

Lipoedema – myths and facts Part 3

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Keywords

Lipedema, lipoedema, obesity, weight loss, scientific evidence

Summary

Lipedema is associated with numerous myths. In this short series we offer an overview of the myths of lipedema, and we throw a critical eye over popular statements concerning the disease; statements that found their way into scientific publications decades ago, and which have been accepted and repeated since then without criticism; statements which have become widely accepted facts for lipedema patients, and particularly for lipedema self-help groups. In the first part of this series we took a critical look at two popular myths about lipedema. We found that there is no scientific evidence for neither "Lipedema is a progressive disease", nor "Lipedema causes mental illnesses". In the second article about the myths of lipedema we focussed on the edema aspect, on the "edema in lipedema" and the subsequent therapeutic consequences – manual lymph drainage. We were also able to show that there is no scientific evidence for the popular statement: "lipedema is primarily an "edema problem"; manual lymph drainage is thus the essential standard therapy to be conducted regularly". There is thus no basis for the prescription of long term regular manual lymph drainage with the aim of removing edema. In this, the third part of the series on familiar and often quoted "lipedema statements" we take a closer look at two other myths: 4: "lipedema makes you fat" and 5: "weight loss

has no effect on lipedema". For both statements there is neither a reasonable physiological or pathophysiological construct nor is there any scientific evidence in the literature. Furthermore both statements contradict our many years of daily clinical experience with lipedema patients to a high degree. It actually seems that the converse is true: Weight gain seems to be a decisive trigger for the development of lipedema, in patients with the right genetic disposition. Lipedema and obesity are two different diseases, but mostly they appear together. We see patients on almost a daily basis, who effectively lose weight after successful bariatric surgery as part of our obesity program, including in the limbs. Lipedema patients regularly experience considerable improvement in the pain symptoms typical for lipedema through sustained weight loss. These patients are often symptom-free, so that we can then say that the lipedema is in remission. In our fourth contribution to the series we will examine the significance of liposuction for lipedema, in order to then present a therapeutic concept in the last part of the series, that is not only based on scientific evidence, but should also contribute to a long-term and comprehensive improvement in the symptoms of lipedema patients.

Schlüsselwörter

Lipödem, Adipositas, Gewichtsabnahme, Wissenschaftliche Evidenz

Zusammenfassung

Um das Lipödem ranken sich zahlreiche Mythen! In dieser kleinen Übersichtsreihe über die Mythen des Lipödems werfen wir einen kritischen Blick auf populäre Statements zum Lipödem; Statements, die vor Jahrzehnten schon Eingang in wissenschaftliche Publikationen gefunden haben und seither unkritisch und stetig wiederholt werden; Statements, die dadurch inzwischen zum selbstverständlichen Wissensallgemeingut von Lipödempatientinnen und vor allem auch von Lipödem-Selbsthilfegruppen geworden sind. Im ersten Teil unserer Darstellung haben wir uns kritisch mit zwei populären Mythen über das Lipödem auseinandergesetzt. Hierbei haben wir festgestellt, dass sowohl für das Statement „Das Lipödem ist eine progrediente Erkrankung“ als auch für das Statement „Ein Lipödem macht psychisch krank“ keine wissenschaftliche Evidenz vorliegt. In einem zweiten Beitrag über die Mythen des Lipödems fokussierten wir uns auf den Ödemaspekt, auf das „Ödem im Lipödem“ und die hieraus erfolgende therapeutische Konsequenz: die Manuelle Lymphdrainage. Wir konnten darlegen, dass für das populäre Statement „Das Lipödem ist in erster Linie ein „Ödem-Problem“, daher ist die Manuelle Lymphdrainage essenzielle und regelmäßig durchzuführende Standardtherapie“ ebenfalls keine wissenschaftliche Evidenz existiert. Der regelmäßigen und dauerhaften Verordnung von Manuellen Lymphdrainagen mit dem Ziel der „Ödembeseitigung“ fehlt daher jede Grundlage. In diesem dritten Teil der Auseinandersetzung über bekannte und oft zitierte Lipödem-Statements beschäftigen wir uns mit zwei weiteren Mythen: 4. „Das Lipödem macht dick“ und 5. „Gewicht abnehmen hat keinen Effekt auf das Lipödem“. Für beide Statements gibt es weder ein sinnvolles physiologisches bzw. pathophysiologisches Konstrukt noch eine sich in der Literatur findende wissenschaftliche Evidenz. Darüber hinaus widersprechen beide Statements in

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hohem Maße unserer seit Jahren bestehenden täglichen klinischen Erfahrung mit Lipödempatientinnen. Tatsächlich scheint das Gegenteil richtig: Gewichtszunahme wirkt als entscheidender Trigger, um – bei entsprechender genetischer Disposition für ein Lipödem – dieses überhaupt erst zu entwickeln. Lipödem und Adipositas sind zwei unterschiedliche Erkrankungen, die jedoch in den meisten Fällen gemeinsam auftreten. Fast

täglich sehen wir Lipödempatientinnen, die sich aufgrund ihrer morbid Adipositas einer bariatrischen Operation unterzogen und dadurch effektiv – auch im Bereich der Extremitäten – Gewicht verloren haben. Patientinnen mit Lipödem erfahren durch diese meist nachhaltige Gewichtsabnahme regelhaft eine deutliche Besserung ihrer lipödemytischen Schmerzen. Häufig sind die Patienten dann beschwerdefrei, sodass wir dann von einem Lip-

ödem in Remission sprechen können. In einem vierten Beitrag werden wir uns mit dem Stellenwert der Liposuktion beim Lipödem beschäftigen, um dann in unserer letzten Darstellung ein therapeutisches Konzept vorzustellen, das nicht nur wissenschaftlich fundiert ist, sondern auch zu einer nachhaltigeren und umfassenderen Beschwerdeverbesserung unserer Lipödempatientinnen beitragen soll.

Introduction

This is already the third part of a presentation that casts a critical eye over popular statements about lipoedema, myths that have surrounded the disease for decades.

