The influence of adrenaline on echocardiographic parameters of left ventricular function in the horse

Heidrun Gehlen*, Silke Marnette and Peter Stadler
Clinic for Horses, School of Veterinary Medicine Hannover, Bischofsholer Damm 115, D-30173 Hannover, Germany
*Corresponding author: heidrun.gehlen@tiho-hannover.de

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Abstract
The purpose of the present study was to investigate the effect of adrenaline on cardiac function parameters. The infusion of adrenaline for induction of stress, its prospective side-effects as well as its effect on left ventricular function were investigated. A clinical examination and echocardiography were performed on 10 healthy horses before and after infusion of adrenaline. During a period of 6 min, infusion of 1 mg adrenaline kg$^{-1}$ min$^{-1}$ led to a significant increase in mean heart rate, from 36.5 to 55 beats min$^{-1}$. Echocardiography during adrenaline infusion revealed a significant thickening of the interventricular septum (during systole) as well as a significant decrease in left ventricular diameter at the papillary muscle level (during both systole and diastole), and thus an increased contractility. The left ventricular area and the left ventricular volume were significantly decreased during adrenaline infusion, expressing the increased left ventricular contractility (during both systole and diastole). Other echocardiographic parameters of regional left ventricular function changed with only low-grade significance or without any significance at all. During and after infusion of adrenaline, the horses showed sweating, muscle tremor and other symptoms of discomfort. The study revealed that the increases in heart rate and myocardial contractility after infusion of adrenaline were low compared with changes observed during physical performance. Furthermore, severe adverse effects were observed. Taking into account the possible cardiotoxic effects of adrenaline, we do not consider this method appropriate for stress induction, particularly in horses with cardiac disorders.

Keywords: equine; catecholamine; stress echocardiography

Introduction
To perform stress echocardiography, the heart can be stressed actively by physical performance or passively by application of drugs. In humans, active cardiac stress is commonly induced by bicycle exercise and passive cardiac stress is induced pharmacologically by administration of sympathomimetic drugs in order to increase the inotropy and chronotropy of the heart$^1$. Dipyridamole, adenosine and dobutamine are used for pharmacologically induced cardiac stress tests. In the USA, dobutamine is used in most cases$^{2-4}$; in Europe, dipyridamole was used for stress echocardiography in the past, but dobutamine has been preferred in more recent studies$^{5-8}$. Adenosine is rarely used, although there are some published studies in which it is described as a cardiac stressor$^9$. In man, cardiac stress testing is used to elicit changes of myocardial function, myocardial ischaemia or cardiomyopathy that are not evident at rest. Myocardial ischaemia is caused by disproportionate oxygen supply and myocardial oxygen demand. It can be induced by either an increase in oxygen demand (bicycle or treadmill exercise) or a decrease in oxygen and blood supply (administration of dipyridamole and/or adenosine)$^8$.

In horses, echocardiographic examination has been carried out after treadmill exercise and during dobutamine infusion at different dosage rates$^{10-15}$. Dobutamine administered at a low dosage rate results in only a moderate increase in heart rate, whereas at high dosage rates it leads to myocardial toxicity. Stress echocardiography after treadmill exercise is difficult to perform and heart rate often decreases...
rapidly within the first few minutes after exercise. Thus adequate cardiac stress tests, comparable to those used in human patients, do not yet exist in equine medicine.

