special veins, veins that change so drastically numerous times in a lifetime and can return to their initial shape under natural conditions.

In contrast to this, here the leg veins come into contact with this substance. Veins that were already dilated before the pregnancy (due to the other factor that must yet be mentioned) even if this often cannot be recognized. (Sometimes it can be recognized, because some women report slight varicosis before pregnancy, some have not noticed any.)

These leg-veins can also reduce in diameter to a certain degree postpartal, but always remain more dilated than before the pregnancy. If this form of dilation period is multiplied by several pregnancies, the veins that are affected in this manner become increasingly wider. This explains an increase in varicosis in many multipar women.

Pregnancy varicosis in this model is therefore considered to be a varicosis, which already exists before the pregnancy - a varicosis, which also exists among men –, which is only intensified in women during pregnancy.

When viewed from a phlebological-pathophysiological point of view, the long vasodilative period of the pregnancy in women is similar to the frequent brief episodes of vasodilation of the erection of men. An indication for this is the increased occurrence of varicocele during puberty among men, which is similar to the incidence of varicosis after pregnancy in women.

Other pathways of pathological flow from the pelvis to the leg.

In addition to the gonadal veins (V ovarica- spermatica), a reflux of hormone into the leg is also possible through insufficient branches of the V. iliaca interna in a number of ways. Connections to the great saphenous vein by means of side branches or to dorsal and lateral thigh varices are familiar in women and radiologically depicted.
The path through the V. iliaca externa in accordance with the ileo-femoral dumping syndrome (reflux by means of branches of the V. iliaca interna to the V. iliaca externa – into the great saphenous vein with or without insufficiency of the V. ovarica) has also been documented. We also find the same thing in men in urological literature[9].

[9] **Gall H.,** Die idiopathische Varikozele, Berlin 1990, p. 69 - 72

Matting – dilative substances

I suspect that the reason for matting after invasive therapy of certain types of varicosis to be similar to pregnancy varicosis. In the affected legs, there is still a connection to the dilative substances after removal of the macrovaricosis due to smaller varice branches to the pelvis that were not removed.
A higher level of hormones – whether this be estrogen, testosterone, or another dilative substance – now works on smaller blood vessels, which somehow had less blood flow before the operation. The occurrence of these intracutaneous venectasies after operation or sclerosing happens remarkably quickly. Anyway more quickly than the classic recurrence.
Matting exists in men and women. The same or a similar substance could be at work here.

Premenstrual pain in varices

The goal of my first study with estradiol in women was originally to find the cause of pain that many women experience shortly before and in the first 1–2 days of menstruation.
I came across an increase in estradiol in the varice blood. However, estradiol cannot cause the pain. At the time of menstruation, the concentration is at its lowest in the cycle. Out of curiosity, I asked the female patients to see me again 2 weeks later. Their hormone values were now clearly higher than during the period, the pathological ratio between the concentrations in the varice blood / arm blood was the same, but there was no pain at this time. So, a much higher level of estradiol in the varicose vein without triggering pain.
In further tests, I also found symptomatic women without a difference in the estradiol samples from varice and arm blood. Thus, presumably no insufficiency of the ovarian veins and yet cycle-related pain in the leg.
My explanation at that time was based on the model of pregnancy varicosis. Pain is triggered by substances, which are carried into the leg with the venous blood in varices. The varice portion has its reflux-origin in branches of the V. iliaca interna, which in turn are connected to a gonadal insufficiency or can be insufficient on their own. Thus, a large amount of estradiol is sometimes carried downwards in them or perhaps not. The substance that triggers the pain most likely comes from the region of the uterus. I suspect that it must be the same substance, which is responsible for the expulsion of the mucosa in the uterus. It now reaches the vein walls of the leg in these women by means of preexisting pathological flow pathways and causes irritation there, which is perceived as pain.

In conversations with gynecologists, **prostaglandin** came up again and again as a pain-triggering substance. Prostaglandin is formed in utero. My attempts to also test for this substance in the varice blood, have failed until now due to the fact that the laboratory tests for prostaglandin are very expensive.
In addition, the substance is very unstable. Financial and organizational obstacles have therefore prevented me from following this lead any further. What causes a similar change among men? What corresponds with menstruation when dealing with men? – Ejaculation?