In the first part of our series on lipoedema, we showed that there is no scientific evidence for the widely disseminated assertion that “lipoedema is a progressive disease” and that the frequently misused term “lipolymphoedema” is therefore obsolete (1). It is not the disease of lipoedema that is progressive, but often the disease of obesity. If both lipoedema and also lymphoedema are present in a severely obese female patient, this is, therefore, not “lipolymphoedema” but obesity related lymphoedema (2).

In addition, our pilot study showed that the statement “Lipoedema causes mental illnesses” is also a myth with no scientific basis whatsoever. Instead, it appears that a high psychological vulnerability contributes significantly to the development of lipoedema – not least due to the increased perception of pain (3).

In the second part of our review, we devoted our attention to the statement “Lipoedema is primarily an “oedema problem”; regular manual lymphatic drainage is therefore essential”. We showed that neither clinical, imaging nor histological evidence exists for “oedema in lipoedema”. Hence, there is no indication for long-term and regular manual lymphatic drainage (4).

This third part of our presentation on the myths of lipoedema concerns the sensitive topic of “lipoedema and obesity”. There are two particular statements that accompany our daily clinical work with lipoedema patients. First of all, the statement particularly popular with patients that “Li-

poedema makes you fat!” and secondly, the message spread by many lipoedema experts “Weight loss has no effect on lipoedema!”

Right at the beginning of our remarks, we have to point out that there is no scientific data to support these two myths. However, there are also none for the contrary thesis, i.e. for the statement that lipoedema does not lead to weight gain and that weight loss certainly does lead to an improvement in symptoms!

Nevertheless, it appears obvious that there is a close correlation between being overweight or obese and the disease of lipoedema. We saw 2344 women diagnosed with lipoedema in our SHI-accredited outpatient lymphology clinic in 2015. Only 3% of these patients were of normal weight, 9% were overweight (BMI between 25 and 30 kg/m²) and 88% of our lipoedema patients were obese (BMI > 30 kg/m²) (5). Our patient collective appears largely representative in this regard. For example, Bosman reported that up to 80% of his female lipoedema patients in a centre in the Netherlands were overweight and/or obese (not separated into overweight and obesity) (6). In 2011, the British research group of Child and Gordon reported that only 4% of their lipoedema patients were of normal weight, 11% were overweight and 85% obese (7). These figures clearly demonstrate the strong relationship between lipoedema and obesity. A lipoedema patient of normal weight is a rarity!

It must be borne in mind in this context that the BMI is of only limited value in lipoedema patients in the overweight region (between 25 and 30 kg/m²). There is the rare group of patients with a largely slim upper body and marked increase in fat in the extremities. Overweight in the strict

sense is illusory in such patients because it is not actually present due to the fat distribution through the lipohypertrophy of the legs. The WHtR (Waist-to-Height Ratio) is the more suitable measure for these patients. It describes the ratio between waist circumference and height and gives a better indication of the distribution of body fat. In the Földi Clinic, we measure both the BMI and the WtHR in all patients with lipoedema.

So how can this apparent relationship between lipoedema and obesity be explained?

In the absence of scientific data, we want to examine the plausibility and empirical evidence for the two popular statements.

Myth 4: Lipoedema makes you fat!

Patients as well as the media are keen to explain the connection between lipoedema and obesity as being that lipoedema is responsible for the weight gain in women with lipoedema.

For example the magazine Stern has written, “Women who suffer from lipoedema are generally incorrectly considered to be simply overweight”... and... “many women affected (and a few men) have been slim all their lives before the fat pads on their arms and legs suddenly increase uncontrollably” (8).

Sputnik, the youth-oriented station of Central German Radio, describes lipoedema on its website as a “mysterious disease that makes people fat” (9).

Evidero, a German internet health portal, lists lipoedema as the first disease that can lead to becoming overweight, even be-

fore hypothyroidism. On its website it states that “lipoedema has nothing to do with eating” (10).

During the preparation of this article, the German NDR television channel broadcast an item on lipoedema in its programme “Market”. A severely obese woman laboriously climbing stairs was shown right at the start of the clip. At the same time, the off-stage voice was heard saying “Climbing stairs – a painful torture for R. W. She suffers from a rare disease called lipoedema, a disorder of fat distribution that causes her to constantly put on weight” (11, here only the initials of the patient are given, not the name).

A severely obese lipoedema activist has now propagated the notion “Lipoedema in the belly” on YouTube (12).

This approach is even more widespread in the USA, where there is a coalition of doctors and women patients who explain their weight gain by the disease lipoedema. In a specialist article by Dr. Karen Herbst, endocrinologist and protagonist of the American lipoedema scene, a patient who weighs more than 200 kg writes:

“I have a congenital condition called lipoedema that causes my body to produce excess adipose tissue, also known as fat” (13). A US internet portal for overweight people, states that “Lipoedema May Increase Obesity in Women” (14).

As a result of this view propagated in the media, we are confronted on a daily basis in the Specialist Lymphology Clinic or also in the SHI-accredited outpatient clinic, with women diagnosed with lipoedema who tell us things such as, “My legs are becoming fatter and fatter and hurt”. Mind you: the patient who made this statement weighs 90 kg or 140 kg, for example. But the focus of the patient was explicitly on her legs. Then this is often followed by the second statement, “Because of my lipoedema, I am constantly putting on weight”.

There is no scientific evidence for this view! There is also no conclusive pathophysiological construct to explain how lipoedema leads to weight gain, to weight gains of 20 kg, 50 kg or 70 kg!

There is general agreement that a disproportionate increase in fat in the legs (sometimes also in the arms) is an import-

ant criterion for this disease. This initially often only slight disproportionality is frequently seen even during puberty. The young women are generally asymptomatic at this time; at most there is mild lipohypertrophy of the legs that is of no pathological significance whatever. The risk of symptoms and hence of the development of lipoedema only starts when the weight progresses and the associated over-proportional increase in fat in the lower half of the body occurs.

There is therefore much to be said for the opposite view that it is not the lipoedema that leads to weight gain, but that weight gain (often obesity) can, in the case of the relevant (genetic) disposition, result in lipoedema.