Adrenaline (epinephrine, suprarenine) and noradrenaline are both naturally occurring sympathomimetic drugs that have an \( \alpha \)- and \( \beta \)-adrenergic effect\textsuperscript{14–17}. In veterinary science, adrenaline is used for the treatment of cardiovascular system dysfunction, either in combination with other drugs or as a single drug for emergency treatment of cardiac failure, e.g. during general anaesthesia (intravenous or intratracheal application)\textsuperscript{18} and during a state of shock (the drug of first choice for treatment of systemic anaphylaxis). It is also used as an additive to local anaesthetics\textsuperscript{14}. In the peripheral blood vessels, \( \beta \)-adrenoreceptors react to a lower blood concentration of adrenaline than do \( \alpha \)-adrenoreceptors. Subsequently, the effect of adrenaline on peripheral resistance depends on the applied dosage. At a low dosage rate (\(< 1 \mu \text{g} \cdot \text{kg}^{-1} \text{ intravenously}\)) adrenaline stimulates mainly \( \beta \)-adrenoreceptors, resulting in vasodilation and a decrease in diastolic blood pressure, and thus a decrease in peripheral resistance. At a high dosage rate (1–3 \( \mu \text{g} \cdot \text{kg}^{-1} \)), adrenaline has mainly an \( \alpha \)-adrenergic effect, causing vasoconstriction and an increase in blood pressure, and thus an increase in peripheral resistance\textsuperscript{16,19}. The heart is stimulated by the effects on \( \beta_1 \)-adrenoreceptors, regardless of whether a low or a high dosage is used\textsuperscript{14}. Cardiac stimulation with adrenaline results in an increase in contractility and heart rate\textsuperscript{20,21}. Adverse stress effects such as muscle tremor and sweating are described after application of adrenaline\textsuperscript{22,23}. Due to its rapid enzymatic decomposition, the effect of adrenaline lasts for only a short period of time\textsuperscript{14}. Although adrenaline is not used in humans for cardiac stress testing, the aim of the present study was to evaluate the stress effects of adrenaline on left ventricular function in the horse. Physiologically, physical performance and stress result in an increase in blood adrenaline concentration\textsuperscript{17}, and adrenaline is useful to specify emergency treatment of cardiovascular dysfunction in anaesthetised horses. Therefore, adrenaline-induced alterations in cardiac function were studied and compared with those observed during physical exercise.

**Materials and methods**

Eight Warmbloods and two Trotters owned by the Clinic for Horses, School of Veterinary Medicine Hannover, Germany, were included in the present study. Basic data of the horses (breed, sex, age, weight and height) are listed in Table 1.

A physical and a specific cardiovascular examination, including electrocardiography and echocardiography, were performed on each horse. Clinical lung examination and arterial blood gas analyses were normal. None of the horses included in the study showed clinical signs of disorders of the cardiovascular system, the respiratory system or the musculoskeletal system.

The baseline echocardiogram as well as the echocardiogram during adrenaline infusion included the M-mode and the B-mode technique (VINGMED 600E; General Electrics, Garching, Germany) with a 2.5 MHz transducer. The maximum depth for B- and M-mode imaging was 30 cm. For subsequent analysis, the echocardiographic sequences were recorded on an S-VHS video recorder (AG 7350; Panasonic, Osaka, Japan).

Simultaneously with the echocardiogram, a bipolar electrocardiogram (ECG; lead II, ‘base–apex ECG’) (VINGMED 600E; General Electrics), which was integrated in the ultrasound machine, was recorded. Heart rate was documented up to 28 min after adrenaline infusion for further monitoring of heart rate and heart rhythm. Mean heart rate was calculated from three consecutive heart cycles.

**Echocardiographic examination**

**B-mode examination**

For the B-mode echocardiograms, standard, long-axis, two-dimensional real-time echocardiography was performed\textsuperscript{24,25}.

The thickness of the interventricular septum and the left ventricular posterior wall, and the left ventricular internal diameter, were all determined from the right parasternal long-axis image of the left ventricle (four-chamber view), at the level of the apex, at papillary muscle level and below the mitral valve (Fig. 1, measurements 1–9). Additionally the left ventricular longitudinal axis was measured (Fig. 1, measurement 10).
Measurements were taken at peak systole (maximal upswing of left ventricular free wall) and end diastole, at rest and during adrenaline infusion.

**M-mode examination**

For M-mode examination, the heart was visualised in the long axis (four-chamber view) on the right side of the chest, and the M-mode cursor was positioned in the B-mode image at different positions (Fig. 2) for assessment of different parameters of regional and global left ventricular function.

