



Ouabain - the optimal prevention of myocardial infarction

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Ouabain - the optimal solution for the problem of myocardial infarction by a well tried herbal substance and a newfound hormone

Extracts from the book "Ouabain - the possible victory over myocardial infarction"

by Rolf-Jürgen Petry* (e-mail: strophanthin@web.de)

(until now only in german language available: "Die Lösung des Herzinfarkt-Problems durch Strophanthin"

with a preface from Prof. Hans Schaefer / Heidelberg)

A shorter form of this article is also published at

<http://www.infarctcombat.org/heartnews-17.html>

main page = <http://www.infarctcombat.org>, the homepage of a clinic in Sao Paulo which had excellent results with ouabain in acute myocardial infarction (like other clinics and thousands of doctors).

Content of this article:

--- The therapeutical results of oral and intravenous ouabain in angina pectoris and myocardial infarction (and other diseases)

--- The stimulation of the sodium pump: ouabain and digitalis behave oppositely at the cellular level

--- Ouabain affects several components: heart muscle cells, nerves, arteries, erythrocytes

--- The effect of ouabain in other diseases

--- The false dogma of the bad absorption of orally administered ouabain

--- The detection of ouabain as a new hormone and the criticism of its pretended role as a cause of hypertension

--- The solution of the problem is not wanted: several decades of prejudice and resistance

--- Some mostly unknown pathogenetic aspects of acute myocardial infarction

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Introduction

One of the most necessary things in the contemporary medicine is to call attention to a topic that seems to be unbelievable at first sight: Ouabain (in German: g-Strophanthin), an extraction of an African plant called "Strophanthus gratus", which since 1991 is discovered as an endogenous substance - a new hormone, prevents angina pectoris and myocardial infarction by 80 - 100 percent without side effects. There is an overabundance of studies and documented experiences - the clinical ones mainly from Germany, Austria and Switzerland, but also from Italy, Great Britain, Brazil and USA, and the pharmacological studies from all over the world.

The effectivity of (especially orally administered) ouabain is quite obvious, even when the big clinical double-blind study is missing. But there is a mighty inscrutable opposition against the therapy with orally administered ouabain. Intravenous Ouabain was once a totally accepted, even celebrated therapy for decades, but as a substance without patent rights isn't assisted by a big pharmaceutical company.

Ouabain has a positive effect also in heart failure, hypertension (!), stroke, dementia, arterial occlusive disease (mostly in the legs), glaucoma, sepsis, endogenous depression, asthma bronchiale. The therapeutic success is sensational in arterial occlusive disease and dementia. (Because of a lack of time the references aren't indicated, but this will be made up soon.)

There are two wrong dogmas creating an impermeable wall:

1) Ouabain is like digitalis classified as a cardiac glycoside, with the indications "heart insufficiency" and "arrhythmia". Because digitalis has negative effects in angina pectoris and myocardial infarction and the story goes "that all glycosides act similarly, the outstanding therapeutic results of ouabain don't attract any attention at all in the medical establishment. Because of the classification as a heart glycoside, in the "Red List", the German pharmaceutical thesaurus, oral ouabain is associated with all the bad side effects which are noticed in digitalis medication but have never been noticed with the oral ouabain therapy.

2) In the textbooks is written that ouabain has a very bad oral absorption - but there are over 20 studies which indicate the contrary.

The worldwide best therapeutic results in angina pectoris and myocardial infarction

Short preliminary remark: Most references regarding the therapeutic effect are published in German. An important reference in English, which shows the reduction of cardiac pain during a bicycle ergometry by ouabain, is attainable with free full text as PDF on <http://www.pubmedcentral.nih.gov/picrender.fcgi?artid=458511&blobtype=pdf>

Another study shows the extensive parallel of the effect of oral ouabain with a nitro preparation, the generally accepted medicament used in angina pectoris pain attacks:

http://www.ncbi.nlm.nih.gov/entrez/query.fcgi?cmd=Retrieve&db=pubmed&dopt=Abstract&list_uids=6428911

Both studies are outlined below.

The best example for the indeed excellent therapeutic results of oral ouabain in angina pectoris and myocardial infarction is Prof. R. Dohrmann from Berlin (West), the leader of a public hospital, starting 1975 with this therapy. 1984 Dohrmann and

pectoris. 148 patients with severe stenosis visible in coronary angiography, who received for years all the medicaments modern medicine offers and who are dissatisfied because of continuous heart attacks and in part unpleasant side effects, have been switched over (with their agreement after an information discourse) to the therapy with oral ouabain from one day to the other, i.e. the other medicaments including the β -blockers (!) were discontinued immediately. After one week 122 of 148 patients were completely free from angina pectoris, and after two weeks this success could be seen with 146 patients. They were also free from the side effects of the former medication. The ouabain capsule which is dissolved in the small intestine(*) is sufficient in many cases (in this study 3 x 1 capsule daily), and when in spite of this prophylaxis there is a heart attack, the capsule for lingual absorption - to break with the teeth (**) surely helps in 5-10 minutes in almost every case.

* Strodival mr® (3 mg) from MEDA GmbH, Wiesbaden / Germany

** Strodival® (3 mg) or Strodival spezial® (6 mg).

No side effects

The other two patients (of 148) in the study of Dohrmann & Dohrmann 1984 had to stop the therapy because of some irritation of the digestion tract, the only harmless side effect which sometimes occurs. Strictly speaking, these "side effects" (up to diarrhoea and local inflammation of the tongue) are reversible phenomena that occur only during the absorption of the drug. Once absorbed, there are no side effects reported while the experiences with this medication from 1947 until now are counting several hundred thousand of "patient years" (number of patients x duration of treatment). There are still about 2000-3000 physicians in Germany who are using orally administered ouabain.

The dosage is declared as 1 - 4 x 3 - 6 mg daily; principally success and demand should regulate the dosage. There is no real danger of overdosage as often seen with digitalis preparations. Even suicide attempts with huge amounts of ouabain capsules (900 mg) miscarried and led only to some days of reversible ECG irritations. (Caution ! Intravenous ouabain only 0,25 mg or 0,125 mg with slow injection !)

By the way it is not a necessary condition to discontinue the former medication: there have never been any interactions between oral ouabain and any other medicine, even not with digitalis, on the contrary the experience shows that oral ouabain reduces the side effects of digitalis, when the latter drug is necessary because of tachycardia (see below, regarding the different molecular mechanism of ouabain and dogoxin). So the careful, perhaps doubtful doctor can initially prescribe oral ouabain additionally, and with the mostly instating improvement of symptoms, he can reduce or leave the former medication.