The causes of obesity are varied and highly complex. There is scientific evidence for genetic and especially epigenetic influences (15–18). Biological factors such as stress or addictive behaviour can affect weight (19–20). Psychological diseases such as depression and eating disorders can greatly contribute to obesity (21). Finally, sociocultural changes also play a considerable role in the development of obesity: for example, changes in eating habits, the exorbitant consumption of sugar (particularly in soft drinks and in industrially-prepared foods), and a lifestyle with little exercise. Added to these is a beauty ideal that is enshrined in the underweight range and is therefore closely connected with a diet culture which often begins in adolescence and whose long-term course does not lead to weight loss, but on the contrary, to weight increase (22–26).

In Germany, roughly 25% of the population are now obese (BMI > 30 kg/m²) and in the USA this figure had already reached 40% in 2017 (27–28).

This shows how obesity has become an epidemic in the past decades (29) and as a result – the diagnosis of lipoedema as well!

So if lipoedema doesn't make one fat, how can the broad support for this scientifically untenable position be explained?

The essential basis for the popularity of this view of lipoedema is the stigmatisation of the disease of obesity, which is frequently considered to be the result of incorrect behaviour, a weak will or lack of discipline. Even among medical professionals,

and particularly among doctors, weight-related discrimination and stigmatisation is especially marked (30–32). Even medical students find disparaging jokes about obese people not inappropriate because of the opinion that they are themselves to blame for being overweight. But stigmatising attitudes towards obese patients can also be found among medical specialists in obesity and dieticians (33–34).

The referral letters we receive regarding our patients regularly contain the medical recommendation to reduce weight. As mentioned above, the causes of the disease of obesity are complex – as also recognised by the WHO. “Guilt” (or “the accusation of incorrect behaviour”) is therefore not only a less than helpful explanation for obesity, but is simply wrong!

At the same time, the data concerning the poor prognosis of conservative weight reduction are impressive. Depending on the study, between 80% and 99% of all patients who lose weight conservatively will put it on again in the long term (35–42). In women in particular, attempts to reduce weight that were started during adolescence lead to an often decade-long dieting spiral with a continuous increase in weight (43). The routinely stated medical recommendation to the overweight woman to lose weight is thus highly problematic!

The advice to reduce weight is especially deceitful in lipoedema. Studies by Hohenheim University have shown that women (in contrast to men) regain their weight after weight loss over-proportionately in the lower half of the body (44) – and, as stated above – almost all diet participants put their lost weight back on. In other words, any recommendation by a doctor to female lipoedema patients to lose weight actually increases the risk of a further increase in fatty tissue in the leg region and hence also increases the risk of their symptoms increasing.

In our opinion, the “boom” in lipoedema is also due to the way the unloved disease of obesity is handled by medical professions as outlined above. On the part of patients, lipoedema has thus developed into an “excuse”, into a kind of “alibi” for weight gain and for being overweight. If obesity is judged as culpable behaviour, then – according to the view popular

among patients – the disease of lipoedema is the explanation for the constantly increasing weight. “I am not fat, I suffer from lipoedema” is the credo of many lipoedema self-help groups, that then also finds resonance in the media (45–47).

On a daily basis we are confronted in the Specialist Lymphology Clinic by women with lipoedema who are convinced, based on this false information (published in a variety of channels) that the lipoedema is responsible for their long history of progressive weight gain. It is laborious and time-consuming to convince patients that they are misinformed. It is laborious and time-consuming to convince patients that there are other factors that lead to an increase in weight – but definitely not lipoedema. It is laborious and time-consuming to convince patients that the frequently desired treatment options – manual lymphatic drainage or liposuction – will in no way result in a substantial, permanent or even only approximately satisfactory weight loss.

Conclusions

There is no evidence that lipoedema leads to weight gain. Our clinical experience, repeated numerous times a day, points in the opposite direction. Given the right genetic disposition for lipoedema, weight gain appears a decisive trigger for developing it in the first place. This is confirmed by the data from our internal obesity programme, in which currently more than 400 patients are being extensively diagnosed and documented (48).

Normal or slightly overweight women with highly disproportional fat distribution and soft tissue symptoms do exist, but in view of the overwhelming majority of obese and morbidly obese women with lipoedema, they form a very small minority.

Myth 5: Losing weight has no effect on lipoedema!

This statement represents a further dogma within lipoedema mythology. With almost complete regularity, it is lamented that medical professionals confuse lipoedema

with obesity. For example Schmeller wrote that “often lipoedema is confused with obesity or is regarded as a partial symptom of obesity”. (49); or in another publication: “even doctors and medical professionals frequently overlook the disease or confuse it with obesity or lymphoedema” (50). Cornely and Gensior were similarly worried, whose website states that “Lipoedema is rarely diagnosed quickly and correctly. Often patients have to put up with hearing: You are too fat” (51).

Karen Herbst, endocrinologist and protagonist in the American lipoedema scene entitled her article that mainly discussed lipoedema, “Rare adipose disorders (RADs) masquerading as obesity”. In it she wrote that “One of the most common misconceptions about patients with lipoedema is that they suffer, instead, from lifestyle- or diet-induced obesity”; and at another point: Lipoedema “may be misdiagnosed as obesity” (52).

This view of lipoedema is taken up with alacrity by the media and by patients. The magazine Stern said that “Women who suffer from lipoedema are usually falsely accused of simply being overweight and an actress writing about lipoedema (and whose book has risen to become a best seller in the lipoedema scene in Germany) believes that many women with lipoedema are regarded in medical practices as “just being obese” (53–54). It goes without saying that this popular view is shared by lipoedema self-help groups and internet fora (55).

It cannot be emphasised enough: the great majority of women with lipoedema are actually ALSO obese!

To repeat the figures already given above just one more time: a woman of normal weight with lipoedema is actually rarity! In the 2015 patient collective seen at our outpatient clinic (more than 2300 female patients), only 3% of those with lipoedema were of normal weight; similar figures have been reported from England and the Netherlands. In other words, by far the greater number of these women suffer from two problems – from obesity AND lipoedema. Focussing on the lipoedema in a woman with a BMI of 35 or 45 or 55 kg/m² appears to us grotesque and does little to achieve the objective; the division of the

two problems is not helpful. In our experience, many of our female patients with lipoedema often suffer more from the progressive weight increase (and hence the associated “currently unfashionable body shape”) than from the actual pain in the legs.

