



Improvement of Microvascular Function in Patients with Morbid Obesity After Bariatric Surgery Revealed by Imaging Photoplethysmography

Maria E. Vasilieva¹ · Victor A. Kashchenko^{1,2,3} · Elena V. Shmidt^{1,2} · Irina A. Mizeva⁴ · Polina M. Dolotovskaya⁵ · Valery V. Zaytsev¹ · Nikita B. Margaryants⁶ · Alexei A. Kamshilin^{1,7}

Received: 23 November 2024 / Revised: 29 January 2025 / Accepted: 4 February 2025

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Abstract

Background Morbid obesity leads to serious complications such as diabetes mellitus, arterial hypertension, and atherosclerosis. Bariatric surgery aims to reduce body weight and correct metabolic disorders and associated macro- and microvascular dysfunction. Metabolic disturbances can be assessed by biochemical markers, whereas microvascular function can be assessed by the response to provocative stimuli. The aim of this work is to quantitatively assess the cutaneous microcirculatory change caused by bariatric surgery using imaging photoplethysmography.

Methods The study included 20 patients with obesity whose body mass index (BMI) was greater than 40 kg/m² who underwent bariatric surgery and 20 control subjects. Microvascular function was assessed twice, before and 6 months after surgery, by measuring the perfusion response to local forearm heating up to 40 °C using imaging photoplethysmography.

Results The perfusion response to local heating was found to be significantly lower in patients with obesity before surgery compared to the control group, but 6 months after surgery, it approaches indicators of the control group, along with a decrease in BMI from 48 ± 5 to 36 ± 5 kg/m², $P < 0.001$. Besides, bariatric surgery improves biochemical markers of metabolic syndrome (glycated hemoglobin decreases from 6.3 ± 1.0 to 5.2 ± 0.4% and cholesterol from 5.2 ± 1.4 to 4.2 ± 0.8 mmol/l).

Conclusion Based on these results, we conclude that microvascular abnormalities caused by obesity could be repaired after bariatric surgery and subsequent conservative treatment.

Keywords Bariatric surgery · Microcirculation · Imaging photoplethysmography · Microvascular function · Morbid obesity

✉ Alexei A. Kamshilin
alexei.kamshilin@yandex.ru

Maria E. Vasilieva
vasilevamarina28@gmail.com

Victor A. Kashchenko
surg122@yandex.ru

Elena V. Shmidt
e.shmidt@list.ru

Irina A. Mizeva
mizeva@icmm.ru

Polina M. Dolotovskaya
p.yurkova.p@yandex.ru

Valery V. Zaytsev
Zaytsevphoto@gmail.com

Nikita B. Margaryants
nikita.optic@gmail.com

¹ North-Western District Scientific and Clinical Center Named After L.G. Sokolov, FMBA, Saint Petersburg, Russian Federation

² Saint Petersburg State University, Saint Petersburg, Russian Federation

³ Beloostrov Clinic, Vsevolozhsk District, Leningrad Region, Russian Federation

⁴ Institute of Continuous Media Mechanics, Perm, Russian Federation

⁵ Institute of the Human Brain, Saint Petersburg, Russian Federation

⁶ ITMO University, Saint Petersburg, Russian Federation

⁷ Institute of Automation and Control Processes, Primorskiy, Russian Federation

Introduction

Excessive accumulation of adipose tissue in the body can cause serious diseases such as diabetes mellitus, arterial hypertension, atherosclerosis, coronary heart disease, and stroke [1]. Impaired microvascular vasomotor function is a serious complication of obesity, manifesting itself in the form of a significant reduction in skin microcirculation due to a lower vasodilatory ability of microvessels [2], and which aggravates as the body mass index (BMI) increases [3]. As a result of obesity, adipocyte dysfunction, oxidative stress, insulin resistance, and inflammation develop, leading to microvascular dysfunction [4]. Microvascular dysfunction is an early stage of atherosclerosis associated with an increased risk of developing cardiovascular diseases [5, 6] and also contributes to the onset of various microvascular complications.

Recently, much attention has been paid to the study of microcirculatory dysfunction in people with obesity [7]. In obesity, microvascular disease is mediated by adipokines/cytokines causing chronic, subclinical inflammation with reduced NO-mediated dilatation, changed endothelial- and smooth muscle-dependent vasoregulatory mechanisms, altered vasomotor control with increased sympathetic activity, and impaired cardiovascular adaptation to metabolic demands [8]. These changes form the pathological basis for the development of microcirculatory complications in patients with obesity, particularly in the context of insulin resistance and chronic inflammation [9]. A comprehensive program is currently used to treat obesity, including bariatric surgery, which is applied when non-invasive approaches such as diet, increased physical activity, and drug therapy do not provide satisfactory results. Bariatric surgery is aimed not only to reduce body weight but also to correct metabolic disorders and associated vascular impairments, including hyperglycemia, diabetes mellitus, and hypercholesterolemia [10]. Regarding the influence on microcirculation, it has been shown that bariatric surgery reduces the severity of microvascular manifestations of diabetes mellitus [11, 12], reduces the risk of developing retinopathy [13], and improves blood microcirculation in the retina [14]. Bariatric surgery has also been shown to improve both coronary microcirculation and peripheral microvascular reactivity [15–17]. Structural changes in the microcirculation in patients with severe obesity are associated with rarefaction of the capillary network, and even with a significant reduction in BMI caused by bariatric surgery, the density of the capillary network is not restored [18]. It has been found that in severe obesity, the rigidity of vessel walls in the microcirculatory system does not increase, unlike large and medium-sized arteries [18], while the mechanical properties of vessels in people with

obesity and the control subjects are similar and practically do not depend on a reduction in BMI [19]. However, the significant reduction in body weight resulting from bariatric surgery leads to a significant increase in the number of endothelial progenitor cells and collagen content [19], as well as to normalization of blood rheological parameters [20].