For the assessment of regional left ventricular function, the fractional shortening (FS in %) of the left ventricle at the level of the apex, at papillary muscle level and below the mitral valve (Fig. 2, 1–3) was measured using data gained from the one-dimensional, long-axis, M-mode measurements at rest and during adrenaline infusion.

Calculation was performed using the left ventricular end-diastolic diameter (LVDD) and the left ventricular end-systolic diameter (LVSD) in the following formula:

\[
FS \% = \left( \frac{LVDD^2 - LVSD^2}{LVDD^2} \right) \times 100
\]

**Parameters of global left ventricular function**

For the assessment of global left ventricular function, the area, volume and stroke volume of the left ventricle were calculated with the integrated calculation program of the ultrasound machine.

The systolic and diastolic left ventricular areas were determined planimetrically (by rounding the endocardial line) and the left ventricular volume was calculated with the Simpson’s disc summation method, which was done automatically by the Vingmed software. For this purpose, the ventricle is divided into 20 slices perpendicular to the long axis. Their dimensions are first calculated using the thickness and diameter of each slice and, subsequently, they are summed.

The stroke volume (SV, ml per heart beat) was determined using the end-diastolic volume (LVDV) and the end-systolic volume (LVSV) of the left ventricle in the following formula:

\[
SV = LVDV - LVSV.
\]

The ejection fraction (EF in %) was calculated using the end-diastolic volume of the left ventricle (LVDV) and the end-systolic volume of the left ventricle (LVSV) in the following formula:

\[
EF = \left( \frac{LVDV - LVSV}{LVDV} \right) \times 100.
\]

**Infusion of adrenaline**

Intravenous adrenaline (Suprarenin®; Aventis, Bad Soden, Germany) infusion of 1 μg kg⁻¹ min⁻¹ for a period of 6 min was carried out using a perfusor (Perfusor® IV; Braun, Germany). The ECG and heart rate curve were recorded by a telemetric electrocardiograph (telemetric ECG) (ETM 2000 and E1R 1001; Elmed ETM, Augsburg, Germany).

After preparation for echocardiography, an intravenous catheter was applied to the right jugular vein for adrenaline infusion. The perfusor was attached to the catheter using a drip set. Stress echocardiography was performed between 1 and 1.5 min after the beginning of adrenaline infusion.

Heart rate was recorded by the telemetric ECG for 50 min and the curve diagram of the heart rate was printed out using a video printer (Video Graphic...
Printer®; Sony, Japan). A clinical follow-up examination of the horses was performed 3 h and 1 day after adrenaline infusion.

Data analysis
Data were analysed by use of SAS, Version 8.2 (SAS Institute, Cary, NC). For the assessment of normal distribution, the Shapiro–Wilk test was used. All data were included in the descriptive statistics using mean value, standard deviation, minimum value and peak value. The heart rates and echocardiographic parameters before and after infusion of adrenaline were compared by Student’s t-test. The level of significance was set at $P < 0.05$.

Results

Heart rate
The mean heart rate at rest was 36.5 beats min$^{-1}$. During infusion of 1 μg adrenaline kg$^{-1}$ min$^{-1}$ for 6 min, the mean heart rate increased significantly to 55 beats min$^{-1}$ (maximum value: 91 beats min$^{-1}$; minimum value: 49 beats min$^{-1}$; standard deviation: 12 beats min$^{-1}$; Fig. 3). The peak heart rate was reached 2–3 min after adrenaline infusion had stopped. In two horses, the heart rate was significantly higher than in the other horses (horse no. 2 and horse no. 4: 73 and 91 beats min$^{-1}$, respectively; Fig. 3).