As a consequence of this view propagated by many lipoedema experts – and therefore also by lipoedema patients – there is the assumption that weight loss also has no influence on the lipoedema (and/or the symptoms that occur with the lipoedema). It would merely reduce the “obesity fat” but not the “lipoedema fat”. The above-mentioned endocrinologist, Herbst has written that “Although lifestyle changes and bariatric surgery work effectively for the obesity component” ... “these treatments do not routinely reduce the abnormal subcutaneous adipose tissue” of lipoedema (56).

Schmeller has also stated that weight loss helps “but only in the case of obesity and it merely reduces truncal circumference. Since the lipoedema-specific fat accumulations cannot be lost by going on a starvation diet, the discrepancy between trunk and extremities gets even worse” (57). Under the heading “The facts are therefore”, Cornely declared that “An improvement through dieting and sport is not possible” (58). A doctor called Stutz who, like Schmeller and Cornely, performs liposuction is convinced that the fat pads on the legs are not the same as stored reserve fat in obesity. In his patient flyer Stutz declares that “This fat has a quite different structure and therefore cannot be lost through dieting and sport” (59).

It is conspicuous – and this remark is permissible – how strongly especially doctors who practice liposuction in Germany commit themselves to this approach. For example, the websites of other liposuction colleagues contain the information that sport and weight loss have no positive effects on lipoedema (e.g. 60–62).

This view is also gratefully adopted by patients and lipoedema self-help groups. The lipoedema portal frequently visited by women with lipoedema considers unsuccessful weight reduction to be the “key symptom” of lipoedema: “The amount of fatty tissue in lipoedema cannot be reduced

either by dieting or by sport (diet-resistance). Even extreme weight loss diets that make the upper body and face emaciated and bony, do not reduce the lipoedema (key symptom)” (63, emboldened in the original).

Leaving aside the fact that neither the authors nor (on asking them) the senior physicians and the medical director of the Specialised Lymphology Clinic have set eyes on lipoedema patients in the past decade looking “emaciated and bony” and there is just one single photo of someone on the Internet that is shown time and time again (96), the question remains as to the scientific evidence of this viewpoint that is so popular with experts and patients.

What is the basic pathophysiological construct for the assumption that weight reduction in the obese, female lipoedema patient does not also lead to substantial loss of fat in the extremities?

It is clear that the above-named authors also provide no explanatory model in their publications that justifies their published statements to any extent at all. In that respect all that remains are myths, myths that for years have been passed on and adopted by women with lipoedema. The reasons why even the doctors practicing liposuction have a considerable part in the spreading of these myths is a matter for speculation.

These myths contradict the historical publications of Allen and Hines, the first people to describe lipoedema in 1940 and 1951, who wrote that “In cases of generalized obesity, sharp reduction of weight may help” (64–65).

This description of the first authors also corresponds to the clinical experience with lipoedema patients that we and our colleagues in the Specialised Lymphology Clinic have amassed over many years.

Again and again, we see women with lipoedema, who – at least for a short time – have succeeded in losing weight through conservative therapy and thereby also achieved – at least temporarily – a reduction in fat in the region of the extremities (each patient at the Földi Clinic undergoes detailed measurements of the arms and/or legs in addition to measurements of height, weight, BMI, abdominal circumference and waist-to-height index).

More permanent and more substantial are weight losses that women have experienced as part of our multimodal obesity programme, in which lymphoedema and also lipoedema patients with a BMI upwards of 40 kg/m² are prepared – provided they are suitable and have consented – for surgical obesity treatment. The obesity programme of the Földi Clinic has already been outlined in an earlier issue of this journal (66). The weight reduction achieved with this programme is also regularly reflected in a generally proportional reduction in circumference of the extremities.

In other words, the weight loss achieved within the framework of bariatric surgery (gastric bypass or sleeve gastrectomy) by women with lipoedema is also experienced substantially and generally permanently in the region of their arms and legs. A certain disproportionality of the legs usually remains after successful weight loss. However, the sole asymptomatic disproportionality of the legs is generally not a pathological condition. The aim of this medical treatment is not a leg that approaches the current beauty ideal – this would be the task of cosmetic surgery that, of course, would not be undertaken at the expense of society as a whole! The aim of this medical treatment is primarily to improve or abolish symptoms. The overwhelming majority of our patients experience a marked improvement in their symptoms as a result of the often considerable fat reduction in their legs, many even become pain-free. We then speak of “lipoedema in remission”. If the course of body weight remains largely stable also in subsequent years, it is unlikely that symptoms will recur. Thus the lipoedema becomes a – generally only still mild – lipohypertrophy of no pathological significance whatsoever. These positive experiences of weight loss on lipoedema, collected over many years, are currently the subject of a study with the University of Freiburg.

We advise obese patients with a BMI below 40 kg/m² or patients in whom (e.g. due to internal or psychological contraindications) surgery for obesity is not possible to undertake long-term weight stabilisation. At the same time, we explicitly advise against diets or conservative weight reduction programmes. We justify this ap-

proach by the disastrous long-term prognosis of attempts at conservative weight reduction as proved by the consistent data already mentioned above and confirmed by our long clinical experience with patients who have been under our lymphological care for years and whose weight has continuously increased during their “dieting careers” often over many years. Instead of “diet and exercise” the credo of our clinic is “stabilize and exercise”. In addition, alongside the compression therapy essential for all women with lipoedema, the promotion of self-acceptance – especially the acceptance of one’s own body – is of overriding importance for this group of patients. Only when this is achieved is it possible for them to escape from the vicious circle of diets, yo-yo effects and worsening of the lipoedema.

As well as the improvement in their lipoedema symptoms, women who have undergone bariatric surgery also experience other positive effects such as a marked increase in their mobility and hence also in their quality of life (67–68).

Added to that is the cardiovascular benefit of surgical treatment of obesity: diabetes mellitus, high blood pressure and sleep apnoea syndrome improve, or often disappear completely (69–73).

Finally, several large studies show the efficacy of bariatric surgery in terms of reduction in long-term mortality. Following sleeve gastrectomy or gastric bypass operations, patients live longer and healthier lives than comparably obese patients who do not have such procedures (74–77).