Do sex hormones create varices?
There are also varices in the leg without an increase in hormones and varices in the leg, the groin, or on the stomach as a result of deep vein occlusions in the sense of physiological collateralization.
Up until now, I have described how I found hormone increases in varices and suspect other substances, which I cannot yet find. However, the dilative substances to not lead to varicose veins. They are probably a mitigating factor in their development[10]. It can also be assumed that they are responsible for certain characteristics, such as:
thin-walled aneurysmatic, menstrual symptoms, deeply blue varices, overheating, pain when subjected to heat, inclination for swelling, possible inclination for bleeding …
The varicose vein was already there. The hormones reach the region of the leg veins through the paradox "collateral circulatory system" of the varicose vein that already exists and create effects there that do not happen here from a physiological standpoint. Sex hormones do not cause varicose veins.

Sex hormones do not cause varicose veins.

If that is so, my tests and considerations could be completely unimportant. Basically, we will not use any other therapy and continue as before. We're O.K. We are the specialists and could be satisfied, because we are respected and paid for this.

[10] **Ciardullo A. v.,** high endogenous estradiol is associated with increased venous distensibility and clinical evidence of varicose veins in menopausal women, J vasc surg; 32, 2: 544-549

The reason that I am producing this pamphlet, however, is my own dissatisfaction with my current status. Sure, I can relieve the symptoms of many patients, can achieve apparent successes, but I am not always able to do this and the means are only troublesome at best and usually destructive in one form or another and ultimately crippeling. Even if I give the patient what he/she wants, this is done at the cost of taking or destroying an initially wonderful organ. This situation irritates me and is the cause for my preoccupation with the riddle of varicogenesis. Hormones do not cause varicose veins …

The consideration of the symptom and its various technical depictions will apparently never lead us to clarify the question regarding the cause for the change from physiology to pathology.

How do these varicose veins – all varicose veins – come to beeing originally?

Statistical records refute the well-known doctrine, that women have more varices than men. In developing countries, some of which have an original occurrence of varicose veins of 0 percent[11] – meaning that people never develop varicose veins, just like all other wonders of nature, all other animals on this earth, even though people walk upright there as well – and sometimes upright indeed! – In these countries, it is the men who develop the first varicose veins. I found a similar observation in literature on European[12] and Japanese[13] medicine around the turn of the last century. So, in the beginning men come more into contact with the factor that causes varicose veins. In the further development of civilized industrialization, women are then just as effected, with time possibly even more so.
D. Burkitt[14] mentions in a study from 1976 that one of 100 persons were effected from 1000 pregnant Indian women who were examined. One percent compared to the current

[11] **Trowell, H. C./Burkitt, D. P.,** Western Diseases their emergence & prevention Harvard University Press, Cambridge, Massachusetts 1981, 123
[12] **Zancani, A.,** Ueber die Varicen der unteren Extremitäten, Arch. klin. Chir. 1911; 91-142
[13] **Miyauchi, K.,** Die Häufigkeit der Varicen am Unterschenkel bei Japanern und der Erfolg einiger operativ behandelter Fälle, Archiv für klinische Chirurgie 100, 1913, Seite 1079-1093 Berlin
[14] **Burkitt, D. P.,** Varicose Veins Facts and Fantasy, Arch Surg-Vol 111, Dec 1976, 1327 1332

rate of pregnancy varicosis among German women of between 30 and 50 percent (estimated information from inquiries directed at friends in the branch of gynecology).
So it is not natural for man to develop varicose veins. In nature, there are no varicose veins. Man is a part of nature. No animal, no child has a varicose vein. It must be an unnatural factor, to which the miracle "human beeing" is adapting. This natural process of adaptation then leads to the answer in the form of unnatural varicose veins.

In my opinion, this factor is the chair. It is man "sitting" on the chair, or more precisely man being held by the chair. The sitter is fooled. Although you probably think that you are sitting as you are reading this, in reality you are doing nothing. The chair is holding you. I, on the other hand, am writing these lines lying on my stomach in my study. Of course, no animal sits on an aid that takes away the body's own tension in nature. Sitting on the ground is completely different from being held by a chair.

