Oedema Drainage and Cardiac Insufficiency – When is there a Contraindication for Compression and Manual Lymphatic Drainage?

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Keywords

Compression therapy, decongestive therapy, lymphatic drainage, decompensated heart failure, volume shift

Summary

Decompensated heart failure is considered a contraindication for compression therapy and lymphatic drainage. Since the treatment is usually prescribed by phlebologists, dermatologists and GPs, and less often by cardiologists, it is important to understand the correlation between compression and heart failure, and to establish the significance of the latter. According to the European Society of Cardiology, heart failure is characterised by a shortness of breath, peripheral oedema, palpitations and reduced cardiac output. Underlying primary diseases include CHD, hypertension, valvular heart disease and post-inflammatory changes. Decompensated heart failure is classified under NYHA stages III and IV in which the symptoms mentioned above appear even at low levels of physical activity or when the body is at rest. The contraindication for using compression therapy with decompensated heart failure is based on the idea that blood volumes in the extremities shift towards the heart causing a volume overload in the pulmonary circulation and possibly resulting in a pulmonary oedema. Scintigraphy and air-plethysmography reveal the displacement of regional blood volumes when medical compression stockings (MCS) are used. This is compensated for in healthy heart patients. Structural cardiac disease has been shown to change the behaviour of myocardial regulation processes. An increased volume in the right atrium produces a local rise in pressure and an increased expression of natriuretic peptides. However, studies have shown that this increase is temporary and is not accompanied by clinically relevant haemodynamic changes. Thus MCSs pose no threat at NYHA stages I and II. Invasive measurements of patients suffering from heart failure NYHA stages III and IV have also identified that haemodynamic changes caused by compression are compensated for after a few minutes and usually only have minor clinical impact. Nevertheless, drainage therapy on patients with decompensated heart failure should be strictly monitored due to its prognostic implications.

Schlüsselwörter

Kompressionstherapie, Entstauungstherapie, Manuelle Lymphdrainage, dekompensierte Herzinsuffizienz, Volumenverlagerung

Zusammenfassung

Die dekompensierte Herzinsuffizienz gilt als Kontraindikation für Kompressionstherapie und Manuelle Lymphdrainage. Da die Verordnung der genannten Heil- und Hilfsmittel in der Regel durch Gefäßmediziner, Dermatologen und Hausärzte erfolgt, weniger jedoch durch Kardiologen, soll der Zusammenhang zwischen Kompression und Herzinsuffizienz untersucht und der Stellenwert der Herzinsuffizienz definiert werden. Entsprechend der Leitlinie der European Society of Cardiology ist eine Herzinsuffizienz gekennzeichnet durch Luftnot, periphere Ödeme, Palpitationen und Leistungsverlust. Ursächliche Grunderkrankungen sind KHK, Hypertonie, Herzklappenvitien und postentzündliche Veränderungen. Als dekompensierte Herzinsuffizienz sind die Krankheitsstadien NYHA III und IV anzusehen, die durch das Auftreten der o.g. Symptome bei bereits geringer körperlicher Belastung bzw. in Ruhe gekennzeichnet sind. Die Gegenanzeige für Kompressionstherapie bei dekompensierter Herzinsuffizienz ergibt sich aus der Überlegung, dass das Blutvolumen der Extremitäten herzwärts verlagert wird und eine Überlastung im kleinen Kreislauf mit Ausprägung eines Lungenödems bewirken kann. Szintigraphische und airplethysmographische Untersuchungen belegen eine Umverteilung regionaler Blutvolumina unter medizinischen Kompressionsstrümpfen (MKS), welche vom herzgesunden Patienten kompensiert wird. Im Falle einer strukturellen Herzerkrankung ist ein verändertes Verhalten des Myokards und der Regulationsvorgänge belegt. Eine erhöhte Volumenbelastung im rechten Vorhof bewirkt einen lokalen Druckanstieg, verbunden mit nachweislich vermehrter Expression natriuretischer Peptide. Untersuchungen belegen jedoch, dass der An-

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stieg passager ist und nicht zwingend von klinisch relevanten hämodynamischen Veränderungen begleitet wird. So stellen MKS bei der Herzinsuffizienz in den Stadien NYHA I und II keine Gefährdung dar. Invasive Messungen an Patienten der Stadien NYHA III und IV belegen ebenfalls, dass hämodynamische Veränderungen durch Kompression nach wenigen Minuten kompensiert werden und in der Regel nur eine geringe klinische Beeinträchtigung zur Folge haben. Dennoch hat eine entstauende Therapie von Patienten mit dekompensierter Herzinsuffizienz wegen der prognostischen Bedeutung grundsätzlich unter engmaschiger klinischer Kontrolle zu erfolgen.