The frequent assertion by nutritionists that the metabolic outcome of conservative nutritional programmes is comparable to that of sleeve gastrectomy or gastric bypass, is, in our opinion, more wishful thinking than scientific evidence. For example, G. Faerber wrote in this journal, “These results are therefore comparable to those after bariatric procedures” (78). The author provides two studies as a basis for this statement, with an extremely low sample size (11 and 29 subjects respectively!) and also of extremely short duration (maximum 8 weeks!). During these eight weeks, the subjects were subjected to a 600 kcal diet (Optifast from Nestle) (79, 80). Various metabolic parameters were then

measured. The question as to how long these subjects continued this 600 kcal diet after the end of the study remains open and it is not known how the metabolic parameters subsequently developed.

The conclusions drawn from these few studies that have compared results from those with bariatric procedures – which, as mentioned above (69–71, 73–77), have involved several thousand patients over many years – are astonishing and ultimately only understandable if one shares the critical attitude of Faerber to surgical treatment options. The failure of conservative weight reduction programmes in the long term has been documented by many high-grade studies (81–91) and has already been described by Bertsch (2) in this journal. The author (Faerber) also expressly warns against “malnutrition or nutritional deficiency, osteoporosis, oesophagitis” and more. A wider training of nutritional specialists and dieticians in the art of post-operative care following bariatric surgery would, in our opinion, be a better response than the sweeping dissuasion from using a treatment option that gives very many patients the chance of a healthier, longer and easier life.

In addition, in her article Faerber warns against an increased rate of suicide after bariatric surgery. In the same article in this journal (78) she refers to the study by Tindle (92) in the USA. An increased suicide rate compared to the general population (!) is also described in a review by Peterhänsel (93).

On critical reflection it rapidly becomes clear that a wholly unsuitable comparison was used here and the groups were not comparable with each other: one group of patients who had undergone bariatric surgery was compared with a parallel group matched according to age and gender – from the normal population (92). However, the far more significant BMI was completely ignored, although it is known that persons with a higher BMI in particular also show a higher comorbidity of mental diseases (94).

However, if obese patients prepared to undergo a bariatric procedure are compared with a group of obese patients from the population, then the two groups do not differ with regard to their self-harming



Fig. 1 Lipoedema patient (weight 122 kg, height 168 cm, BMI 43 kg/m²) before sleeve gastrectomy. The volume of each leg was 19 litres).



Fig. 2 Lipoedema patient (weight 122 kg, height 168 cm, BMI 43 kg/m²) before sleeve gastrectomy. The volume of each leg was 19 litres).

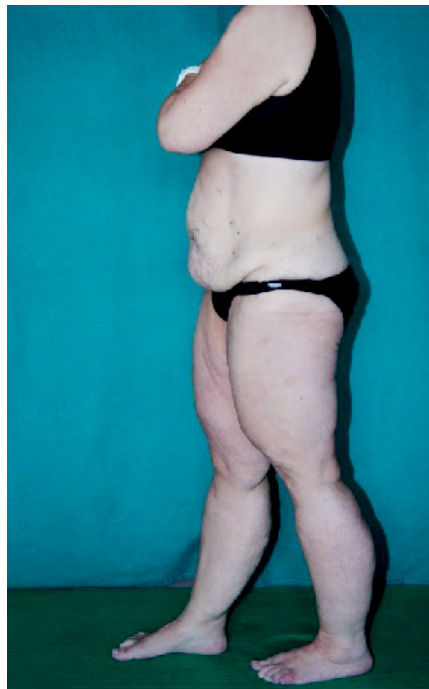


Fig. 3 Patient from Figs. 1 und 2, 11 months after bariatric surgery. Weight now 74 kg, BMI 26 kg/m².

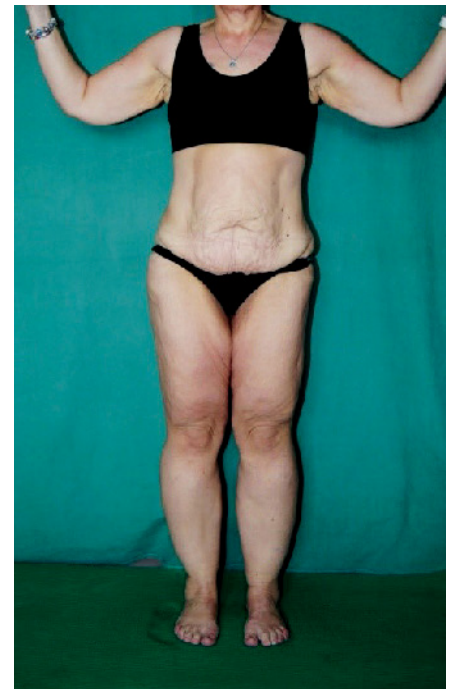


Fig. 4 Patient from Figs. 1 und 2, 11 months after bariatric surgery. Weight now 74 kg, BMI 26 kg/m².



Fig. 5 Patient with lipoedema and meanwhile also predominantly distal leg oedema.



Fig. 6 Patient from Fig. 5, one year later, after a gastric bypass.



Fig. 7 Patient from Fig. 5, one year later. Excess skin with subcutaneous fat.

behaviour or the frequency of attempted suicides (95). The alleged increased suicidality is therefore not – as is often wrongly assumed – the result of the bariatric surgery, but much rather the consequence of the increased psychological vulnerability of the group of patients with severe obesity.

To explain the suicides, Tindle et al (92) also concluded: “Suicides are not necessarily attributed to the bariatric surgery, but may be related to myriad factors”.

So Faerber’s argumentation comes to nothing. Irrespective of this, the importance of thorough preparation and aftercare of patients who subject themselves to surgery for obesity cannot be emphasised enough – also to identify those patients who require psychotherapeutic support and to provide it promptly and competently.

The treatment of morbidly obese people is only successful in the long term if specialised professional groups pull together: nutritionists, psychotherapists,

specialists in internal medicine, general practitioners as well as surgeons.

► Figures 1 and 2 show a lipoedema patient (weight 122 kg, height 168 cm, BMI 43 kg/m²) before a sleeve gastrectomy. The volume of each leg is 19 litres).