Methods to assess microcirculation impairment are usually based on measurements of cutaneous blood flow in response to a provocative stimulus [7, 21]. A number of studies have shown that in the long term, the bariatric surgery leads to normalization of microcirculation and endothelial function [22–24]. However, ultrasound examination of the blood flow response in carotid arteries to arterial occlusion before and after bariatric surgery showed a deterioration in endothelial function [25], which contradicts the results of other studies that reported its improvement [16, 26, 27]. In addition, there are studies that have indicated no improvement in endothelial function after bariatric surgery [28–30]. Therefore, given the methodological difficulties in quantifying blood flow, the effect of bariatric surgery on microcirculatory parameters requires more in-depth study.

Recently, imaging photoplethysmography (iPPG) has been demonstrated to be effective in assessing microcirculatory parameters during a local forearm heating test [31]. The iPPG technique made it possible to identify the presence of endothelial dysfunction in patients with high cardiometabolic risk [32]. The aim of our study was to assess cutaneous perfusion parameters in patients with morbid obesity using iPPG and to evaluate their changes after bariatric surgery.

Materials and Methods

The study involved 20 patients with metabolic syndrome, 12 of whom were diagnosed with type 2 diabetes mellitus. All patients had stage 3 obesity (BMI > 40 kg/m²), and all of them underwent bariatric surgery, namely laparoscopic mini-gastric bypass surgery combining restrictive and malabsorptive mechanisms. This type of surgery was chosen because of its high efficacy in reducing body weight, improving metabolic parameters such as insulin resistance and hypercholesterolaemia, and correcting cardiometabolic risks [33, 34]. Assessment of cutaneous perfusion response to local heating was carried out in all patients before and 6 months after bariatric surgery. In addition, anthropometric characteristics, cholesterol, and glycated hemoglobin were measured both before and 6 months after surgery (see Table 1). The control group consisted of 20 healthy normotensive subjects aged 27 ± 10 years (5 men and 15 women) without cardiovascular or metabolic diseases and with BMI within the normal range. Exclusion criteria included a history of obesity, recent major surgeries, or any other chronic

Table 1 Clinical and anthropometric characteristics of patients with obesity

Parameter	Before the surgery	After the surgery	Significance of difference
Number (male/female)	20 (3/17)	20 (3/17)	
Age (years)	42 ± 13	42 ± 13	
BMI (kg/m ²)	48 ± 5	36 ± 5	<i>P</i> < 0.001
Glycated hemoglobin (%)	6.3 ± 1.0	5.2 ± 0.4	<i>P</i> < 0.001
Cholesterol (mM/l)	5.2 ± 1.4	4.2 ± 0.8	<i>P</i> = 0.004

BMI body mass index

health conditions that could affect microcirculation. In the control group, microcirculation response to local heating was measured in all 20 subjects at the beginning of the study and repeated in 11 of them 6 months later to simulate the data collection protocol in patients with obesity. All patients and subjects were informed of the study objectives and protocol and signed an informed consent form.

Local Heating Test

Measurements of changes in cutaneous microcirculation in response to local heating were performed in a darkened room at a temperature of 23 ± 1 °C. Prior to the study, the subject adapted to the measurement conditions for approximately 15 min. A schematic of the experimental setup is

shown in Fig. 1. The subject was in a comfortable supine position. We used a multimodal system described in detail in our recent paper [31], which distinctive feature is the synchronous registration of the iPPG signal and an electrocardiogram (ECG) to assess microcirculatory dynamics during a local heating test. Briefly, perfusion assessment was performed on the subject's right forearm by video recording of a sufficiently large area of skin (about 2×5 cm²) through a transparent temperature-controlled electric heater (Fig. 1B). Vaseline oil was used to ensure heat transfer from the heater to the skin. To minimize the impact of the skin-heater contact on microcirculation, the heater pressure on the skin was controlled to be constant during the measuring session and not to exceed 15 mmHg in all subjects. The iPPG module (Fig. 1C) consisted of 8 green LEDs emitting at a