The study of Dohrmann et al. 1977 (2) deals with lingually applied ouabain (6 mg) to all patients coming to the hospital with severe heart attacks, acute myocardial infarction (AMI) or suspected AMI. In 170 of 264 cases (= 64 %) the attack was totally stopped within 5-10 minutes, in the rest there has just been an AMI in 55 cases, when there is no success to be expected any more, so that only in 15 % of the patients with Angina pectoris (but without AMI) there was no positive result with the first application of ouabain. The above mentioned study (1) shows that the optimal effect of ouabain is reached within some days after repeated intake.

In acute myocardial infarction (AMI) 1975-1987 Prof. Dohrmann used a new therapy with initially 1) i.v. cortison to stabilize the lysosomal membranes and 2) i.v. k-strophanthin (0,25 mg, repeated every 24 h). Additionally oral ouabain (lingual absorption, capsule to break with the teeth, 6 mg) was given when there was heart pain in the following days. The quota of nonsurvivors (30 days) after myocardial infarction previously was very high (38,8 %) because in Berlin (West) have been much

reached the best rate of survival in whole Europe - in the first year with oral ouabain 17,6 % nonsurvivors (3), and 1987, after 12 years, 15,1 % with experiences with 1056 patients (4). A multicenter study of northern Germany reported a quota of 26 % mortality (30 days) in 1977 (5). Prof. Dohrmann was outnumbered only by Prof.DeMesquita from a clinic in Sao Paolo (6) who used ouabain i.v. from 1972 -1979 in 1037 cases (until ouabain lost the license in Brasilia): they reached 9,6 % mortality during the stay in hospital, which could be reduced to 5-7 days with the ouabain therapy.

Another example is a coal mine in Gelsenkirchen/Germany (7) where the average number of workers dying because of acute myocardial infarction (AMI) in the mine, under the surface of the earth, was 3 every year; the way to the doctor lasted more than half an hour. After the doctors of the mine began with oral ouabain therapy directly in the mine in 1974 - given only when there was an acute heart attack, not prophylactically given -, the mortality concerning AMI was reduced to zero in the following 10 years with this therapy. In two cases there was no possibility to give oral ouabain (accidents) and the workers died. The cases concerning severe angina pectoris attacks and non-mortal cardiac infarctions that forced to drive the workers out of the mine were reduced by 80 % with oral ouabain in 1974-1984.

Salz & Schneider 1985 (8) carried out a placebo controlled doebel-blind study with 30 patients with coronary heart disease. They found after 14 days of prophylactic application of oral ouabain (3 x 6 mg Strodival mr® daily) a highly significant effect on the ECG (elevation of the lowered S-T-segment), the angina pectoris attacks and the subjective state of health in comparison of the verum and the placebo group and also an amelioration of hypertension. With placebo there was seen a deterioration of all parameters, see table below:

Salz & Schneider 1985, double-blind study

the effect of Strodival® in 16 patients

.....patients without change ...moderate improvement..essential improvement

exercise-ECG.....0.....5.....11

angina pectoris-attacks..1.....2.....13

subjective condition.....0.....1.....15

the effect of the placebo in 14 patients

.....patients with deterioration

exercise-ECG.....12

angina pectoris-attacks.....10

subjective condition.....10

In 1984, the small firm "Herbert Pharma" (the former producer of Strodival® - the only available ouabain medicament nowadays - in Wiesbaden / Germany) had made an inquiry to 3650 doctors with experiences of the oral ouabain therapy (9). Ca. 98,5 % answers were positive, 1,5 % were positive with some limitation and no doctor gave a negative feedback. Reading the released extract, the answers of 300 physicians, published with full address, is really convincing; very often they say: "excellently effective", "no side effects", "better than the rest", "I don't see deadly myocardial infarctions any more" and so on... (The small firm hadn't enough money to corrupt such a great number of physicians to make such definite statements. The author

Dr. Berthold Kern (Stuttgart), the explorer of the oral ouabain therapy in 1947, had documented very well his experiences from 1947 to 1967 with more than 15.000 patients (10). His patients, who were severely ill above the average, have had only 20 non-fatal myocardial infarctions, a very low number. Also after this period until his retirement 1991 he saw this good success, like the other doctors of his working group in and around Stuttgart and like the thousands of doctors in Germany who had used or are still using this therapy.

The double-blind experiment of Kubicek and Reisner 1973 (69) with angina pectoris-patients under hypoxia showed in 19 of 22 patients a marked improvement of the electrocardiogram (S-T-alterations) - in 7 cases a total normalization - after 6 mg oral Strophoral® (90 % ouabain, 10 % k-strophanthin) in comparison to a control group, and the result of subjective state of health is as follows: control: 18 patients with pain or giddiness and only 4 without trouble. After oral ouabain: Only 4 patients with pain or giddiness and 18 patients are without trouble. A placebo showed no effect. Digitalis had a negative effect, so that some experiments had to stop before the regular end (several drugs in differentiated dosis: Digoxin i.v. 0,4 mg, 0,8 mg, β -Methyl-Digoxin oral 0,05 mg, 0,2 mg, 0,8 mg). Also Sharma et al. 1972 (70) had similar good results with 0,7 mg i.v. ouabain. After ouabain the patients had much less angina pectoris pain using bicycle exercise. The ECG didn't change, perhaps because of the very high dosage. This is the corroboration of the therapeutical results reported by Prof. Dohrmann and others, see above.

There are many other articles about the experiences of doctors with orally administered ouabain. Manfred von Ardenne, the great scientist of the German Democratic Republic, made many investigations dealing with ouabain and wrote a review in English language about the therapy with orally administered ouabain (11).

The author could motivate some physicians to use ouabain. Their reports about a good therapeutical success are a good current authentication of the findings in the literature. The anthroposophic "Ita Wegman Klinik" in Arlesheim / Switzerland are using ouabain since 2002 with the expected success, also the "Neue Wicker Kliniken" in Bad Nauheim / Germany March 2004.

In Germany intravenous ouabain / k-strophanthin was accepted in acute heart insufficiency for decades until 1992 (13). Later incomprehensibly Digoxin was recommended and nowadays criticised because of too slow onset of action. Anyhow, 35 % of german doctors on emergency classified digoxin as indispensable and 15 % as desirable in 2001 (14). They are advised to use ouabain / k-strophanthin again, the cardiac glycosides with the quickest onset of action.

There are many pharmacodynamical studies dealing with ouabain, which are described in the next but one chapter.