In ► Figures 3 and 4 we see the same patient 11 months after bariatric surgery. Her weight was now 74 kg, BMI 26 kg/m² and the leg volume per leg now 9 litres. The patient was completely free of symptoms, so that in this case we speak of “lipoedema in remission”. Plastic surgery to tighten the thigh skin was not absolutely necessary in this patient.

► Figure 5 shows a patient with lipoedema and also distal obesity related lymphedema of the legs prior to a gastric bypass operation, ► Figure 6 shows the same patient one year later. The remaining symptoms of lipoedema are, at most, mild and occur rarely; the obesity related lymphoedema of the legs also underwent a great improvement. The excessive skin with subcutaneous fat is obvious in ► Figure 7. The

next step in this case is dermatolipectomy by the plastic surgeons.

The lipoedema patient in ► Figures 8 and 9 lost 65 kg in weight within 14 months after a sleeve gastrectomy. After weight stabilisation of just one year, the plastic surgeons tightened the hanging belly overlying the genital region and the loose thigh skin. There were no longer any symptoms of lipoedema, so we could again speak of lipoedema in remission. In the case of this patient, there was certainly no longer any lipohypertrophy.

Conclusions

There is neither any scientific nor any empirical evidence for the popular statement that weight loss does not improve lipoedema. Our more than 10 years’ clinical experience points to the exact opposite. Persistent weight loss leads to a marked improvement in symptoms and the patients are often symptom-free. We then speak of lipoedema in remission.



Fig. 8 Lipoedema patient who lost 65 kg in weight within 14 months of a sleeve gastrectomy (Fig. 8 and Fig. 9: shown with the permission of Prof. Dr. N. Torio, Freiburg).



Fig. 9 After weight stabilisation of just 1 year, the hanging belly in the genital region and the loose thigh skin were tightened by plastic surgeons.

Outlook

In a fourth article, we will discuss the place of liposuction in lipoedema, so that in our final paper we can present a treatment concept that is not only scientifically based, but should contribute to a more persistent and more comprehensive improvement in the symptoms of our female lipoedema patients.

Conflict of interests

The authors declare that there is no conflict of interests.

Ethical guidelines

Preparation of the manuscript did not involve any studies on humans or animals.

References

1. Bertsch T, Erbacher G. Lipödem – Mythen und Fakten Teil 1. *Phlebologie* 2018; 47: 84–92.

2. Bertsch T. Adipositas-assoziierte Lymphödeme – unterschätzt und unterbehandelt. *Phlebologie* 2018; 47: 75–83.
3. Bertsch T, Erbacher G. Lipödem – Mythen und Fakten Teil 1. *Phlebologie* 2018; 47: 84–92.
4. Bertsch T, Erbacher G. Lipödem – Mythen und Fakten Teil 2. *Phlebologie* 2018; 47: 120–126.
5. Bertsch T, Martin KP. Adipositasprävalenz unter Lipödempatientinnen in einer kassenärztlichen lymphologischen Ambulanz im Jahr 2015 (unveröffentlichte Daten)
6. Bosman J. Lipoedema: Poor knowledge, neglect or disinterest? *Journal of Lymphoedema*, 2011, Vol 6, No 2, 109–111.
7. Child AH. Lipoedema: an inherited condition. *Am J Med Genet A*. 2010;152A(4):970–976.
8. STERN TV. Was Sie über Lipödeme wissen sollten. Abrufbar unter: <https://www.stern.de/tv/faq-krankhafte-fettverteilung--was-sie-ueber-lipoedeme-wissen-sollten-7339120.html>
9. Sputnik.de. Mysteriöse Krankheit, die dick macht: Was ist Lipödem? Abrufbar unter: <https://www.sputnik.de/sendungen/agesupdate/lipoedem-100.html>
10. Evidero.de. Dick durch Krankheiten und Medikamente: Dick oder krank? Diese Krankheiten beeinflussen Gewicht und Aussehen. Abrufbar unter: <https://www.evidero.de/krankheiten-beeinflussen-das-gewicht>
11. NDR.de. Markt vom 12.03.2018. Lipödem: Kasse zahlt Behandlung nicht. Abrufbar unter: <https://www.ndr.de/fernsehen/sendungen/markt/Seltene-Krankheit-Kasse-zahlt-Behandlung-nicht,markt12014.html>

12. YouTube. Lipödem im Bauch – gibt's das überhaupt? JA! Abrufbar unter: <https://www.youtube.com/watch?v=tjbdntLfo2Q>
13. Herbst KL. Obesity and Lipedema – What's the link? Abrufbar unter http://www.obesityaction.org/wp-content/uploads/Lipedema_and_Obesity_online.pdf
14. Obesityhelp.com. Could you have Lipedema? Abrufbar unter: <http://www.obesityhelp.com/articles/could-you-have-lipedema>
15. Stunkard AJ et al. An Adoption Study of Human Obesity. *N Engl J Med* 1986; 314: 193–198
16. Stunkard AJ et al. The Body-Mass Index of Twins who have been reared apart. *N Engl J Med* 1990; 322: 1483–1487.
17. Plagemann A (Ed.). *Perinatal Programming The State of the Art*. Berlin/Boston: Walter de Gruyter 2012; 11–22.
18. Herrera B et al. Genetics and epigenetics of obesity. *Maturitas* 2011; 69 (1): 41–49.
19. Hewagalamulage SD et al. Stress, Cortisol and obesity: a role for cortisol responsiveness in identifying individuals prone to obesity. *Domest Anim Endocrinol* 2016; 56 Suppl: 112–120.
20. Volkow ND et al. Obesity and addiction: neurobiological overlaps. *Obesity reviews* 2013 (1): 2–18
21. Nemiary D et al. The Relationship Between Obesity and Depression Among Adolescents. *Psychiatr Ann*. 2013; 42 (8): 305–308.
22. Seese B. Pathophysiologie der Adipositas. Abrufbar unter: https://www.cme-kurs.de/cdn2/pdf/Handout_Adipositas.pdf
23. Taubes G. *The Case against Sugar*. New York: Alfred A Knopf 2016.
24. Malik, V.S. & Hu, F.B. Sweeteners and Risk of Obesity and Type 2 Diabetes: The Role of Sugar-Sweetened Beverages. *Curr Diab Rep* (2012) 12: 195.
25. Wang JW. Consumption of added sugars and development of metabolic syndrome components among a sample of youth at risk of obesity. *Applied Physiology, Nutrition, and Metabolism*, 2014, 39(4) 512
26. Pietiläinen KH et al. Does Dieting Make You Fat? *International J of Obesity* 36 (3) 2012. 456–464
27. Robert Koch-Institut 2016. Studie DEGS1, Erhebung 2008–2011.
28. Sifferlin A. 40 % of Americans Are Obese – And the Trend Isn't Slowing. In *TIME HEALTH*, October 2017. Abrufbar unter: <http://time.com/4980225/obesity-rates-adults-children/>
29. WHO. Controlling the global obesity epidemic, 2017. Abrufbar unter: <http://www.who.int/nutrition/topics/obesity/en/>
30. Hilbert A et al. Stigmatisierung bei Adipositas. *In Adipositas* 2013; 3: 150–153.
31. Jung FU et al. Stigma and Knowledge as Determinants of Recommendation and Referral Behavior of General Practitioners and Internists. *Obes Surg* 2016; 26: 2393–2401.
32. Kimberly AG et al. Physicians build less rapport with obese patients 2013; 21 (10): 2146–2152.
33. Brownell KD et al. *Weight Bias: Nature, consequences and remedies*. New York: Guilford Press 2005.
34. Hilbert A, Geisert M. Stigmatisierung von Adipositas: Implikation für die Kommunikation mit adipösen Patienten. In: Lewandowski K, Bein T (Hrsg). *Adipositas in Anästhesie, Intensiv- und*