Men are the first to "sit" in developing countries, as they are the first to hold respected positions in offices, schools, or other chair-filled institutions of our civilization. Women follow later. But once the woman's household has become completely automated, then the only thing left for the woman to do – especially in cold countries and small isolated environments – is to "sit" on the supposedly natural (because they are used everywhere in the immediate environment) aids.
I see the chair in all of its destructive forms as the cause for varicose veins, and thus also for this form of gonadal varicosis. (A much more detailed account of this point of view regarding varicogenesis is too extensive to be included here – possibly in a separate publication in a similar form)

In general, the cause of the varicogenesis remains after the removal or destruction of the varicose vein in the leg. The patient continues to "sit" on a chair. If he had a varicose vein with sole involvement of the large leg veins, nature no longer has any other possible collateralization pathways from this point of the saphenofemoral junction.
Nature is finished.
The varicose vein will not appear again.

If, however, the original collateral pathways – which somehow begin outside of the saphenofemoral junction – originate from the pelvis, the large flow pathways are lacking after an operation and a temporary clinical improvement occurs.
But, nature still has ways of forming new branches from these "roots" that still exist in the groin. It still has the stimulus to form varicose veins due to "sitting" on a chair. The recurrence will form depending upon the original size of the suprasaphenal varice portion and certainly depending upon the hormone concentration therein in a more or less fast and distinct manner.

In all of our procedures, we merely eliminate the symptom rather than the cause.

If a person continues to "sit" on a chair and the varicose vein arises from a venous insufficiency origin that cannot be reached and completely eradicated, the person's body will always succeed in forming some sort of new varicose vein – it simply must.
The varicose vein is a chronic degenerative malady. It is a symptom for the lack of body tension; like many so-called civilization illnesses, it is a symptom of a complex system derailment with disfiguring, ugly, unpleasant, sometimes even fatal consequences for the individual, caused by the unnatural act of being held by the chair.

The appearance of varicose veins and their recurrence is only fateful, as long as we accept "sitting" on a chair as our fate.

It is a matter of course that the body will convert a vein into a varicose vein in a wondrous manner, if we force it to do so. Only nature can do this. We cannot subdue the power of nature, unless we destroy it completely.

Epilogue 4.0

The recognition of a gonadal varicosis does not lead immediately – and I almost hope never – to a practical new therapy. This is also not the sole purpose of our medical profession.
If we phlebologists are able to eliminate the fateful nature of illnesses and we only uncover loopholes in knowledge or therapy, we win credibility. To not know something is not the same as being incompetent, but rather is to be human and true. We are the ones who should understand correlations, without needing to be able to dominate them. The patient of today does not necessarily expect miraculous cures from the physician, even if medical marketing tries to convince us of this "truth". The majority of patients is satisfied if one can explain to them the phenomena of their bodies. Our goal should not always be to remove the reactions of the miracle "body" to unnatural stimuli by means of surgery or injections or to conceal them in some manner. In the long run, recognizing pathological correlations is the only way to protect the body against the natural reactions that force it to change and that make it ill, by keeping unnatural reaction-inducing factors away from it. In this case and in my opinion, the chair. There is a natural prophylaxis for varicose veins!

There is a natural prophylaxis for varicose veins!

The knowledge that we have regarding nature is the recorded result of observation and, at best, an incomplete image of the wonder.

Knowledge is only another form of belief in things that others say to us or that we figure out for ourselves. Proof of knowledge is – like knowledge – dependent upon belief.

However, the reality of nature is free of knowledge and the need for proof – and

wonderful!

I do not know, I do not want to know.
I am happy to observe and experiment …

Thank You

I would especially like to thank my two previous assistants, Dr. Ulrich Wohllaib M.D., now in Krumbach, and Dr. Christoph Heintze M.D, now in Berlin, who supported me with joy and enthusiasm in my first and decisive work. My committed pursuit of phlebology would have been impossible without the loving tolerance and the energy-provoking resistance of my wife.

Our blue wonder

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