Introduction

According to the international and German guidelines, decompensated heart failure is an absolute contraindication to vascular and lymphatic compression bandages (S2 guideline 2009–2014), medical compression stockings (S1 guideline 2006–2011), and manual lymphatic drainage (S2 guideline 2017) (1–3). Isolated case reports on heart failure triggered by decongestive therapy can be found in the literature (4, 5). The authors of a recent review of 20 international guidelines and consensus papers on the treatment of varicose leg ulcers published in the years between 2009 and 2016 concluded that only pulmonary oedema should be regarded as an absolute contraindication to compression therapy, whereas this view is not fully shared for heart failure in general (6).

As these medical aids and therapies are usually prescribed by specialists in vascular medicine, dermatologists and general practitioners, and less often by cardiologists, we need to look more closely at the association between heart failure and compression therapy.

Chronic heart failure and decompensated heart failure

According to the 2016 European Society of Cardiology (ESC) guideline (7), which the German Society of Cardiology (DGK) also goes along with, heart failure is characterised by shortness of breath, ankle swelling, and general fatigue. These symptoms are accompanied by clinical signs such as raised jugular venous pressure, pulmonary crackles, and peripheral oedema. Structural defects and dysfunctions that lead to reduced cardiac output and/or raised intracardiac pressure on exertion or at rest have been mentioned as possible causes. Heart failure is not only the result of systolic dysfunction but also of diastolic dysfunction, when the ejection fraction is normal. Diseases that can act as triggers include coronary artery disease, hypertension, valve defects, and post-inflammatory changes. Transthoracic echocardiography is the recommended imaging modality, with cardiac magnetic resonance imaging used in cases where the ultrasound imaging quality is poor. Coronary angiography should be performed when chronic ischaemic heart disease is suspected (recommendation category IC).

Depending on the severity of the condition, additional diagnostic laboratory tests (including natriuretic peptide) or right heart catheterisation to determine pressures in the pulmonary circulation may be performed.

The internationally recognised New York Heart Association (NYHA) classification distinguishes four stages of heart failure (8) that are also reflected in the International Classification of Diseases (ICD-10) (▶ Table 1).

Terminological difficulties arise with the term "decompensated heart failure". Although it is used in both the NYHA classification and the ESC guideline, it is not explicitly defined. Taking heart failure to be a chronic disease, decompensation has to be considered as an acute deterioration in cardiac function. The ESC guideline therefore uses the terms "advanced heart failure" and "acute heart failure". These terms correspond to the NYHA stages III and IV. Decompensated heart failure is therefore a condition characterised by increasing dyspnoea, fatigue, and palpitations with mild physical activity or even at rest.

The ESC guideline mentions specific factors that can trigger an acute decompensation of chronic heart failure, such as infection, stress, and surgery. It has to be said that neither compression therapy nor manual lymphatic drainage appear as trigger factors in this list.

NYHA stage	Clinical picture	ICD-10 code
I	No limitation of physical activity	I50.11
II	Slight limitation of physical activity in which ordinary physical activity leads to fatigue, palpitations and dyspnoea; no symptoms at rest	I50.12
III	Marked limitation of physical activity in which less-than-ordinary activity results in fatigue, palpitations, and dyspnoea; no symptoms at rest	I50.13
IV	Inability to carry on any physical activity without discomfort and symptoms of heart failure at rest, with increased discomfort if any physical activity is undertaken	I50.14

Tab. 1 Heart failure – New York Heart Association classification of severity (based on Dolgin 1994 [8]) showing diagnosis code

Compression therapy displaces the blood volume

About 70–85% of the blood volume is to be found in the venous and capillary compartments of the circulation. It can be concluded, therefore, that compression therapy to the limbs leads to the blood being squeezed into the major vessels. Contraindications to compression therapy in decompensated heart failure come from the

consideration that blood mobilised from the skin and muscles of the legs or arms is transported to the heart via the inferior or superior vena cava and results in volume overload. In the worst-case scenario, life-threatening pulmonary oedema can be expected from pulmonary congestion as the result of displacing the peripheral oedema into the major vessels (▶ Fig. 1).