- Notfallmedizin. 2012. Medizinisch-Wissenschaftliche Verlagsgesellschaft 71–77.
35. Stunkard A. The Results of Treatment for Obesity. A Review of the Literature and Report of a Series. *AMA Arch Intern Med* 1959; 103(1): 79–85.
 36. Bennett W, Gurin J. The Dieter's Dilemma: Why Diets Are Obsolete—the New Setpoint Theory of Weight Control. New York: Basic Books 1982.
 37. Cogan J, Rothblum E. Outcomes of weight-loss Programms. *Genetic, Social and General Psychology Monographs* 1993; 118 (4).
 38. Perri MG. et al. Success and failure in the treatment of obesity: Where do we go from here? *Medicine, Exercise, Nutrition and Health* 1995; 4: 255–272.
 39. Hensrud DD, Weinsier RL. A prospective study of weight maintenance in obese subjects reduced to normal body weight without weight-loss training. *American clinical Nutrition* 1994; 60(5): 688–694
 40. Mann T, Tomiyama AJ et al. Medicare's search for effective obesity treatments: diets are not the answer. *Am Psychol* 2007; 62(3): 220–233.
 41. Nordmann A et al. Effects of low carb vs low fat diets on weight loss and cardiovascular risk factors: a meta analysis of randomized controlled trials. *Arch Internal Med* 2006; 166 (8).
 42. Fildes A, Charlton J. Probability of an obese person attaining normal body weight. *American Journal of Public Health* 2015.
 43. Pietiläinen KH et al. Does Dieting Make You Fat? *AAO*.
 44. Bosy-Westphal A. Effect of weight loss and regain on adipose tissue distribution, composition of lean mass and resting energy expenditure in young overweight and obese adults. *International Journal of Obesity* 2013; 1–7.
 45. Goddard S. I'm not fat, I've got lipedema. *BBC.com* Abrufbar unter: <http://www.bbc.co.uk/bbcthree/item/5d58f224-4bf7-498f-97d4-4073754a8f7f>
 46. Seo C. Movie: The disease they call fat. Abrufbar unter: <https://diseasestheycallfat.tv>
 47. Balduz A. Diagnose Lipödem: Der tägliche Kampf mit dem krankhaften Fett. Fokus am 24.1.2017. Abrufbar unter: https://www.focus.de/gesundheitsratgeber/frauenmedizin/mehr-als-drei-millionen-betroffene-diagnose-lipoedem-der-taegliche-kampf-mit-dem-krankhaften-fett_id_6522464.html
 48. Adipositasprogramm der Földiklinik. Unveröffentlichte Patientendaten
 49. Schmeller W, Meier-Vollrath I. Lipödem – Aktuelles zu einem weitgehend unbekanntem Krankheitsbild. *Akt Dermatol* 2007; 33(7): 251–260.
 50. Schmeller W, Meier-Vollrath I. Moderne Therapie des Lipödems: Kombination von konservativen und operativen Maßnahmen. *LymphForsch* 2004; 8(1): 22–26.
 51. Cornely M, Gensior M. Website abrufbar unter: <http://www.cg-lymph.de/de/krankheitsbilder/lipoedem>
 52. Herbst KL. Rare adipose disorders (RADs) masquerading as obesity. *Acta Pharmacologica Sinica* Volume 2012; 33: 155–172.
 53. Stern TV. Was Sie über Lipödem wissen sollten. Abrufbar unter: <https://www.stern.de/tv/faq-krankhafte-fettverteilung--was-sie-ueber-lipoedem-wissen-sollten-7339120.html>
 54. Kaniuth M. Dicke Beine trotz Diät: Mein Leben mit Lipödem. 2015. MGW-Verlag München S. 9
 55. Lipödemportal.de. Alles über das Lipödem. Abrufbar unter: <http://www.lipoedemportal.de/lipoedem-probleme.htm>
 56. Herbst KL. Rare adipose disorders (RADs) masquerading as obesity. *AAO*. S. 155.
 57. Schmeller W, Meier-Vollrath I. Lipödem – Aktuelles zu einem weitgehend unbekanntem Krankheitsbild. *AAO*. S. 256.
 58. Cornely M. Das Lipödem an Armen und Beinen. Teil 2: Zur konservativen und operativen Therapie des Lipödems, genannt Lipohyperplasia dolorosa. *Phlebologie* 2011; 40: 146.
 59. Website Dr. Stutz. Abrufbar unter: <http://stutz-dr.com/wp-content/uploads/2018/01/Download-File-1.pdf>
 60. Website Rosenbergklinik. Abrufbar unter: <http://www.rosenbergklinik.de/index.php?id=28>
 61. Website LipoClinic Dr. Heck. Abrufbar unter: <http://www.falk-heck.de/krankheitsbild-lipoedem-ursachen-problem-untersuchungen-stadien.html>
 62. Website Lipocura. Abrufbar unter: <https://lipocura.de/lipoedem/diagnose/>
 63. Lymphverein.de. Die typischen Kennzeichen des Lipödems. Abrufbar unter: <http://www.lymphverein.de/lipoedem.html>
 64. Allen E, Hines E. Lipedema of the legs: a syndrome characterized by fat legs and orthostatic edema. *Proc Staff Mayo Clin* 1940; 15: 184–187.
 65. Wold LE, Hines EA, Allen EV. Lipedema of he legs; a syndrome characterized by fat legs and edema. *Ann Intern Med* 1951; 34(5): 1243–1250.
 66. Bertsch T. Adipositas-assoziierte Lymphödeme – unterschätzt und unterbehandelt. *AAO*.