Also with homoeopathic ouabain (D4, Strophactiv® from magnetactiv, Wiesloch / Germany) there is seen a quite good therapeutic success. The blind study of Hupe & Balint 1988 (12) showed that 60 % of the patients with angina pectoris improved their pathological ECK (S-T segment) after Strophactiv®, but only 15 % of the patients of the placebo group. In cases of emergency a regular dose (1 ml) will probably not be sufficient, but there are reports of a good effect of much larger doses, i.e. 25-50 ml (see below the expected blood levels after homoeopathic ouabain).

Ouabain and digitalis behave oppositely at the cellular level

and some other substances, is the sodium pump (= Na-K-ATPase), which is present in the wall of every cell in a great number and which is pumping sodium out of the cell and kalium into it. This is very important for many fundamental functions of the cell.

Ouabain is extensively used in many scientific in vitro experiments to block the sodium pump. In all textbooks and articles there is written that cardiac glycosides are inhibitors of the sodium pump. But for all that, this is only the toxilogical and not the physiological part of the truth: The inhibition of the sodium pump occurs only with high concentrations of ouabain (ca. 10^{-7} Mol to 10^{-3} Mol and higher), which are easily attainable in a laboratory. On the contrary, the low concentrations of ouabain, which are present in the human body after taking the medicine or naturally because of the endogenous nature of ouabain, have the opposite effect. There are over 50 unnoticed and unrefuted studies that report of the stimulation of the sodium pump by low doses of ouabain (ca. 10^{-13} Mol to 5×10^{-8} Mol), the first one to mention is Kurt Repke 1961 (15, with k-strophanthin), the discoverer of the sodium pump. The working group of Prof. Godfraind (Brussels) had made extensive studies on this topic (for example 16). The most important nowadays is Gao et al. 2002 (17) from the University of New York: "Isoform-specific stimulation of cardiac Na/K pumps by nanomolar concentrations of glycosides" in "Journal of General Physiology". They report about a stimulation of Na-K-ATPase by ouabain and dihydro-ouabain in guinea pig, canine and human heart cells. In the animal studies there was a stimulation of the high affinity isoforms alpha-2 (guinea pig) and alpha-3 (canine), but not of the low affinity isoform alpha-1 of NA-K-ATPase by up to 107 %. In human heart cells the differentiation is difficult because all isoforms, also alpha1, have equal affinities to ouabain.

Meanwhile some of the leading scientists are accepting the real cellular mechanism of low doses of ouabain, for example Prof. Schoner from Giessen / Germany in his parting lecture 2003:

<http://www.vetmed.uni-giessen.de/biochem/schoner/Abschiedsvorlesung/Folie25.PNG>

and Prof. Hakuo Takahashi (Osaka, personal communication), Ferrandi and Manunta 2004 (18), Balzan et al. 2007 (19), Hambarchian et al. 2004 (20) and Su et al. 2003 (21). The result of the stimulation of the sodium pump is a reduction of intracellular sodium and also a reduction of intracellular calcium because sodium and calcium are associated via the sodium-calcium-exchanger. For digitalis the data is poor, but it seems to be that digitalis is not able to stimulate the sodium pump (19). This is the explanation for the differences between ouabain and digitalis preparations seen in many pharmaco-dynamical studies (see below) and especially seen in the therapeutical results.

Actually oral ouabain can reduce the side effects of digitalis, as generations of physicians have used it (266). Already the world famous Prof. Edens (Düsseldorf / Germany) mentioned this indication in his classical "Digitalis-Fibel" ("Digitalis Fibula") (267), besides ten other cardiac indications being better influenced by ouabain than by digitalis. Pharmacological animal studies (268) show that an injection of a small dosis of ouabain augments the necessary amount of the following deadly dosis.

Two study groups have detected a new receptor for cardiac glycosides inside the cell, at the sarcoplasmic reticulum, the calcium store of the cell (22-23). The effect is a release of calcium. Ouabain acts tenfold weaker than Digoxin at this new intracellular receptor. Furthermore Santana et al. 1998 (24) could show, that already 0,1 nanoMol of digoxin, a concentration below those that are found in the human blood after digoxin medication, have the halfmaximal effect of opening the sodium channels for calcium influx, letting in 30 percent of the total calcium influx, whereas ouabain needs nearly the hundredfold concentration to achieve this effect. These differences

different therapeutical effect of ouabain and digitalis in angina pectoris and cardiac infarction.

The study of Horackova and Mullen 1988 shows a reduction of the Ca⁺⁺ content of isolated cardiomyocytes with low doses of ouabain, unchanged results with intermediate and a rise with high concentrations of ouabain (25).

Ouabain affects several components

In ischemia the activity of the Na-K-ATPase of heart muscle cells is diminished (26-28). This occurs in a situation where the number of sodium pumps is already reduced. The first sentence of Ko et al. 1995 (28) is: "Na-K-ATPase (sodium pump) may play a key role in the prevention of reperfusion injury caused by Ca⁺⁺ overload." By stimulating the sodium pump ouabain could prevent the ischemic and reperfused heart from calcium overload and could at least prevent the border areas of infarcted myocardium from necrosis and minimize infarct size.

Because the sodium pump is present in every cell, ouabain can influence not only the heart muscle cells. Because the cells exchange a part of their sodium for calcium, ouabain reduces the intracellular calcium concentration and therefore acts similar to a calcium antagonist. In addition ouabain combines the qualities of a whole train of medicaments.

--- Riehle & Bereiter-Hahn 1994 (29) showed that 10⁻¹⁰ Mol ouabain markedly increase fatty acid oxydation of cardiomyocytes (but not the oxydation of other substrates), which is diminished in ischemia. Gousious et al. 1967 made a study (30) which investigates the influence of ouabain in the isolated heart without ischemia: there was an increase in fatty acid oxidation in absence of increased uptake. In this connection it is interesting that carnitine, which is necessary for the transport of fatty acids into mitochondria and is also a strong stimulator of fatty acid oxydation, is taken up by the cell via Na-K-ATPase (31). Perhaps ouabain could prevent the loss of carnitine which occurs in ischemia (32).

--- The nerve cells as well have sodium pumps, they are even the cells with the highest frequency. Sharma et al. 1980 saw a reduced output of noradrenaline in nerve terminals with low doses of ouabain, no change with intermediate concentrations and an increase with high concentrations of ouabain (33). Gutman and Boonyaviroj 1977 (34) report that only low doses of ouabain are reducing the output of epinephrine and noradrenaline in the adrenal glands, while high concentrations of ouabain have the opposite effect. So the medicament ouabain lowers the level of stress-hormones in the whole body and especially in the myocardium and therefore acts also in the field of a beta-blocker. Agostoni et al. 1994 (35) report about a 50 % reduction of noradrenaline levels in patients with heart insufficiency over 3 months of intravenous k-strophanthin 0,125 mg daily (Digoxin: no effect)

--- Because of the sympathetic nervous system lowering (33-35) and parasympathetic nervous system enhancing effect (36-38) ouabain could dilate the small arteries inside the myocardium which are susceptible for a paradoxical reaction to ischemia: they are further contracting instead of dilating (39), which is mediated by the sympathetic (40).