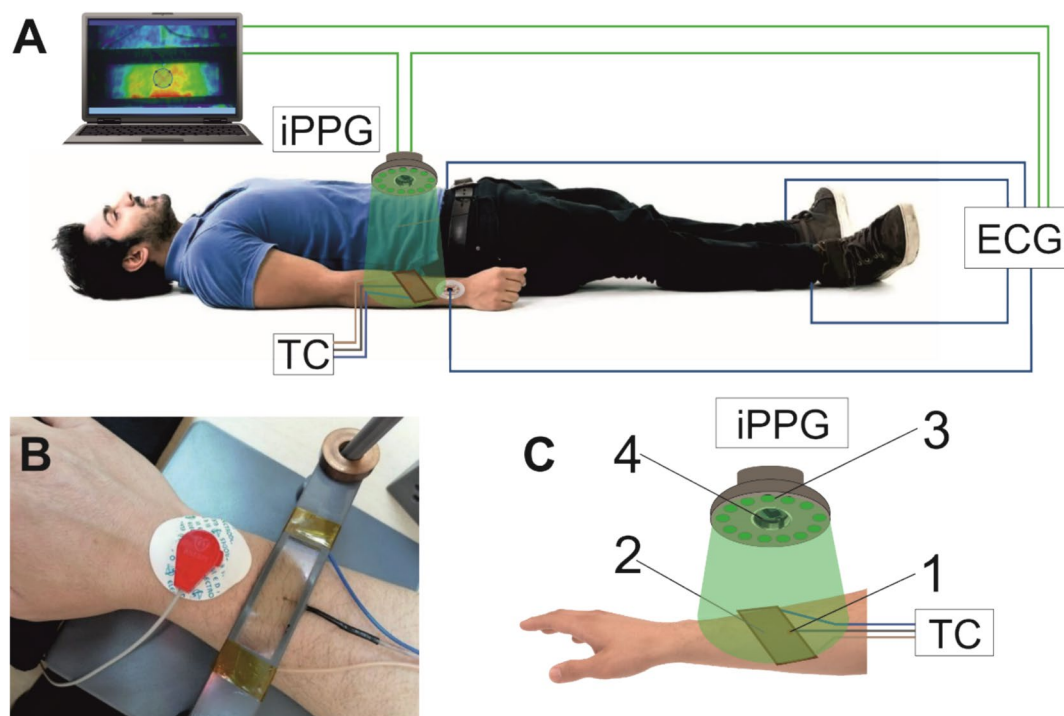


Fig. 1 Schematic of the experimental setup. **A** General view of the installation consisting of three modules: imaging photoplethysmography (iPPG), electrocardiograph (ECG), and temperature controller

(TC). **B** Photo of the heating element on the subject's forearm. **C** TC and iPPG modules: (1) temperature sensor, (2) transparent heating element, (3) green light emitting diodes, and (4) video camera lens

wavelength of 530 ± 25 nm and a digital camera recording of 480×752 pixels frames at a rate of 39 frames per second. Both the video frames and the ECG data were stored on a personal computer for further offline processing.

The study protocol included three stages: (i) basal recording for 5 min; (ii) turning on the heater to heat the skin up to 40°C at a rate of 1°C in 20 s, followed by maintaining a stable skin temperature at 40°C ; and (iii) turning off the heater at the 25th minute of the session and recording skin blood flow relaxation for further 20 min.

Data Processing

The dynamics of the perfusion in the forearm area was assessed using custom software implemented on the MATLAB® platform (MathWorks Inc., MA, USA). We used an algorithm that differs significantly from that used previously [31, 32]. The main difference is that the averaging of iPPG signals in time (over 12–15 cardiac cycles) is replaced by averaging in space. In the first step of the algorithm, the spatial distribution of the perfusion index (PI) is calculated for only one cardiac cycle, starting at 600th second. The iPPG waveform consists of an alternating component (AC), which oscillates with heart rate, and a slowly varying component (DC). In this step, PI is calculated as the ratio of the

AC amplitude to average DC in each cardiac cycle for every 2×2 pixel region of the image. Examples of PI mappings encoded in pseudo-colors and superimposed on the image of subject's forearm with the heater are shown in Fig. 2A. In the second step, in the calculated perfusion distribution, we selected a region of interest (ROI) of about 1 cm^2 in the area with the most pronounced perfusion (circles in Fig. 2A). In the third step, we repeated the PI calculations but did so only for the selected ROI and continuously over the whole session using a floating time-window of duration equal to the mean cardiac cycle to compensate for motion artifacts. While evaluating PI, the beginning and end of each cardiac cycle were accurately determined using the synchronously recorded ECG, and unavoidable motion artifacts were compensated by means of a multi-section image stabilization algorithm proposed in our previous paper [35]. These calculations allowed us to assess the spatial distribution of the PI within the ROI for every cardiac cycle during the entire heating test. After averaging the PI over the ROI, we obtain the PI dynamics (Fig. 2B).

Statistical Analysis

The normality of the distribution of experimental data was tested using the Shapiro–Wilk test. In the case of normal