Echocardiography during infusion of adrenaline

B-mode measurements
During infusion of adrenaline, the contractility of the left ventricle increased significantly at different myocardial locations. The end-systolic diameter of the interventricular septum at papillary muscle level (during adrenaline infusion: 48 ± 2 mm; at rest: 44 ± 1 mm, Table 2) and the end-systolic left ventricular free wall diameter at papillary muscle level (during adrenaline infusion: 50 ± 6 mm; at rest: 44 ± 4 mm, Table 2) and below the mitral valve (during adrenaline infusion: 35 ± 3 mm; at rest: 32 ± 2 mm, Table 2) increased significantly. The end-systolic and end-diastolic left ventricular diameter at papillary muscle level decreased significantly (during adrenaline infusion: 38 ± 6 mm during systole and 92 ± 8 mm during diastole; at rest: 49 ± 2 mm during systole and 101 ± 2 mm during diastole, Table 2). There were no significant differences between the other B-mode measurements obtained during adrenaline infusion compared with their resting values (Table 2).

M-mode measurements
The fractional shortening of the left ventricle was significantly increased at papillary muscle level during infusion of 1 μg adrenaline kg$^{-1}$ min$^{-1}$ (during adrenaline infusion: 56 ± 4%; at rest: 50 ± 3%, Table 2). There was no significant difference in fractional shortening at the level of the apex (during adrenaline infusion: 67 ± 8%; at rest: 64 ± 10%) and below the mitral valve (during adrenaline infusion: 36 ± 3%; at rest: 39 ± 5%).

Parameters of global left ventricular function
The end-systolic and end-diastolic left ventricular area (during adrenaline infusion: 66 ± 15 cm$^2$ systolic and 141 ± 16 cm$^2$ diastolic; at rest: 76 ± 14 cm$^2$ systolic and 159 ± 16 cm$^2$ diastolic, Table 2) and longitudinal axis (during adrenaline infusion: 121 ± 15 mm systolic and 153 ± 8 mm diastolic; at rest: 128 ± 4 mm systolic and 160 ± 5 mm diastolic, Table 2) were decreased significantly during adrenaline infusion.

The stroke volume (during adrenaline infusion: 709 ± 153 ml; at rest: 838 ± 123 ml) and the end-systolic and end-diastolic left ventricular volume (during adrenaline infusion: 329 ± 101 ml during systole and 1038 ± 181 ml during diastole; at rest: 381 ± 101 ml...
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Table 2 Echocardiographically determined thickness (B-mode, long-axis) of the interventricular septum (O IVS), the inner diameter of the left ventricle (O LV) and the left ventricular free wall (O LVW) at different locations, and the left ventricular longitudinal axis (LV La), the fractional shortening (FS) at different locations, the ejection fraction (EF), stroke volume and end-diastolic and end-systolic left ventricular area and volume, at rest and during infusion of 1 μg adrenaline kg⁻¹ min⁻¹. Locations: at the level of the apex = A, at papillary muscle level = M and below the mitral valve = uMV

<table>
<thead>
<tr>
<th>Parameter</th>
<th>At rest</th>
<th>Adrenaline infusion</th>
<th>Significance</th>
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<td><strong>End-diastolic</strong></td>
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<tr>
<td>O IVS (mm)</td>
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<td>116 ± 8</td>
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<td>O LW A (mm)</td>
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<tr>
<td>LV La (mm)</td>
<td>160 ± 5</td>
<td>153 ± 8</td>
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<tr>
<td>LV volume (ml)</td>
<td>1195 ± 52</td>
<td>1038 ± 181</td>
<td>P &lt; 0.05</td>
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<td>LV area (cm²)</td>
<td>159 ± 16</td>
<td>141 ± 16</td>
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<td>O LW uMV (mm)</td>
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<td>LV La (mm)</td>
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<tr>
<td>LV volume (ml)</td>
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<td>329 ± 101</td>
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<td>LV area (cm²)</td>
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<td>68 ± 15</td>
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<td>FS A (%)</td>
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<td>-</td>
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<tr>
<td>FS PPM (%)</td>
<td>50 ± 3</td>
<td>56 ± 4</td>
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<tr>
<td>FS uMV (%)</td>
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<td>39 ± 5</td>
<td>-</td>
</tr>
<tr>
<td>EF (%)</td>
<td>68 ± 2</td>
<td>67 ± 4</td>
<td>-</td>
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<tr>
<td>Stroke volume (ml)</td>
<td>858 ± 123</td>
<td>709 ± 153</td>
<td>P &lt; 0.05</td>
</tr>
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</table>

during systole and 1195 ± 52 ml during diastole) were significantly decreased during adrenaline infusion (Table 2).