Furthermore, in the same way ouabain could reduce coronary spasm which can promote the rupture of vulnerable plaques. In this way ouabain could reduce "coronary events". Perhaps ouabain has also a direct effect on the vulnerable plaques: In the study of Matsumori et al 1997 (41) ouabain caused the quadruplication

and TNF-alpha). Perhaps ouabain could show this effect also in the heart and coronary system and reduce the inflammatory process in the vulnerable coronary plaques.

--- Although in the last years ouabain is regarded as a blood pressure enhancing hormone only based on contradictory 1) studies in vitro, 2) studies with rodents and 3) statistical findings with ouabain-like substances (see below the respective chapter) there is considering evidence that oral ouabain and oral / i.v. k-strophanthin are lowering blood pressure in patients with hypertension (8, 9, 35, 42-44) - but not in normotensive or hypotensive patients (44). Two double blind clinical studies with orally administered ouabain (8) and k-strophanthin i.v.(35) show the in Germany wellknown ouabain effect of blood pressure lowering in patients with hypertension, as do many other reports (most of them in german language only), for example Qi et al. 2001 (42), who report that only i.v. k-strophanthin lowered hypertension and not digoxin. Agostoni et al (35) also observed a lowering of hypertension with k-strophanthin 0,25 mg daily (Digoxin: no effect) in patients with heart failure. Also Pidgeon et al. 1994 (45) demonstrated the reduction of diastolic blood pressure in healthy volunteers after 0,5 mg ouabain i.v..

DeMots et al. 1978 (46) gave the same dosis (1,05 mg / 70 kg, extremely high !!) of ouabain i.v. to patients with coronary artery disease with different velocities (10 sec., 2 min., 15 min.). The two quick injections showed an increase in systemic vascular resistance and a deterioration in myocardial lactate metabolism, whereas the slow injection of ouabain caused the contrary, a tendency towards a reduction of systemic vascular resistance and a profound improval of myocardial lactate metabolism, an effect of ouabain which is wellknown by older german pharamacology, together with a pH improvement.

Saradeth & Ernst 1991 (47) made a randomized, double-blind and placebo-controlled crossover-study with healthy volunteers and found a reduced rise of diastolic blood pressure in exercise after linguallly administered ouabain (6 mg).

--- Ouabain is enhancing the blood flow in the myocardium. This was revealed by von Ardenne 1991 (48) in a double blind crossover study using a szintigraphy with 99technetium showing a marked increase in myocardial blood flow by lingual ouabain (12 mg) in patients with angina pectoris. This confirmed the findings of Vatner & Baig 1978 (49) with ouabain i.v. in dogs.

--- Particularly important is the effect of ouabain on the red blood cells. Their diameter (8 micrometers) is bigger than the diameter of the capillaries, which they must pass (3 micrometers). To manage this, the erythrocytes have to make themselves very thin and long, really like a submarine. But because the acid and the oxygen radicals generated in ischemia or even by an overactivity of the sympathetic nervous system (see below, part two of the appendix) reduce the activity of the sodium pump of the erythrocytes, intracellular sodium and subsequent intracellular water accumulates and the erythrocytes become bulging and unflexible (50-52), like it is mimicked by high concentrations of ouabain (52). However, low concentrations of ouabain are able to stimulate the sodium pump of the erythrocytes. Strobelt et al. 1986 found a marked improved flexibility of erythrocytes even at low pH (53). So they get slim enough to pass through the capillaries again. Here ouabain acts principally like an ASS preparation, like Aspirin®. Saradeth & Ernst 1991 (47, double blind, crossover, healthy volunteers) found an enhancement of erythrocyte flexibility after oral ouabain (6 mg).

A disorder of microcirculation could get harmful. Because of the reduced flexibility of the red blood cells, the bloodflow is further reduced and the acid accumulates. That

could aggravate an ischemic situation, increase the necrotic area in the case of an acute myocardial infarction and perhaps even initiate the death of single heart muscle cells or even of bigger regions, if the microcirculation in the capillaries is downgrading up to a stasis. This could perhaps be a widely unnoticed mechanism of how a myocardial infarction respectively a myocardial necrosis could be generated. This could take place in such cases when a coronary thrombus is absent. There is a number of studies that report of a relatively low frequency of coronary thrombosis in myocardial infarction (54-66), for example 49 % in Murakami et al. 1998 (54) or 20 % in Doerr et al. 1974 (65, Prof. Doerr was the president of the German Society of Pathology). (For more research results supporting an alternative view of myocardial infarction pathogenesis please see the appendix.)

So the action of ouabain on the red blood cells is very important. With the linguallly administered ouabain capsules every patient can help himself in angina pectoris and perhaps even in the case of a beginning acute myocardial infarction, before the ambulance can be present.

--- Pidgeon et al. 1996 (67) report that in sheep intravenous ouabain after 3 weeks caused a reduction of angiotensin II levels, an effect which could also be seen in healthy human volunteers after a single i.v. injection of ouabain (68). Studies in patients are still lacking. Perhaps ouabain could also act like an ACE-inhibitor.

--- Generally speaking, ouabain acts like a nitro-preparation, without two disadvantages that reduce the applicability of nitroglycerin preparations. In the case of acute hypotension with ouabain there is no danger of a further fall of blood pressure, and there is no addiction to the drug - the thousandth capsule is effective like the first ones.

The very good results of the randomized, placebo-controlled double-blind study - Salz & Schneider 1985 (8) in seven doctor's surgeries and the double-blind studies of Kubicek & Reisner 1973 (69) and Sharma (70) are already presented above.