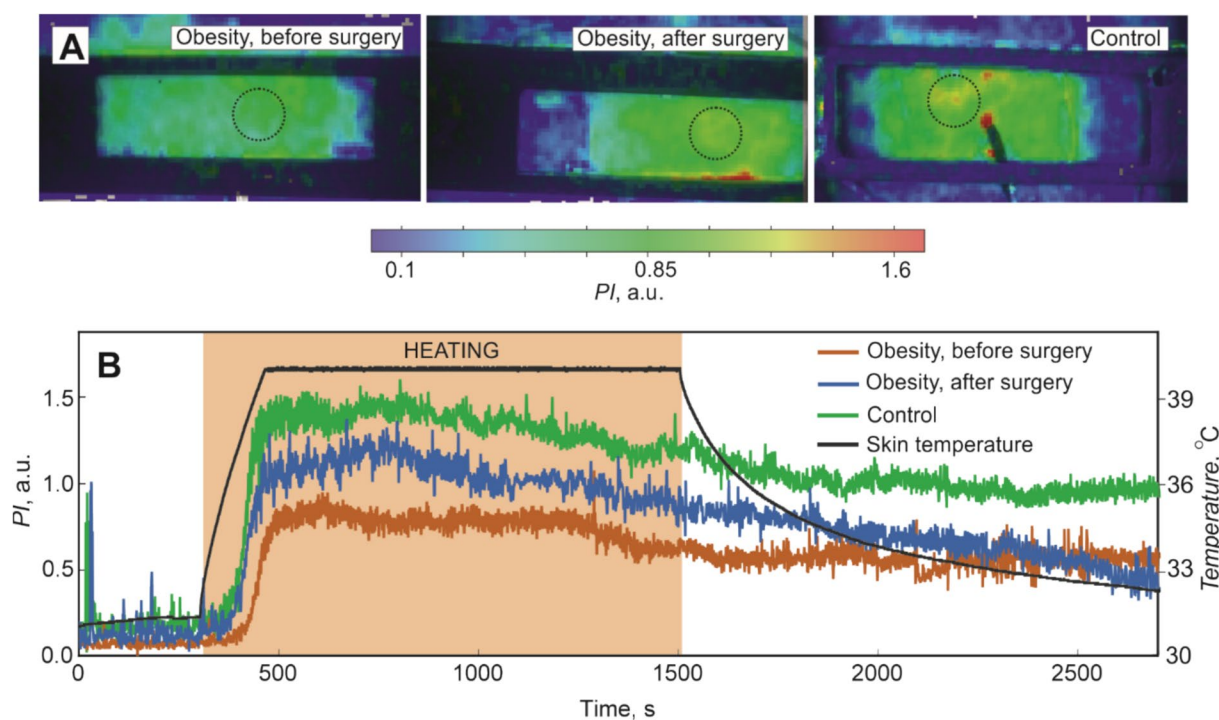


Fig. 2 Cutaneous microcirculation responses to local forearm heating in a patient with obesity (before and after bariatric surgery) and control subject. **A** Map of the perfusion index (PI) distribution assessed through a transparent heater on the forearm at 600 s (300 s from the start of heating), a dark rectangle in the images shows the heater

frame. **B** Evolution of PI averaged within the circles marked in the images shown in panel **A**: red curve for the patient before surgery, blue curve in the same patient 6 months after surgery, green curve for the control subject, and black curve is skin temperature dynamics

distribution, the data was expressed as mean with standard deviation ($M \pm SD$) and otherwise as median with interquartile range ($Me[Q1-Q3]$). The significance of the difference in independent samples was estimated by the Mann–Whitney test and in pairs by the Wilcoxon test. A 95% confidence interval was used, and the significance level of all of the statistical indicators is $P < 0.05$.

