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, uncompensated 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 uncompensated 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 uncompensated heart failure should be strictly monitored due to its prognostic implications.

Zusammenfassung

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.

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 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 on the lower leg but also on 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).

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-

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**Table 2**

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<thead>
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<th>BNP and NT-proBNP as biomarkers for the acuteness of heart failure. NT-proBNP is also age-dependent (according to Luchner 2017 [21])</th>
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<td><strong>Acute decompensation unlikely</strong></td>
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<tr>
<td>BNP</td>
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<tr>
<td>NT-proBNP, &lt;50 years</td>
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<td>NT-proBNP, &gt;50 years</td>
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<td>NT-proBNP, &gt;75 years</td>
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**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).
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 no 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.

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