Belz et al 1984 (71) made a placebo-controlled double-blind crossover-study, which shows that linguallly administered ouabain (12 mg) has a constant and significant (in part highly significant) effect on the heart contractility of healthy volunteers that is different from the effect of ouabain i.v. and similar to that of nitroglycerine, that is a negative inotropic effect. Dohrmann & Schlieff-Pflug 1986 (72) repeated the above mentioned study with patients which had severe coronary heart disease and instable angina pectoris. Also in these patients linguallly administered ouabain had the same effect like Nitrolingual®, that is in 2/3 of the patients a positive inotropic effect and in 1/3 of the patients a negative inotropic effect. Perhaps the different effect of ouabain on heart contractility is due to a different intracellular calcium content, because a calcium overload has a negative inotropic effect (73) and in this case the reduced intracellular calcium content by ouabain could have a positive inotropic effect (73). The latter was also seen in the studies of Piscitello u. Maggi 1973 in patients after orally administered ouabain (74) and Su et al. 2003 in isolated heart preparations (21, with strophanthidin). There are still open questions, because Horackova & Mullen 1988 in myocytes (25) saw a reduction of intracellular calcium with low doses of ouabain, an augmentation with high doses and no change with medial concentrations, but in all cases a positive inotropic effect. In any case, oral ouabain is effective in mild heart insufficiency. In severe cases it was reported (75) that oral ouabain is effective after a transient treatment with intravenous ouabain, which perhaps should be repeated from time to time (several weeks to months). Intravenous k-strophanthin (and probably ouabain) in the therapy of severe heart failure is more potent than oral digoxin. This was shown by the double blind study of Agostoni et al. 1994 (35): Only with k-strophanthin the performance in bicycle

(the latter by 50 %). Qi et al. 2001 (42) report about an increment in cardiac output of 25 % with digoxin and of 41 % by k-strophanthin and a significant fall of diastolic hypertension only by k-strophanthin.

As we have seen, ouabain combines the actions of several medicaments without their side effects and is the optimal pharmacological solution in the case of angina pectoris and myocardial infarction.

What are the effects of low concentrations of ouabain in ischemia / reperfusion injury? I never have seen an animal study or an in vitro / ex vivo study in this direction at all. I think such an investigation could be a breakthrough !

The effect of ouabain in other diseases

There are many other diseases other than angina pectoris, myocardial infarction and heart failure, in which the activity of the sodium pump of the concerning tissues or of the red or white blood cells is lowered: diabetes, cancer, multiple sclerosis, Morbus Parkinson and Alzheimer, epilepsy, dementia, Huntington chorea, schizophrenia, obesity, anorexia nervosa, cataract, acratia, hypothyroidism and hyperthyroidism, inflammable intestinal diseases, arthritis, cystic fibrosis, McArdle's disease, pulmonary edema, allergies, toxications, sepsis. In this cases low doses of ouabain as a stimulator of the Na-K-ATPase speculatively could have a positive influence. There are almost no studies regarding the effect of ouabain in this diseases up to now. The diseases in which a therapeutic effect of ouabain in patients is documented are cerebral ischemia (76-77), asthma bronchiale (78) and endogenous depression (79).

Hsieh et al. 2003 (80) and other studies report about a rise in intracellular Na⁺ in patients (80-81) and animals (82-84) with sepsis which perhaps could be the cause of the disturbances of Na-K-ATPase activity - on the one hand a reduction in erythrocytes (81) and erythrocytes, skeletal muscle and liver (85) of patients and in the rat heart (82, 86) and on the other hand a stimulation (the most other references) have been reported. Tang et al. 1993 (87) report about time-dependent changes in rat heart, first a stimulation, then a decrease in Na-K-ATPase activity.

The effect of oral ouabain in patients with sepsis is an open question. Regarding the theoretical background sepsis could be a contraindication because of the stimulation of Na-K-ATPase by low doses of ouabain. Anyway, in several decades of oral ouabain therapy there has never been an announcement of an aggravating effect of sepsis by oral ouabain, but it could be possible that 1) perhaps there has never been an oral ouabain therapy in sepsis or 2) that perhaps a negative effect hasn't been attributed to a possible oral ouabain therapy. Nevertheless Levy et al. 2005 (88) report about a beneficial effect of ouabain released by microdialysis probes in the skeletal muscle of patients with sepsis, probably in such a high concentration able to inhibit the local Na-K-ATPase. Also other studies report of this mechanism in animals (89-90). A head of department in a german clinic told the author that he has given intravenous Ouabain (0,125 mg or even less) in all cases of acute sepsis from 1975 to 2000 and is sure that because of this medication none of his patients died. The dose rate is so low that one can suppose that it could act like oral ouabain. Maybe that in sepsis low concentrations of ouabain have different effects in skeletal muscles and heart, which cannot be excluded. In any case, regarding the latter there is a number of studies showing an enhanced utilization of lactate after low dosed ouabain - DeMots et al. 1978 (46) and some german studies from 1941 to 1972 (91-98), see also the review of Ardenne 1978 (11, in english). In the study of Matsumori et al 1997 (41) intraperitoneally applied ouabain caused the quadruplication of survival in rats with

studies attribute the Na-K-ATPase stimulation in sepsis to catecholamines (89, 99-102) which were reduced by low doses of ouabain (33-35). Further studies are necessary.

The false dogma of the bad absorption of oral ouabain

The opinion that ouabain is very poorly absorbed (0-4 %) when enteral administered is not valid, as shown in the table below (arranged according to the duration of the study).

Absorption of radioactively labelled Ouabain

Study species application time absorption

Greenberger et al. 1969 (103), intraduodenal, 30 min,

rat: ouabain: 17 % (+ 11 % in the mucosa), digoxin: 27 % with equal blood levels of both glycosides

guinea pig: ouabain: 19 %, digoxin: 15 %

Ohlmeier & Ruiz-Torres 1968 (104), rat, intraduodenal, 30 min, 28 %

Forth et al. 1969 (105), cat, intraduodenal, 1 h, 11 %

Forth et al. 1969 (106), intraduodenal, 1 h,

rat: ouabain: 24 % digoxin: 75 % digitoxin: 86 %,

guinea pig: ouabain: 48 %, digoxin: 20 % (!), digitoxin: 59 %

Marzo et al. 1974 (107), guinea pig, intraduodenal, 5 h, ouabain 36 %, k-Strophanthin 38 %

Leuschner & Winkler 2001 (108), guinea pig, oral, 6 h, absorption 45 %, systemic bioavailability 43-50 %

Garbe & Nowak 1968 (109), guinea pig, oral, 7 days, 67 %

There are additional examples reporting of a much higher absorption than the textbooks (110-112).

Citation Forth et al. 1969 (106) p. 207 (translated by the author): "The findings indicate that the polarity (lipid solubility) is not the only important quality of cardiac glycosides regarding their absorption."

Kitano et al. 1998 (113) refer of a high absorption of radioactively labelled and orally administered ouabain (only 0,03 mg / 70 kg daily) to rats, comparable with that of equimolar digoxin (blood levels after 14 days: ouabain 0,024 nano-Mol, digoxin 0,033 nano-Mol).