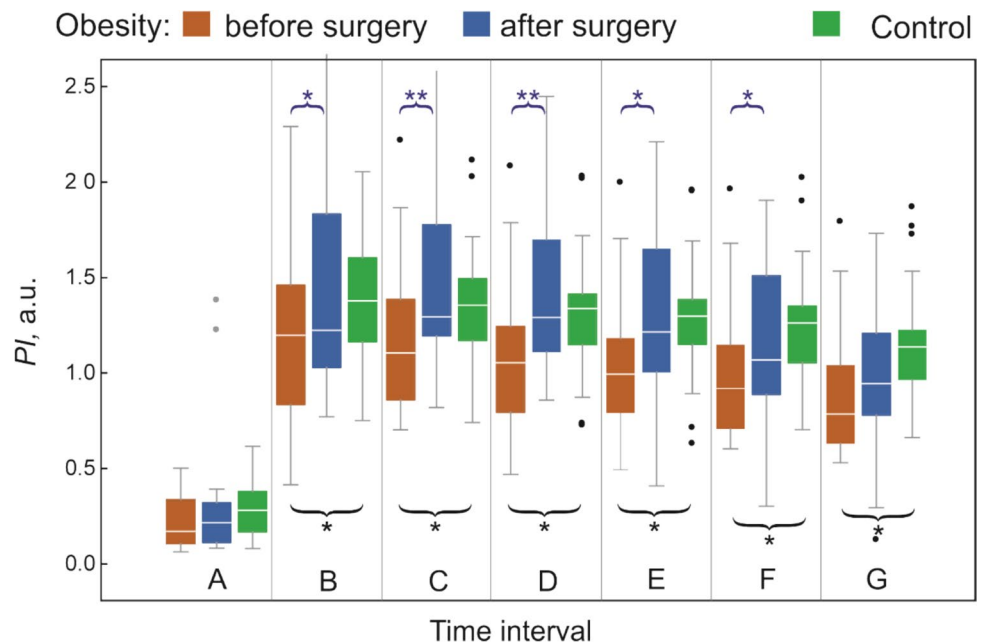
Results

Forearm skin perfusion before local heating (baseline) is not statistically different when comparing either patients with obesity before and after surgery ($0.17[0.10-0.33]$ vs $0.21[0.11-0.30]$, $P = 0.5$, paired Wilcoxon test) or control and preoperative patients ($0.28[0.16-0.38]$ vs $0.17[0.10-0.33]$, $P = 0.052$, Mann–Whitney test) or control and postoperative patients ($0.28[0.16-0.38]$ vs $0.21[0.11-0.30]$, $P = 0.22$, Mann–Whitney test). Heating to 40°C leads to a multiple increase in PI in both patients and control subjects, as shown in Fig. 2B, where examples of PI dynamics are given for a patient with obesity before and after bariatric surgery (by the red and blue lines, respectively) and for the control subject by the green line. It is clearly seen that the response of PI to heating, measured in the same patient 6 months after the surgery, is higher than before surgery but both responses are weaker than in the control subject. It is worth noting that in all the cases shown, the perfusion index remains quite high for a long time after the heating is switched off, despite a fairly rapid decrease in skin temperature.

As one can see in Table 1, the result of bariatric surgery was both a decrease in BMI in all patients with obesity and a significant improvement in biochemical markers of the metabolic syndrome (cholesterol and glycated hemoglobin). To assess changes in the microcirculation response to heating against the background of a reduction in BMI after bariatric surgery, we selected seven time intervals in which PI values were averaged and compared both between patients before and 6 months after surgery and between each of these group and the control group. These time intervals are as follows: A (50–250 s), B (400–500 s), C (500–750 s), D (750–1000 s), E (1000–1250 s), F (1250–1500 s), and G (1500–1750 s). Boxplots of the data evaluated in these intervals are shown in Fig. 3. During the basal recording (interval A, 50–250 s), no statistical difference in perfusion index was found between any of the groups. However, in intervals II–VI (from 400 to 1500 s), there is a statistically significant increase in PI in patients with obesity after bariatric surgery compared to what they had before surgery (paired Wilcoxon test). The most significant increase in PI is observed in the interval C (500–750 s): $1.10[0.85-1.28]$ vs $1.29[1.18-1.69]$, $P = 0.005$. It is worth noting that after turning off the heater (interval G, 1500–1750 s), PI in patients before and after surgery is closer: $0.78[0.61-1.04]$ vs $0.94[0.78-1.14]$, so the difference is not significant, $P = 0.22$.

Figure 3 also shows that after heating (intervals B to G, from 400 to 1750 s), perfusion in the group of preoperative patients becomes significantly less than in the control group. In contrast, there is no statistically significant difference between the postoperative patients and control subjects in any of the time intervals. Note that similar studies in the control group assessing the perfusion response in 11 subjects

Fig. 3 Boxplots of the perfusion index (PI) averaged within each of the seven intervals (whose positions on the time scale are given in the text) for patients before surgery (red boxes), 6 months after surgery (blue boxes), and for control subjects (green boxes). The statistical significance of the difference between the boxes connected by curly brackets is indicated by * when $P < 0.05$ and ** when $P < 0.01$



twice with a difference of 6 months also showed no statistically significant difference in pairwise comparison.

Discussion

The perfusion response to the local skin heating is a well-established provocative stimulus for microvascular function assessment [36]. Our study has revealed that people with obesity have a significantly lower perfusion response to the local forearm heating of compared to the control subjects. Nevertheless, 6 months after bariatric surgery, the response increases and approaches to that of healthy subjects. It is worth noting that under basal conditions (before heating), there was no statistically significant difference in forearm perfusion between patients with obesity and control subjects. In this study, the cutaneous microcirculation and its response to heating were assessed using the multimodal system consisting of imaging photoplethysmography synchronized with an electrocardiogram. This technique is advantageous for its simplicity, accessibility, and the ability to monitor cutaneous perfusion with minimal external impact on the vasculature. In addition, the synchronous recording of video frames of the region under study and an electrocardiogram allows us to significantly suppress motion artifacts and increase the signal-to-noise ratio [31, 35], which is extremely important for the correct and reliable assessment of tissue perfusion and its dynamic changes *in vivo*. It should be emphasized that effective heat transfer from the heater to the epidermis requires the presence of thermally conductive contact between them. However, any physical contact with living tissue will seriously affect the blood flow in the area of contact. In our iPPG system, this effect is both minimized and controlled throughout the data acquisition process. The minimization of the contact influence was achieved by using a sufficiently large contact area ($> 5 \text{ cm}^2$), while the monitoring of the heater-skin pressure was possible due to the fact that the vaseline-oil boundary is well identified in each image, allowing us to assess the actual contact area.

In every cardiac cycle, the main force driving red blood cells (RBC) in vessels is the difference between systolic and diastolic pressure. The greater this difference, the more RBC will be transported during the cardiac cycle to provide tissue perfusion. According to the recently proposed theory of the iPPG-signal formation [37], it is this pressure difference that causes the green light to be modulated with heart rate, thereby determining the AC/DC ratio of the iPPG signal or PI. Another implication of the new iPPG theory is that the superficial capillary layer serves as a distributed sensitive network for assessing the state of the deep arterial vessels feeding the area under study [38]. However, the pressure-to-PI conversion factor can vary greatly from one biological tissue to another, as well as from one subject to another.