Mostbeck and Partsch investigated the redistribution of regional blood volumes (9) by compressing the legs with pressures of 25 mm Hg and 40 mm Hg using inflatable rubber boots and measuring the changes in blood volume with scintigraphy. Compression reduced the blood volume in the legs by 33.4% and 38.0%, respectively, while the blood volume of the organs in the chest and abdomen, including the liver, increased by 6–7%. Lattimer et al. used air plethysmography to determine a volume displacement of about 40 ml per leg when below-knee class I or II medical compression stockings (18–32 mm Hg) were worn (10). Only a fraction of the blood reaches the right atrium, however, because of the elastic properties of the inferior vena cava and the many visceral veins. In addition, changes in heart rate adjust the cardiac output to compensate for the increased load. Experience from diving medicine shows that healthy hearts tolerate an increase in the preload without any problem, even with massive compression. Immersion at the depth of 40 m customary in sports diving exposes the body to a 100-fold increase in pressure (approximately 3000 mm Hg), although this pressure is exerted not only over the lower leg but over the whole surface of the body (and the core structures).

In the case of structural heart disease, however, altered myocardial function and changes in the regulatory processes are to be expected: these changes have been demonstrated in various studies (Dereppe et al. [11], Bain et al. [12]). An increased volume in the right atrium increases the local pressure, and stress in the walls resulting from overstretching triggers an increase in natriuretic peptide expression (13). The ESC guideline recommends measuring the levels of B-type natriuretic peptide (BNP) and of the by-product NT-proBNP, as biomarkers for the presence of heart failure (▶ Table 2).

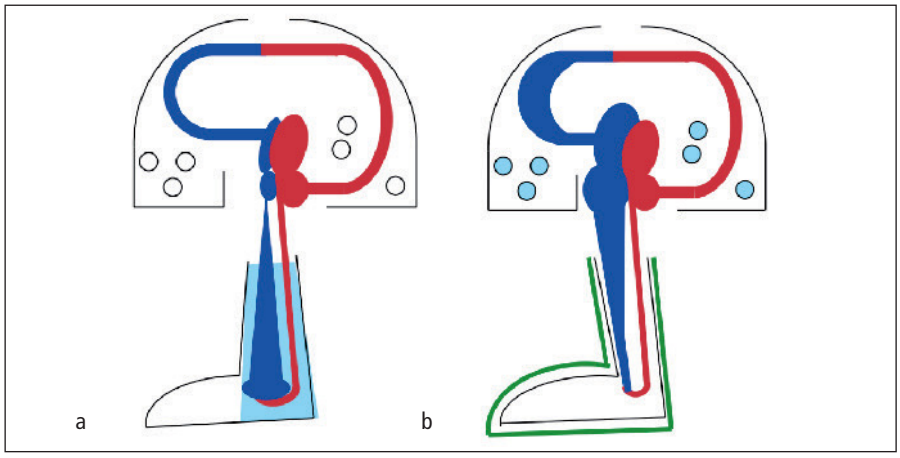

Fig. 1 The "compression homunculus", consisting of leg and heart with the pulmonary circulation, illustrates the displacement of the blood volume under compression. a) The leg veins (blue) store a considerable proportion of the blood. Peripheral oedema is shown in light blue. b) The compression stocking (green) reduces the volume of the oedematous limb. Blood in the leg veins is therefore shifted towards the heart via the inferior vena cava. The load on the right atrium and right ventricle is increased. When cardiac function is impaired, congestion may lead to the development of pulmonary oedema (light blue).