1952 Dr. Berthold Kern (114) administered ouabain to patients with artificial anus and analysed the feces with picric acid and KOH, a very sensitive colorimetric method: very little amounts of ouabain (0,02 mg) evoke a marked coloration in the control experiment with only 2 % result variation. In every case there was no ouabain detectable. This affirms the older findings that only 0,7 % (guinea pigs, Lendle 1938

administration of deadly doses of ouabain.

Lauterbach suggested that there is an active transport process for polar cardiac glycosides like ouabain through the intestinal cells (117-118). As a parallel there is an uptake process per endocytosis into the intracellular compartments of the myocardium (119).

After orally administered 3H-ouabain in humans there are high blood levels of ouabain (up to 8 nMol = 8×10^{-9} Mol, which is far more than claimed for the therapy of heart failure) in the studies of Erdle et al. 1979 (120) - see the diagram below - and Marchetti et al. 1972 (121), which astonishingly never have been cited, for example they are not mentioned in the "Handbook of Experimental Pharmacology" (122). Here only the third respective study of Lahrtz et al. 1968 (123) is mentioned, in which 1) too little of ouabain was given (0,04 mg; a normal therapeutic dose is 3-12 mg) and 2) indeed there was given too little of radioactivity, even below the detection limit, so that a positive result was impossible even if there had been an i.v. application. However, also the effects of orally administered ouabain on human haemodynamics support the finding of a high and linear, not uncertain absorption (Piscitello & Maggi 1973) (74). Interesting are also the double blind study of Belz et al. 1984 (71) and the study of Dohrmann & Schlieff-Pflug 1986 (72).

Investigations with the RIA-method show smaller amounts of ouabain-immunoreactivity in human blood after oral application (0,3 - 0,5 nMol = $2-5 \times 10^{-10}$ Mol with Strodival® and up to 1,4 nMol = $1,4 \times 10^{-9}$ Mol with Purostrophan®) (124-125), which were always ruled as a prove for the ineffectiveness of the oral ouabain therapy. But also these concentrations of ouabain are fully within the concentration range that shows a distinct stimulation of the sodium pump, a knowledge that just recently begins to become accepted. Strobach et al. 1986 (126) only investigated the excretion in the urine and found a very low absorption rate for ouabain (1,4 %). But they found an urinary excretion of intravenous ouabain of only 33 % - this is the half of the excretion found in other studies (for example 124-125). This casts doubt on the methods of this study.

Riehle et al. 1991 (with Prof. Bereiter-Hahn, the vice president of the university of Frankfurt on the Main / Germany (127) could show that even a concentration of 10-13 M (= 60 quadrillionth gram in one millilitre) of ouabain has a reproducible effect on cardiomyocytes regarding the oxygen metabolism, in some cases even a concentration of 10-15 Mol did so; both are concentrations that can be produced by diluting but cannot be measured any more by any method. Even after homoeopathic treatment (1 ml Strophactiv®, D4) there are ouabain blood levels expected much higher than 10 -13 Mol, i.e. $1,3 \times 10^{-10}$ Mol.

Only a hair raising inconsistency or a prearranged deception ?

The often published statement of the uncertain enteral absorption of ouabain has its only root in a single examination of a ouabain preparation called Purostrophan®, which was available in the 1970ies and showed indeed some fluctuation of the results concerning the blood and urine levels of ouabain. Beside the fact, that this fluctuation wasn't bigger than that of the commonly used Digitalis medicaments, the author discovered, that the real examination of oral Purostrophan® wasn't made in the oftenly cited study (124) of Prof. Greeff et al. 1974, the accepted authority at that time, but was made one year before in a dissertation of his institute (125). The peculiarity of this dissertation is, that the examination of Purostrophan® has been made with two different groups of patients: one group took ouabain before and the

expected result is written: The group who took the medicine before the breakfast had absorbed more ouabain than the other group. Already in the dissertation the different results were summarized to one diagram and then were repeated by Prof. Greeff in his study (124) and other articles without citation, so that the origin remained hidden, and also without naming the methodological anomaly. This is the origin of the intensively published statement of an uncertain absorption of ouabain, which was used for the adoption of the prescription requirement. This nonscientific results are published in the same study that reports of the low enteral absorption of ouabain and influenced the majority of the physicians not to prescribe ouabain to their patients. The also tested Strodival® (lingual absorption) showed only minimal deviations, with even minor fluctuation of blood levels than that after i.v.application.

The detection of ouabain as a new hormone and the birth of a new false dogma

Since 1991 ouabain is identified as an endogenous compound (128), produced by the adrenal glands and / or by the brain (hypothalamus). By the way, the possibility that ouabain is taken up with the food cannot be excluded - this would be another proof for the good absorption of ouabain. First described as an isomer of ouabain (129-130) which implicates the possibility of different physiological properties, it could be shown that this was an artefact (131-132). Meanwhile it was assured that the endogenous ouabain is completely identical to original plant ouabain (132-134). For all that recently there are new findings, together with reanalysis of the before mentioned studies (132-133) that used ¹H-NMR (nuclear magnetic resonance spectrometry), which suggest that perhaps endogenous ouabain could be yet an isomer (with 11β-hydroxylation), which is very hard to observe in HNMR (135).

Because ouabain antibodies are crossreacting with other substances (for example 136-145), the results with an immuno-assay should be always verified by a chromatographic method (HPLC). Without HPLC it should not be spoken of ouabain but of OLS (ouabain-like substances) or similar terms. Unfortunately not all working groups pay attention to this important point. In human plasma 9 - 190 pM ouabain was found. Some other endogenous cardiac glycosides were identified: digoxin (146-147), dihydro-ouabain (148) proscillaridin (133), 19-norbufalin (149), marinobufagenin (150), telocinobufagenin (151) and other, up to now unidentified compounds (for example 152-153). The ouabain specific binding protein described by the workgroup of Giessen / Germany (154-155) turned out to be an artefact (personal communication).

Unfortunately one can observe the birth of a new false tenet: the role of endogenous ouabain as the cause of hypertension, which is based only on contradictory 1) studies with rats, 2) in vitro studies and 3) statistical findings. The extensive clinical observations with ouabain (g-strophanthin) and k-strophanthin which all report about a hypertension-lowering property of these substances (see above) is not known by the international scientists because most of the studies were published only in german language except Agostoni et al. 1994 (35) and Qi et al. 2001 (42). Even Prof. Schoner (Giessen / Germany) didn't know these facts.