This may be the main reason why our study did not reveal a statistically significant difference in PI between people with obesity and control subjects under basal conditions (before forearm heating). However, local forearm heating in patients with obesity resulted in a significantly lower increase in PI than in the control group or in the same patients, but after bariatric surgery. This suggests that in patients with obesity, relaxation of microvascular tone caused by the local skin heating is impaired, indicating an abnormality in microvascular function.

The mechanisms underlying the remission of type 2 diabetes mellitus after bariatric surgery are extensively discussed [39]. Meta-analyses, non-randomized, and randomized clinical trials have shown that metabolic surgery is the most effective treatment for patients with type 2 diabetes mellitus [11, 40]. Compared to conservative treatment and lifestyle changes, metabolic surgery provides better results in terms of improving glycemia [41]. In our study, a significant decrease in the level of glycated hemoglobin (from 6.3 ± 1.0 to $5.2 \pm 0.4\%$) was demonstrated 6 months after surgery. The basics of glycemic control in bariatric surgery are not yet fully understood but involve complex interaction between improvements in beta-cell function and insulin secretion, insulin sensitivity, intestinal gluconeogenesis and changes in intestinal glucose utilization and absorption, and changes in the secretory pattern and morphology of adipose tissue [40]. Several contributions are proposed, such as calorie restriction, elimination of glucose toxicity, improvement of insulin resistance, and changes in signals coming from the gastrointestinal tract [42]. In our study, we achieved a reduction in BMI from 48 ± 5 to 36 ± 5 in 6 months, while it has previously been shown that in a late postoperative period, weight loss caused by bariatric surgery plays an important role and is the main driver of additional improvement in insulin sensitivity [10, 11]. Significant weight loss leads to a reduction in adipose tissue and an improvement in body composition, mainly due to visceral intramuscular fat depots. Adipose tissue alters the secretory profile, adipocyte morphology, glucose, and lipid metabolism. All these reasons lead to the diabetes remission by bariatric surgery. It is known that in remission of type 2 diabetes mellitus, microvascular function is restored, and even microvascular morphology improves [43, 44]. The aim of the present study was to determine whether bariatric surgery leads to changes in any microcirculatory parameter assessed by iPPG. We focused on identifying the overall effect of surgery, rather than analyzing correlations with indicators such as BMI, glycated hemoglobin, percentage of adipose tissue, or medication use. The study of the effect of glycated hemoglobin on biophysical markers of microvascular dysfunction will be useful and may provide new insights into the field of physiology and pathophysiology of the blood circulation. In this work, we have made the first attempt to assess whether iPPG can identify at least one

parameter of microcirculatory function that has been significantly altered as a result of bariatric surgery.

Our results show that bariatric surgery significantly improves the perfusion response to local heating, which is undoubtedly one of the parameters of microvascular function. It should be noted that improvements in perfusion after surgery are similar to changes observed with pharmacological treatment of type 2 diabetes or a low-carbohydrate diet [45, 46]. However, the surgical approach shows a more consistent effect in normalizing microvascular function, which may be associated with greater changes in body weight and reduction in chronic inflammation [10, 47]. In contrast to the transient effects observed with conservative therapy, surgery provides long-term benefits in improving cardiometabolic health. Our future plans include studying the relationships between perfusion parameters, metabolic indices, and body weight loss to better elucidate the mechanisms of the effects of surgical treatment on the microcirculation.

Study Limitation

The age difference between the groups is a potential limitation, as the mean age of the control group was lower than that of the patient group, albeit with notable variance due to the presence of older subjects. The age of the control group has a non-Gaussian distribution, which may influence the outcomes. However, our findings suggest a lower likelihood of microvascular dysfunction in older healthy subjects as compared with age-matched patients with obesity. Future studies may be aimed at selecting an age-appropriate control group to further elucidate the age-related risk factors for microvascular complications in obesity.

Conclusion

BMI reduction caused by complex surgery and conservative treatment for 6 months improves the cutaneous microvascular function. Based on our results, obtained by the assessment of the perfusion index evaluated by the multimodal iPPG + ECG system, we conclude that microvascular dysfunction caused by obesity is reversible. Our results confirm that bariatric surgery is an effective method of correcting microcirculatory abnormalities in morbidly obese patients. The improvement of microcirculatory function is associated with both weight reduction and improved glycemic control, highlighting the importance of surgery in the long-term prevention of cardiovascular complications.

Author Contributions M.V., V.K., and A.K. drafted the study plan; M.V., V.K., and E.S. were responsible for the both therapeutic and

surgical treatment of patients with obesity; A.K. and V.Z. developed the conceptual framework for iPPG video analyses, designed and manufactured the technical setup as well as developed the data processing software; M.V., P.D., and N.M. collected the experimental data; I.M., N.M., and A.K. processed and analyzed the experimental data; I.M. prepared the figures; I.M. and A.K. wrote the main manuscript text; All authors read, reviewed, and approved the manuscript.

Funding Ministry of Science and High Education of Russia (agreements 125020301282-0 and 124012300246-9); Government of the Russian Federation for state support of scientific research carried out under the supervision of leading scientists (agreement 075-15-2022-1110).