There was no significant difference in the ejection fraction during infusion of adrenaline (67 ± 4%) and at rest (68 ± 2%).

**Behaviour of the horses during adrenaline infusion**

During infusion of adrenaline, all horses showed sweating. Furthermore, muscle tremor (n = 7), head shaking (n = 3) and rapid tail (n = 2) and leg movements (n = 2) were observed.

**Clinical follow-up examination**

During the clinical follow-up examination which was performed 3 h and 1 day after adrenaline infusion, all clinical parameters including heart rate and heart rhythm were normal.

**Discussion**

In human medicine, stress echocardiography including two-dimensional echocardiography has become a reliable method for examining the heart under work load. It has a diverse field of application and is a well established technique for diagnosis and prognosis of left ventricular myocardial dysfunction, vitality diagnostic of the myocardium and coronary artery disease. In horses, a stress echocardiogram is also desirable because it is often difficult or even impossible to determine the clinical significance of a minor degree of cardiovascular dysfunction at rest. Furthermore, some disorders of the cardiovascular system only become apparent during work load.

In horses, physical performance results in a significant increase in blood adrenaline concentration, with the highest concentration being found at maximum work load. There is also a significant correlation between the intensity of work load and the increase in blood adrenaline concentration, as well as between blood lactate concentration and blood adrenaline concentration.

There are some studies reporting cardiovascular examinations in horses including stress echocardiography under active and passive stress induction, but there are no established methods for routine diagnostics. For this reason, we conducted the present study to provide more data about cardiovascular diagnostics, particularly in the Warmblood horse. To induce stress pharmacologically, adrenaline was used in the present study. The positive inotropic and positive chronotropic effect of adrenaline has been documented previously in the horse and adrenaline has also been documented for emergency treatment of cardiac failure in equine medicine. So far, the influence of adrenaline on the cardiovascular system has been investigated in horses, but there are no published studies about stress echocardiography during infusion of adrenaline.

The changes in heart rate during infusion of different dosages of adrenaline are known. Snow used an infusion of 1.1 μg·adrenaline·kg⁻¹·min⁻¹ to investigate the influence on metabolic blood parameters, e.g. blood concentration of glyceral, glucose and lactate as well as haematocrit. He did not observe a significant influence on the heart rate. A similar dosage of adrenaline (1 μg·kg⁻¹·min⁻¹) was used for medical treatment of left dorsal displacement of the large colon. Despite the desired contraction of the spleen, tachycardia of up to 72 beats min⁻¹ was observed. An increase in heart rate of 43% was found after intravenous infusion of 0.2 μg·adrenaline·kg⁻¹·min⁻¹ over a period of 10 s. This significant increase in heart rate may be a consequence of rapid bolus...
injection of adrenaline. The intramuscular application of 10 μg adrenaline kg⁻¹ resulted in an increase in heart rate to 44 beats min⁻¹ in only one of three horses. These studies reveal that a sufficient increase in heart rate for cardiovascular diagnostic purposes does not occur. In contrast to the published data, a significant increase in mean heart rate to 62 beats min⁻¹ was documented after infusion of 1 μg adrenaline kg⁻¹ min⁻¹ in the present study.