Tab. 2 BNP and NT-proBNP as biomarkers for the acuteness of heart failure. NT-proBNP is also age-dependent (according to Luchner 2017 [21])

	Acute decompensation unlikely	Grey area	Acute decompensation probable
BNP	<100 pg/ml	100–400 pg/ml	>400 pg/ml
NT-proBNP, <50 years	<300 pg/ml	300–450 pg/ml	>450 pg/ml
NT-proBNP, >50 years	<300 pg/ml	300–900 pg/ml	>900 pg/ml
NT-proBNP, >75 years	<300 pg/ml	300–1800 pg/ml	>1800 pg/ml

In a group of 102 subjects with chronic lymphoedema, Todd et al. demonstrated raised BNP levels in seven subjects who also had confirmed cardiac abnormalities (14). In a comparative study, Galm et al. looked at subjects with healthy hearts and subjects with heart disease (NYHA II) wearing class II compression stockings. They showed a significant increase in human atrial natriuretic peptide (hANP) due to the compression stockings only in the group with heart disease, whose members did, however, already have high baseline concentrations. The rise in hANP was transient and not accompanied by haemodynamic changes (heart rate, median blood pressure). The authors concluded that compression therapy with below-knee class II compression stockings did not pose a risk to patients with NYHA stage II heart failure (15). This conclusion was supported by Wilputte et al., who performed right heart catheterisation (Swan-Ganz catheter) to study haemodynamic changes with multi-layer compression bandages on patients with NYHA III and IV heart failure in a coronary care unit. They determined an initial elevation in the right atrial and ventricular pressures after the bandages were applied, but values returned to baseline after just 10 minutes, without any sustained clinical impairment. From their results, however, the authors concluded that compression therapy should not be used in cases of severe oedema because the effects on this patient population could not be calculated (16). Leduc and his team evaluated the effects of local manual lymphatic drain-

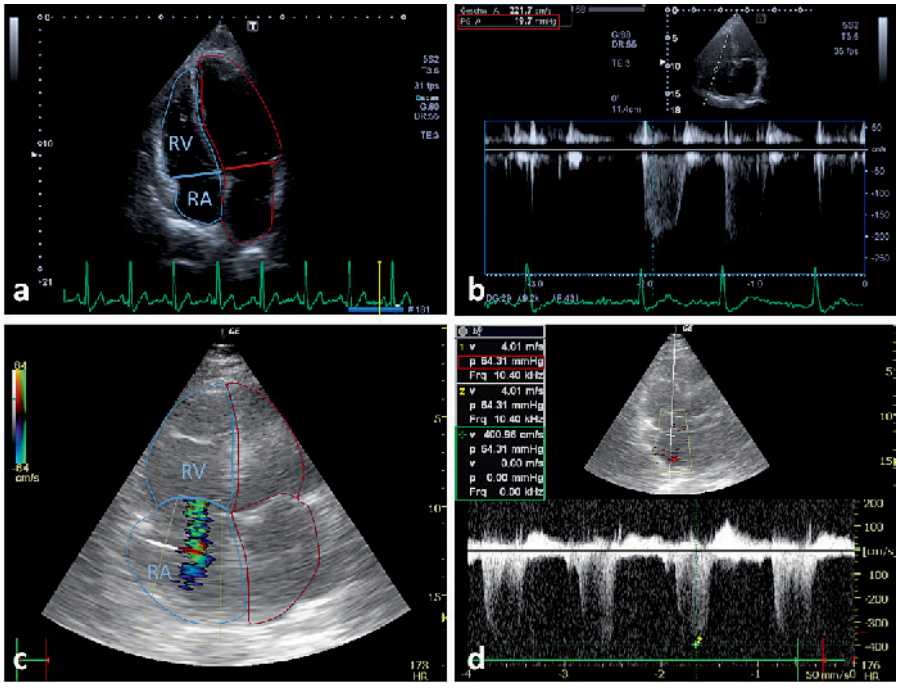

Fig. 2 Heart failure on echocardiography. a) In the normal heart, the right ventricle (RV) and right atrium (RA) (blue) are smaller than the left heart chambers (red) and nestle into them. b) Continuous wave Doppler scanning allows us to determine the pressure in the right ventricle from the gradient across the tricuspid valve. 19.7 mm Hg is within the normal range. c) With a volume overload, the RA and RV (blue) are dilated. The right heart chambers are even larger than the left (red). Dilatation of the right heart also leads to tricuspid regurgitation (green). d) The measured gradient of 64.31 mm Hg across the tricuspid valve is abnormally high. Adding the central venous pressure of approximately 10 mm Hg gives a pulmonary artery pressure of about 75 mm Hg (normal < 30 mm Hg). If left ventricular function is also impaired, the pulmonary circulation becomes congested and pulmonary oedema threatens. The findings correlate with considerable clinical impairment.