Data Availability The datasets generated during and/or analyzed during the current study are available from the corresponding author upon reasonable request.

Declarations

Competing Interest The authors declare no competing interests.

Ethics Approval All procedures performed in studies involving human participants were in accordance with the ethical standards of the institutional committee and with the 1964 Helsinki Declaration and its later amendments or comparable ethical standards. The study was approved by the Local Ethic Committee of the North-Western District Scientific and Clinical Center named after L. G. Sokolov (Protocol No. 3 of March 13, 2023).

Informed consent Informed consent was obtained from all individual participants included in the study.

References

1. Martínez-Núñez AE, Gamboa-López OE, Bacardí-Gascón M, et al. Long-term complications and side effects of bariatric surgery: a systematic review. *J Negat No Posit Results*. 2017;2:410–5.
2. Bagi Z, Feher A, Cassuto J. Microvascular responsiveness in obesity: implications for therapeutic intervention. *Br J Pharmacol*. 2012;165:544–60.
3. Andreieva IO, Riznyk OI, Myrnyi SP, et al. State of cutaneous microcirculation in patients with obesity. *Wiadomości Lek*. 2021;74:2039–43.
4. Koenen M, Hill MA, Cohen P, et al. Adipose tissue and vascular dysfunction. *Circ Res*. 2021;128:951–68.
5. Ross R. Atherosclerosis - an inflammatory disease. *N Engl J Med*. 1999;340:115–26.
6. Higashi Y, Noma K, Yoshizumi M, et al. Endothelial function and oxidative stress in cardiovascular diseases. *Circ J*. 2009;73:411–8.
7. Sorop O, Olver TD, van de Wouw J, et al. The microcirculation: a key player in obesity-associated cardiovascular disease. *Cardiovasc Res*. 2017;113:1035–45.
8. Selthofer-Relatić K, Bošnjak I, Kibel A. Obesity related coronary microvascular dysfunction: from basic to clinical practice. *Cardiol Res Pract*. 2016;2016:8173816.
9. Deanfield JE, Halcox JP, Rabelink TJ. Endothelial function and dysfunction. *Circulation*. 2007;115:1285–95.
10. Courcoulas AP, Yanovski SZ, Bonds D, et al. Long-term outcomes of bariatric surgery: a national institutes of health symposium. *JAMA Surg*. 2014;149:1323–9.
11. Sheng B, Truong K, Spitler H, et al. The long-term effects of bariatric surgery on type 2 diabetes remission, microvascular and