1) The most in vitro studies are using unphysiologically high ouabain concentrations, which are likely causing an inhibition of the sodium pump. Only very few are dealing with physiological low doses of ouabain which stimulate the Na-K-ATPase. For example Saunders & Scheiner-Bobis 2004 (19) noticed a production of (blood vessel contracting) endothelin in human artery endothelial cells by 10⁻⁹ to 10⁻⁸ M ouabain, but Woolfson & Poston 1991 (156) report on the one hand of a reduced response of human resistance arteries to acetylcholine and on the other hand of an increased response to (blood vessel relaxing) NO (nitric oxide) by 10⁻¹⁰ M ouabain.

bradykinin stimulus, i.e. an enhancement of NO production. Woolfson et al. 1991 (157) report that the sequence of pharmacological actions is also important: The response of human resistance arteries to noradrenaline is increased by ouabain (in high concentrations) only when noradrenaline is added before ouabain. When ouabain is added before noradrenaline, the response to noradrenaline conversely is diminished. Such a detail has never been considered in any other study. Low doses of ouabain didn't alter the response of human arteries to noradrenaline.

The whole net effect seems to be concentration dependent: a good example for the effect of low doses of ouabain on vascular smooth muscle is the study of Branco and Osswald 1986 (36): the authors report of the different actions of three different concentrations of ouabain on dog blood vessels. The two high concentrations caused a constriction (release of noradrenaline), but the low concentration had the contrary effect. Also the studies of DeMots et al. 1978 (46) and Nelissen-Vrancken et al. 1997 (158) show the oppositional effects of high respectively low doses of ouabain..

2) The hypothesis of ouabain as a cause of essential hypertension is based mainly on quite a lot of observations in rodents (mostly rats, 1-2 times mice, the references are widespread in every study and review and here not listed). Exclusively in this species in vivo results supporting the hypothesis are available. It is an old pharmacological knowledge that rodents behave different regarding cardiac glycosides in comparison to other species and humans. So the experiences with rodents are of doubtful value, especially when some studies with very similar protocol (for example over a period of several weeks) do not show a hypertensinogenic action of ouabain (159-161), which is not often cited. Li et al. 1995 (160) are using the same test conditions as Manunta et al. 1994 (162) but don't notice any hypertension in Sprague-Dawley rats over 4 weeks with subcutaneous ouabain infusion (0,7 mg / 70 kg). Tamura et al. 2000 (163) report that a synthetic diet completely without cardiac glycosides causes hypertension in rats, which is prevented by orally administered ouabain in very low dosage (10 microgram / L).

One of the studies (164), in which ouabain caused hypertension in rats, contains a surprising hint for the beneficial therapeutic effects of ouabain. The rats given ouabain showed no cardiac hypertrophy like the rats in the control group. Citation Yuan et al. 1993 (164) p.186: "Ouabain actually may be cardioprotective." There are older studies who also report about the prevention of hypertrophy of heart (165) and adrenals (75). Moskopf & Dietz 1955 (75) report that guinea pigs with orally administered ouabain triplicated their capacity of swimming until exhaustion (the smallest dosis had the biggest effect - 31,5 mg, 63 mg, 94,5 mg / 70 kg). There was no adrenal hypertrophy as noticed in the control group. Similar results with rats are reported by Kuschinsky 1947 (165).

J.M. Hamlyn from one of the leadig workgroups said (166, unpublished data) that rats who had developed hypertension after ouabain had better pelt and were more healthy and agile than the animals without ouabain. Perhaps the hypertension in these rats is not a disadvantage as suggested. A study with these rats concerning the status of health and length of life were interesting.

The studies with other animals (dog, rabbit, sheep) are reporting of an unchanged or even lowered blood pressure after a single dose of ouabain. The sheep used in the study of Pidgeon et al. 1996 (67, double-blind, crossover)) showed a reduction of mean blood pressure, of renin and angiotensin II levels and of sodium excretion after three weeks of 0,25 mg i.v. ouabain daily.

3) Some studies report of statistical correlations between hypertension and ouabain (better: OLS, because all without HPLC) blood levels in patients (for example

correlations do not prove any causal relations (remembering the simultaneous decline of the number of storks and the number of births). Otherwise a high ouabain / OLS secretion in hypertension could be interpreted as a counteraction against hypertension. Anyway, the biggest recent studies - with J.M. Hamlyn - reveal that normotensive (170) as well as hypertensive persons (171) with a mutation of the adducin gene have higher blood pressure and lower OLS blood levels than those with the unmutated gene, who have lower blood pressure and higher OLS blood levels.

The findings of the study of Gottlieb et al. 1992 (167) are a good argument against overhasty causal interpretations: The patients with heart insufficiency had higher OLS blood levels than healthy controls. Could therefore ouabain be seen as the cause of heart insufficiency? The differentiated findings veto this superficial interpretation: Higher OLS levels are found in patients with NYHA I-III, while patients with severe heart insufficiency (NYHA IV) had lower OLS levels. Thus, the higher OLS levels in NYHA I-III could be a physiologic reaction to the disease, while the lower OLS levels in NYHA IV could reflect an exhaustion of the production capacity of the adrenals and the hypothalamus. The fact that ouabain is used in Germany for many decades as a remedy in heart insufficiency pleads for the latter interpretation.

There is considerable clinical evidence that orally administered ouabain attenuates hypertension in patients (8, 9, 35, 42-44) and healthy volunteers (45, 47). On the other hand there are reports by some patients in Germany that oral ouabain is also enhancing hypotension. It is wellknown by the "ouabain doctors" that in acute heart attack oral ouabain can be given also in cases with very low blood pressure, in which a nitro preparation is dangerous because the possibility of a hypotonic crisis. Perhaps ouabain could be regarded as a blood pressure controlling hormone in a physiologically positive manner.

Ouabain was primarily postulated as a natriuretic hormone acting by inhibiting the Na-K-ATPase (sodium pump) in the kidneys and - as a side effect - also in the blood vessels creating hypertension. Meanwhile the natriuretic action of ouabain / OLS could be disproved; in contrast according to Manunta et al. 2001 (172, one of the leading workgroups, with J.M.Hamlyn) there is no difference in OLS blood levels between salt-sensitive and salt-resistant hypertensive patients and ouabain blood levels are elevated in hypertensive patients only in the case of sodium depletion (172). Even the creator of the original hypothesis in 1961 himself, H.D.DeWardener, proclaims already 1997, that ouabain is not a natriuretic hormone (173). Other substances are better candidates (174-175). Also Marinobufagenin has all properties to fulfill the criteria of a natriuretic and blood pressure enhancing hormone (176).