However, jumping horses have a peak heart rate of 190 beats min⁻¹ during physical performance. Warmbloods used for dressage have a heart rate of 154 ± 18 beats min⁻¹ during competition and racehorses have a maximum heart rate of 220–240 beats min⁻¹ when racing. Consequently, the heart rate obtained under adrenaline infusion is not equivalent to the heart rate documented during physical performance, and therefore we do not consider it appropriate for stress echocardiography. Furthermore, severe side-effects, such as sweating and muscle tremor, were observed after infusion of adrenaline at the dosage rate used in the present study (1 μg kg⁻¹ min⁻¹); consequently, the use of a higher dosage is not recommended. Intense sweating (β₂-adrenergic stimulation of the apocrine glands) and a remarkable increase in haematocrit were also described by different authors. An intravenous bolus injection of 1 μg adrenaline kg⁻¹ min⁻¹ resulted in cardiac arrhythmia with ventricular premature contractions for a period of 2–3 min after infusion. This arrhythmie effect of adrenaline can not only induce ventricular premature contractions, but also atrial fibrillation and ventricular tachycardia. Furthermore, there are other side-effects described, e.g. an influence on gut motility.

Besides the documentation of heart rate, echocardiography before and during adrenaline infusion was performed in the present study. The left ventricular heart dimensions at rest were within the normal range for healthy horses. Standard parameters for the B-mode and M-mode examination for healthy horses were established in conformity with several authors. In the present study, the B-mode echocardiography at rest differed slightly from the standardised methods of these authors. This is because we performed measurements of the myocardium and the inner diameter of the left ventricle closer to the base (Fig. 1, measurements 3, 6 and 9) of the heart than described by other authors, to make an additional measurement close to the apex of the heart (Fig. 1, measurements 1, 7 and 4). Consequently, a better understanding of the function of the entire left ventricle, including the apex, is obtained.

In humans, the left ventricle reacts physiologically to active or passive stress induction with an increase in myocardial thickness, a decrease in end-diastolic left ventricular volume, an increase in in-flow velocity and a higher velocity in contractility. After infusion of adrenaline, we also observed an increase in myocardial contractility of the left ventricle in the horse. But its intensity was lower than that described after physical performance. Under physical performance, there is a change in haemodynamics in the coronary blood vessels due to shifting of the ratio of systole and diastole. Muscle contraction during the systolic phase reduces the blood supply to the myocardium, and only during the diastolic phase does blood flow into the myocardium of the left ventricle. Work load shortens diastole, and thus the time available for blood flow into the myocardium. It is important to point out that such shifting of the ratio of diastole and systole did not occur during infusion of adrenaline. Thus the adrenaline infusion in our study has only little vasomotoric effect on coronary blood flow, in contrast to exercise.

As well as myocardial thickness and inner left ventricular diameter, further echocardiographic parameters of global and regional left ventricular function were determined. For the assessment of global left ventricular function, the left ventricular volume is particularly valuable. Its determination allows the assessment of even low-grade changes in left heart morphology and minor changes in myocardial kinetics, which might not be evident if only regional parameters of myocardial function (thickness and diameter of the myocardium and of the chambers) are determined. In the present study, most of the regional parameters (heart dimensions) were either not changed at all or the changes were not significant compared with the values obtained at rest. However, there was a significant difference between the parameters of global left ventricular function (left ventricular area, stroke volume and ventricular volume) determined at rest and during adrenaline infusion.

Considering that healthy horses were examined in this study, an even greater diagnostic relevance may be expected in horses with disorders of the cardiovascular system. Regional wall motion abnormalities have been described not only in humans, but also in horses with cardiovascular disease.

We have used Simpson’s disc summation method to determine the left ventricular volume because, with monopolar targeting and irregular outlines of the ventricle, this method is superior to other methods (area/length method, ellipsoid method) in human medicine. The present study shows that adrenaline stress echocardiography is not appropriate for daily routine diagnostics in horses because severe side-effects were observed, despite the low dosage rate of adrenaline used. Furthermore, the use of adrenaline for diagnostic purposes was limited because the heart rate during adrenaline infusion remained relatively low. Yet, an increase in the horse’s work load was documented with echocardiography in this study during infusion of adrenaline.
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References

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