 67. Herpertz S, Kielmann R, Wolf AM, et al. Does obesity surgery improve psychosocial functioning? A systematic review. *Int J Obes Relat Metab Disord* 2003; 27: 1300–1314.
 68. Buddeberg-Fischer B, Klaghofer R, Sigrist S, et al. Impact of psychosocial stress and symptoms on indication for bariatric surgery and outcome in morbidly obese patients. *Obes Surg* 2004; 14: 361–369.
 69. Buchwald H, Estok R, Fahrback K, et al. Weight and type 2 diabetes after bariatric surgery: systematic review and meta-analysis. *Am J Med* 2009; 122: 248–256.
 70. Wittgrove AC, Clark GW. Laparoscopic Gastric Bypass, Roux-en-Y – 500 Patients: Technique and Results, with 3–60 month follow-up. *Obes Surg* 2000; 10: 233.
 71. Sugerman HJ et al. Diabetes and hypertension in severe obesity and effects of gastric bypass-induced weight loss. *Ann Surg* 2003 237: 751–756.
 72. Rasheid S et al. Bypass is an Effective Treatment for Obstructive Sleep Apnea in Patients with Clinically Significant Obesity. *OBES SURG* 2003; 13: 5.
 73. Courcoulas AP et al. Long-term Outcomes of Bariatric Surgery: A National Institutes of Health Symposium. *JAMA Surg*. 2014; 149 (12): 1323–1329.
 74. Arterburn DE et al. Association Between Bariatric Surgery and Long-term Survival. *JAMA* 2015; 313(1): 62–70.
 75. Sjöström L et al. Effects of bariatric surgery on mortality in Swedish obese subjects. *N Engl J Med* 2007; 357: 741–752.
 76. Adams TD et al. (2007) Long-term mortality after gastric bypass surgery. *N Engl J Med* 357: 753–761
 77. Shubeck S et al. Long-term Outcomes Following Bariatric Surgery. *JAMA* 2018; 319(3): 302–303.
 78. Faerber G. Adipositas und chronische Inflammation bei phlebologischen und lymphologischen Erkrankungen. *Phlebologie* 2018; 47: 55–65.
 79. Lim EL et al. Reversal of type 2 diabetes: normalisation of beta cell function in association with decreases pancreas and liver triacylglycerol. *Diabetologia* 2011; 54: 2506–2514.
 80. Steven S, Taylor R. Restoring hyperglycaemia by very low calorie diet in long and short term diabetes. *Diabet Med* 2015; 32(9): 1149–1155.
 81. Stunkard A. The Results of Treatment for Obesity. A Review of the Literature and Report of a Series. *AMA Arch Intern Med* 1959; 103(1): 79–85.
 82. Bennett W, Gurin J. The Dieter's Dilemma: Why Diets Are Obsolete—the New Setpoint Theory of Weight Control. New York: Basic Books 1982.
 83. Cogan J Rothblum E. Outcomes of weight-loss Programms. *Genetic, Social and General Psychology Monographs* 1993; 118 (4).
 84. Perri MG et al. Success and failure in the treatment of obesity: Where do we go from here? *Medicine, Exercise, Nutrition and Health* 1995; 4: 255–272.
 85. Hensrud DD, Weinsier RL. A prospective study of weight maintenance in obese subjects reduced to normal body weight without weight-loss training. *American clinical Nutrition* 1994; 60(5): 688–694.
 86. Mann T, Tomiyama AJ et al. Medicare's search for effective obesity treatments: diets are not the answer. *Am Psychol* 2007; 62(3): 220–233.
 87. Nordmann A et al. Effects of low carb vs low fat diets on weight loss and cardiovascular risk factors: a meta analysis of randomized controlled trials. *Arch Internal Med* 2006; 166 (8).
 88. Fildes A, Charlton J. Probability of an obese person attaining normal body weight. *American Journal of Public Health* 2015.
 89. Dulloo AG et al. How dieting makes the lean fatter. *Obesity reviews* 2015; 16 (S1): 25–35.
 90. Lowe MR. Dieting: Proxy or Cause of Future weight gain? *Obesity Reviews* 2015; 16 (S1): 19–24.
 91. Pietiläinen KH et al. Does dieting make you fat? A Twin study. *International Journal of Obesity* 2012; 36 (3): 456–464.
 92. Tindle HA, Omalu B, Courcoulas A. Risk of suicide after long-term follow-up from bariatric surgery. *Am J Med* 2010; 123: 1036–1042.
 93. Peterhänsel C, Petroff D, Klinitzke G, Kersting A, Wagner B. Risk of completed suicide after bariatric surgery: a systematic review. *Obes Rev* 2013 May; 14(5): 369–382. doi: 10.1111/obr.12014. Epub 2013 Jan 9.
 94. Baumeister H, Härter M. Mental disorders in patients with obesity in comparison with healthy probands. *Int J Obesity* 2007; 31(7): 1155–1164.
 95. Müller A, Claes L, Smits D, Schag K, de Zwaan M. Lifetime Self-Harm Behaviors Are Not More Prevalent in Bariatric Surgery Candidates than in Community Controls with Obesity. *Obes Facts* 2018; 11: 109–115.
 96. Download picture from: <https://wellroundedma.blogspot.com/2015/07/lipedema-treatment-part-5b-weight-and.html>