- macrovascular complications, and mortality: a systematic review and meta-analysis. *Obes Surg*. 2017;27:2724–32.
12. O'Brien R, Johnson E, Haneuse S, et al. Microvascular outcomes in patients with diabetes after bariatric surgery versus usual care. *Ann Intern Med*. 2018;169:300–10.
 13. Åkerblom H, Franzén S, Zhou C, et al. Association of gastric bypass surgery with risk of developing diabetic retinopathy among patients with obesity and type 2 diabetes in Sweden: an observational study. *JAMA Ophthalmol*. 2021;139:200–5.
 14. Debourdeau E, Gardes G, Nocca D, et al. Longitudinal effect of bariatric surgery on retinal microcirculation and target organ damage: the BASTOD study. *Obes Surg*. 2022;32:1–10.
 15. Karimzad S, Shokr H, Bellary S, et al. The effect of bariatric surgery on microvascular structure and function, peripheral pressure waveform and general cardiovascular risk: a longitudinal study. *J Clin Med*. 2023;12:7379.
 16. Nerla R, Tarzia P, Sestito A, et al. Effect of bariatric surgery on peripheral flow-mediated dilation and coronary microvascular function. *Nutr Metab Cardiovasc Dis*. 2012;22:626–34.
 17. Tarzia P, Lanza GA, Sestito A, et al. Long-term effects of bariatric surgery on peripheral endothelial function and coronary microvascular function. *Obes Res Clin Pract*. 2017;11:114–7.
 18. Grassi G, Seravalle G, Scopelliti F, et al. Structural and functional alterations of subcutaneous small resistance arteries in severe human obesity. *Obesity*. 2010;18:92–8.
 19. De Ciuceis C, Rossini C, Porteri E, et al. Circulating endothelial progenitor cells, microvascular density and fibrosis in obesity before and after bariatric surgery. *Blood Press*. 2013;22:165–72.
 20. Wiewiora M, Sosada K, Wylezol M, et al. Red Blood Cell aggregation and deformability among patients qualified for bariatric surgery. *Obes Surg*. 2007;17:365–71.
 21. Holowatz LA, Thompson-Torgerson CS, Kenney WL. The cutaneous circulation as a model of generalized microvascular function. *J Appl Physiol*. 2007;105:370–2.
 22. Rossi M, Nannipieri M, Anselmino M, et al. Skin vasodilator function and vasomotion in patients with morbid obesity: effects of gastric bypass surgery. *Obes Surg*. 2011;21:87–94.
 23. Gryglewska B, Głuszewska A, Zarzycki B, et al. Post-occlusive reactive hyperemic response of skin microcirculation among extremely obese patients in the short and long term after bariatric surgery. *Microcirculation*. 2020;27:e12600.
 24. Ministrini S, Fattori C, Ricci MA, et al. Microcirculatory improvement induced by laparoscopic sleeve gastrectomy is related to insulin sensitivity retrieval. *Obes Surg*. 2018;28:3151–8.
 25. Borzi AM, Buscemi C, Corleo D, et al. Endothelial function in obese patients treated with bariatric surgery. *Diabetes Metab Syndr Obes*. 2020;13:247–56.
 26. Saleh MH, Bertolami MC, Assef JE, et al. Improvement of atherosclerotic markers in non-diabetic patients after bariatric surgery. *Obes Surg*. 2012;22:1701–7.
 27. Lupoli R, Di Minno MND, Guidone C, et al. Effects of bariatric surgery on markers of subclinical atherosclerosis and endothelial function: a meta-analysis of literature studies. *Int J Obes*. 2016;40:395–402.
 28. Lind L, Zethelius B, Sundbom M, et al. Vasoreactivity is rapidly improved in obese subjects after gastric bypass surgery. *Int J Obes*. 2009;33:1390–5.
 29. Brethauer SA, Heneghan HM, Eldar S, et al. Early effects of gastric bypass on endothelial function, inflammation, and cardiovascular risk in obese patients. *Surg Endosc*. 2011;25:2650–9.
 30. Flores L, Núñez I, Vidal J, et al. Endothelial function in hypertensive obese patients: 1 year after surgically induced weight loss. *Obes Surg*. 2014;24:1581–4.
 31. Kamshilin AA, Zaytsev VV, Belaventseva AV, et al. Novel method to assess endothelial function via monitoring of perfusion response to local heating by imaging photoplethysmography. *Sensors*. 2022;22:5727.
 32. Podolyan NP, Mizeva IA, Mamontov OV, et al. Imaging photoplethysmography quantifies endothelial dysfunction in patients with risk factors for cardiovascular complications. *Biomed Signal Process Control*. 2023;86:105168.
 33. Ahmad WB, Al Shalabi AG, Kabalan Y. Effect of laparoscopic mini-gastric bypass versus laparoscopic sleeve gastrectomy on hypertension and dyslipidemia in obese type 2 diabetes mellitus patients. *Ann Med Surg*. 2023;85:4334–41.
 34. Quan Y, Huang A, Ye M, et al. Efficacy of laparoscopic mini gastric bypass for obesity and type 2 diabetes mellitus: a systematic review and meta-analysis. *Gastroenterol Res Pract*. 2015;2015:152852.
 35. Kamshilin AA, Krasnikova TV, Volynsky MA, et al. Alterations of blood pulsations parameters in carotid basin due to body position change. *Sci Rep*. 2018;8:13663.
 36. Johnson JM, Minson CT, Kellogg DL Jr. Cutaneous vasodilator and vasoconstrictor mechanisms in temperature regulation. *Compr Physiol*. 2014;4:33–89.
 37. Kamshilin AA, Nippolainen E, Sidorov IS, et al. A new look at the essence of the imaging photoplethysmography. *Sci Rep*. 2015;5:10494.
 38. Kamshilin AA, Mamontov OV. Physiological origin of camera-based PPG imaging. In: Wang W, Wang X, editors. *Contactless Vital Signs Monitoring*. Academic Press; 2022. pp. 27–50. Available from: <https://doi.org/10.1016/B978-0-12-822281-2.00010-X>.
 39. Pérez-Pevida B, Escalada J, Miras AD, et al. Mechanisms underlying type 2 diabetes remission after metabolic surgery. *Front Endocrinol*. 2019;10:641.
 40. Courcoulas AP, Belle SH, Neiberg RH, et al. Three-year outcomes of bariatric surgery vs lifestyle intervention for type 2 diabetes mellitus treatment: a randomized clinical trial. *JAMA Surg*. 2015;150:931–40.
 41. Frühbeck G. Bariatric and metabolic surgery: a shift in eligibility and success criteria. *Nat Rev Endocrinol*. 2015;11:465–77.
 42. Batterham RL, Cummings DE. Mechanisms of diabetes improvement following bariatric/metabolic surgery. *Diabetes Care*. 2016;39:893–901.
 43. Sjöström L, Peltonen M, Jacobson P, et al. Association of bariatric surgery with long-term remission of type 2 diabetes and with microvascular and macrovascular complications. *JAMA*. 2014;311:2297–304.
 44. Coleman KJ, Haneuse S, Johnson E, et al. Long-term microvascular disease outcomes in patients with type 2 diabetes after bariatric surgery: evidence for the legacy effect of surgery. *Diabetes Care*. 2016;39:1400–7.
 45. Bolen SD, Chang H-Y, Weiner JP, et al. Clinical outcomes after bariatric surgery: a five-year matched cohort analysis in seven US states. *Obes Surg*. 2012;22:749–63.
 46. Gokce N, Vita JA, McDonnell M, et al. Effect of medical and surgical weight loss on endothelial vasomotor function in obese patients. *Am J Cardiol*. 2005;95:266–8.
 47. Lynch J, Belgaumkar A. Bariatric surgery is effective and safe in patients over 55: a systematic review and meta-analysis. *Obes Surg*. 2012;22:1507–16.

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