D'Urso et al 2004 (177) report that the rat heart is producing ouabain (verified with HPLC) in ischemia. De Angelis & Hauptert 1998 (178) report that rats breathing air with reduced oxygen content had elevated ouabain blood levels.(confirmed by HPLC). This studies remember of the therapeutic effects of orally administered ouabain in angina pectoris and myocardial infarction. Probably the hormone ouabain has a positive physiological function in the heart - and perhaps in other tissues.

The solution of the problem is not wanted

Unfortunately there has been a systematic opposition against the excellent therapy with oral ouabain in angina pectoris and myocardial infarction, a German "speciality", so that the numerous published convincing proves were not proclaimed adequately. From 1905 until the 1950ies ouabain i.v. was the german official therapy in heart insufficiency and also partially in angina pectoris and myocardial infarction, which were rare diseases at that time. The exasperated controversy reaches back to ca.

see results which are unparalleled by any other, even modern medication. The one and only repeated and ruminated "argument" against ouabain was the pretended bad oral absorption of ouabain. Especially in the 1970ies and 1980ies an International Society for Infarct Combat, renamed as International Society for Infarct Prevention (179), (= "Internationale Gesellschaft für Infarktbe-kämpfung" and "...Infarktverhütung" respectively), which was founded by Dr.med. Berthold Kern, the developer of the oral ouabain therapy, with many doctors and some supporting professors, strived for acknowledgement of the respective facts, but the "suppression fraction" of the medical establishment was too strong, In that time Prof. Gotthard Schettler was the main opposer. In part it seemed to be a private war of Prof. Schettler against Dr. Kern. They knew each other from the 1950ies in Stuttgart, where the battlesome Dr. Kern had a big practice and Prof. Schettler were the leader of a hospital and counteracted the therapy of Dr. Kern.

In 1971 Prof. Schettler, who was at that time leader of the German Society for Internal Medicine, invited Dr. Kern to a symposium in Heidelberg to discuss the ouabain therapy. Dr. Kern hopefully went to Heidelberg with six other supporting persons and expected the same number of discussing partners. But he was very surprised when there were 160 professors present and a great number of journalists of every important journal of the medical and the "yellow" press. The agreement that both sides should alternate the presidency of the meeting was ignored: Prof. Wollheim, also a sharp opponent of Dr. Kern, governed alone and blocked the arguments pro ouabain and expanded all, even marginal aspects against Dr. Kern. Dr. Kern who was addressed as "accused Kern", for the most part spoke alone, but the other side was alternating with prepared speech and dia shows. A main part of the discussion did not concern the ouabain therapy but the pathogenesis of myocardial infarction (see above). Dr. Kern had substantial criticism in this point of view. The old positions of the "coronary theory" were really quite contradictory and open to attack - the vulnerable plaques weren't explored in that days. But the medical establishment insisted on its positions, and at the end of this event that lasted seven hours, the exhausted Dr. Kern was shouted down collectively. Prof. Rilling (Stuttgart / Tübingen), one of the supporting team of Dr. Kern, told the author, that Prof. Schettler who knew Prof. Rilling from older student days, said to him: "What do you do here ? Be careful - this will not have a good end. Dr. Kern is just condemned !" In the whole press Dr. Kern was denoted as a medical quack and after this execution which the insiders know as "Tribunal of Heidelberg", only very few doctors and professors dared to support the ouabain therapy in public.

The scepticism of many doctors in testing a substance which is classified as a cardiac glycoside, in angina pectoris, especially when in all textbooks is written that there is 0 % or 1-2 % absorption, is huge. For example there is a doctor, who has seen the excellent success in every of his 150 Angina pectoris-patients, but his colleagues are laughing and even don't want to hear his story to the end. (I hope you are still reading...) There are doctors who have Strodival® in their drawer, and in case of emergency (severe angina attack, suspected AMI) they quickly applicate the capsule, but they don't inform the hospital, what they have done, because they fear to make themselves ridiculous. A real tragical story... The author of this script had also written a scientific book about this theme (see below) with the preface of Prof. Hans Schaefer from Heidelberg, a worldfamous physiologist. Unthinkable that 20 german medical journals don't want to publish an article, even not a recension, after a telephone call to a responsible editor and mailing the book and texts - only 2 had given notice, the others shows no reaction at all, although

It is an urgent challenge to save the real possibility to solve one of the biggest medical problems (really: Oubain could be in Coronary Heart Disease something like insulin is in Diabetes.) and to preserve a blessed medicine that helps so many

there is no prolongation of the licence for Strodival®, the last orally administered ouabain preparation, because the big and cost-intensive clinical double-blind study is missing. Even a big multinational company would not pay a study for a substance without a protection by patent. The orally ouabain therapy may die in 2005, although the existing studies and reports are overwhelmingly convincing. The producer counts on the imminent end of ouabain production to 99,9 percent. A real tragedy...

A recent event (December 2005): For the first time in history the ministry of health as the competent supervisory authority issued an instruction to the federal office that is responsible for the license of medicinal products, namely that the license of Strodival(R) has to be prolonged because of an extraordinary public interest regarding the immense potential of this substance. In november 2005 the author participated at an intern meeting in Bonn between the two parties (ministry: 5 persons, federal office: 3 persons), additionally with two physicians who reported about their experiences with two hundred respectively several thousands of patients. Also 40 letters of patients were presented who reported their great fear about the time when Strodival no longer is available. Nonetheless the unimpressible federal office was furthermore against Strodival(R) and protested against the instruction. There is a close relationship of the federal office with the pharmaceutical industry who is financing the office for the most part.

In 1996 the old producer "Herbert Pharma" in Wiesbaden/ Germany was adopted by "BRAHMS Arzneimittel GmbH" in Wiesbaden (the new name was "Herbert Arzneimittel GmbH"). In 2003 "Herbert" was sold by "BRAHMS" to "MEDA", a Swedish combine. By the way, the director of the mother company BRAHMS AG, Dr. Bernd Wegener, is the first chairman of the organisation of pharmaceutic manufacturers in Germany ! ("Bund Deutscher Pharmazeutischer Industrie") While the old, small producer was very busy in fighting for the ouabain therapy, the new producer is quite inactive. For example information about ouabain (g-strophanthin) was never found on the homepage of "BRAHMS" or "MEDA" (...)

Without doubt ouabain (g-strophanthin) could be the solution of the problem cardiac infarction to a large extent and especially the therapy with orally administered ouabain and the knowledge about this drug has to be brought forward because of medical, ethical and economic aspects.

Rolf-Jürgen Petry - 8. Aug, 18:14