age (30–40 mm Hg) in patients with NYHA III and IV heart failure. They also analysed interventional pressure measurements in the pulmonary circulation, as well as cardiac ultrasound measurements and clinical haemodynamic parameters (▶ Fig. 2). They came to the conclusion that, despite the fact that this method resulted in highly significant reductions in the circumference of the extremities, there were no significant changes in the haemodynamic parameters except for the heart rate. They considered that there was no contraindication to local decongestive therapy with MLD (of the legs alone) in patients with NYHA stage III and IV heart failure (17). Gorelik et al. applied compression bandages to the legs of 106 patients with decompensated heart failure to prevent syncope from orthostatic hypotension. Although there was a measured increase in blood pressure, none of the subjects experienced respiratory problems due to pulmonary oedema (18).

To date, there are no studies on the importance of refractory or unstable hypertension. From the studies presented here and the concurrent observations on volume displacement due to immersion (19, 20), however, we have to assume that compression could provoke pulmonary oedema in these cases.

Management in clinical practice

The international classification of the severity of heart failure is based primarily on the history and clinical findings. The differential diagnosis has to consider carefully whether the oedema is of cardiac origin. This applies particularly to marked bilateral oedema, as unilateral oedema is not viewed as a manifestation of systemic cardiogenic disease.

Because of its prognostic significance, the key symptom of decompensated heart failure (NYHA stages III and IV) is dyspnoea with less-than-ordinary physical activity or at rest. Questioning must determine whether the patient is short of breath and under what conditions this can be induced. Shortness of breath when lying flat (orthopnoea) indicates chronic pulmonary congestion as the manifestation of severe or decompensated heart failuvre and also argues against the uncritical use of compression therapy and MLD. The same argument applies to crackles heard on auscultation of the lungs and unstable hypertension with a history of hypertensive pulmonary oedema.

Further cardiological diagnostic investigations (chest X-ray, NT-proBNP, echocardiography) are required if the history and clinical examination do not permit a clear classification of the cardiological situation.

Previously commenced treatment can be continued if the heart failure is stable, as long as the patient does not complain of dyspnoea, chest pain, or palpitations.

If there is any relevant cardiac comorbidity, the patient may need to be admitted to hospital for treatment.

Conclusions

Case reports published to date and the results of the very few available studies allow us to draw the following conclusions:

1. Heart failure per se is not a contraindication to compression therapy and manual lymphatic drainage.
2. In NYHA stage I and NYHA stage II heart failure, treatment with compression bandages and compression stockings, as well as MLD, is generally possible without reservation.
3. In NYHA stages III and IV, decongestive therapy is possible in some circumstances after careful diagnosis and with close clinical and haemodynamic monitoring.

Conflict of interest

The authors declare that they have no conflicts of interest.

Ethical guidelines

No studies on humans or animals were carried out in the preparation of this manuscript.

References

1. Wienert V, Gerlach H, Gallenkemper G et al. Leitlinie Medizinischer Kompressionsstrumpf (MKS). Phlebologie 2006; 35: 315–320.
2. http://www.awmf.org/uploads/tx_szleitlinien/037–005_S2_Phlebologischer_Kompressionsverband_PKV_2009_abgelaufen.pdf (Download: 12/16/2017)
3. http://www.awmf.org/uploads/tx_szleitlinien/058–001l_S2k_Diagnostik_und_Therapie_der_Lymphoedeme_2017–05.pdf (Download: 12/16/2017)
4. Vaassen MM. Manual Lymph Drainage in a Patient with Congestive Heart Failure: A Case Study. Ostomy Wound Manage 2015; 61(10): 38–45.
5. McCardell CS, Berge KH, Ijaz M et al. Acute pulmonary edema associated with placement of waist-high, custom-fit compression stockings. Mayo Clin Proc 1999; 74(5): 478–480.
6. Andriessen A, Apelqvist J, Mosti G et al. Compression therapy for venous leg ulcers: risk factors for adverse events and complications, contraindications – a review of present guidelines. J Eur Acad Dermatol Venereol 2017; 31(9): 1562–1568. doi: 10.1111/jdv.14390. Epub 2017 Jul 31.
7. Ponikowski P, Voors AA, Anker SD et al. 2016 ESC Guidelines for the diagnosis and treatment of acute and chronic heart failure. Eur Heart J (2016) 37;2129–2200. doi: 10.1093/eurheartj/ehw128
8. Dolgin M. The Criteria Committee of the New York Heart Association . Nomenclature and Criteria for Diagnosis of Diseases of the Heart and Great Vessels. 9th ed. Boston, Mass: Little, Brown & Co 1994; 253–256.
9. Mostbeck A, Partsch H. Umverteilung regionaler Blutvolumina durch Dihydroergotamin und Beinkompression. Med Klin 1978; 73: 801–806.
10. Lattimer CR, Kalodiki E, Azzam M, Geroulakos G. Haemodynamic Performance of Low Strength Below Knee Graduated Elastic Compression Stockings in Health, Venous Disease, and Lymphoedema. Eur J Vasc Endovasc Surg 2016; 52: 105–112.
11. Dereppe H, Hoylaerts M, Renard M et al. [Hemodynamic impact of pressotherapy]. J Mal Vasc (1990);15(3):267–9 (Abstract only, FRENCH)
12. Bain RJ, Tan LB, Murray RG et al. Central haemodynamic changes during lower body positive pressure in patients with congestive cardiac failure. Cardiovasc Res. 1989; 23(10): 833–837.
13. Wilkins MR, Gammage MD, Lewis HM et al. The effect of lower-body positivepressure on blood pressure, plasma ANP concentration and sodium and waterexcretion in healthy volunteers and cardiac transplant recipients. Cardiovasc Res 1988; 22: 231–235.
14. Todd J, Austwick T, Berridge D et al. B-type natriuretic peptide in lymphedema. Lymphology 2011; 44(1): 29–34.
15. Galm O, Jansen-Genzel, von Helden J, Wienert V. Plasma human atrial natriuretic peptide under compression therapy in patients with chronic venous insufficiency with or without cardiac insufficiency. Vasa 1996; 25(1): 48–53.
16. Wilputte F, Renard M, Venner JP. Hemodynamic Response to Multilayered Bandages Dressed on a Lower Limb of Patients with Heart Failure. Eur J Lym 2005; Vol XV(45): 1–4.
17. Leduc O, Crasset V, Leleu C et al. Impact of Manual Lymphatic Drainage on Hemodynamic Parameters in Patients with Heart Failure and Lower Limb Edema. Lymphology 2011; 44: 13–20.
18. Gorelik O, Almoznino-Sarafian D, Litvinov V et al. Seating-induced postural hypotension is common in older patients with decompensated heart failure and may be prevented by lower limb compression bandaging. Gerontology. 2009;55(2):138–44. doi: 10.1159/000141920. Epub 2008 Jun 27.
19. Peacher DF, Martina SD, Otteni CE et al. Immersion pulmonary edema and comorbidities: case series and updated review. Med Sci Sports Exerc 2015; 47(6): 1128–1134. doi: 10.1249/MSS.0000000000000524.
20. Gempp E, Demaistre S, Louge P. Hypertension is predictive of recurrent immersion pulmonary edema in scuba divers. Int J Cardiol. 2014 Mar 15;172(2):528–9. doi: 10.1016/j.ijcard.2014.01.021. Epub 2014 Jan 22.
21. Luchner A, von Haehling S, Holubarsch C et al. [Indications and Clinical Implications of the Use of the Cardiac Markers BNP and NT-proBNP]. [Article in German]. Dtsch Med Wochenschr 2017; 142(5): 346–355.