ORIGINAL ARTICLE

Cardiovascular responses to treadmill exercise in Nigerian hypertensives with left ventricular hypertrophy

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Abstract

Background: Left ventricular hypertrophy (LVH) is an independent risk factor for adverse cardiac outcomes in hypertensive patients.

Objective: This study is designed to assess the cardiovascular responses to treadmill exercise among Nigerian hypertensives with echocardiographically proven LVH.

Materials and Methods: Fifty hypertensive patients with LVH (27 males and 23 females) between 30 and 65 years of age were studied in Nigeria. 50 hypertensive patients without LVH and 50 normal subjects who were age and sex matched served as controls. All patients and control subjects underwent M-mode, 2-D and Doppler ECHO-studies and the Bruce protocol treadmill exercise test.

Results: The study showed that the estimated maximal oxygen consumption (MVO2) in MET reduced progressively from 8.39 ± 1.26 (normotensive control) to 7.62 ± 1.33 (hypertensive without LVH), 6.27 ± 0.99 (hypertensive with LVH) (*P*<0.0001ANOVA). The duration of exercise (s) was also reduced in that order from 455.4 ± 79.1 to 411.6 ±8 2.3, 315.8 ± 75.6 respectively (*P*<0.0001). The systolic blood pressure (SBP) and pressure rate product (PRP) during maximal exercise were also increased in hypertensives with LVH and hypertensive without LVH when compared to normotensive controls. The hypertensives with LVH and hypertensives without LVH also showed significant limitation to heart rate increase with exercise compared to normotensive controls (*P*<0.003).

Conclusion: This study demonstrated significant impairment of exercise capacity in hypertensives with or without LVH compared to normotensive subjects. Both earlier recognition and improved understanding of LVH may lead to more effective therapeutic strategies for this cardiovascular risk factor.

Key words: Cardiovascular responses, hypertensives, left ventricular hypertrophy, treadmill exercise

Introduction

Left ventricular hypertrophy (LVH) has been shown to be a significant risk factor for adverse cardiac outcome both in the general population and in patients with hypertension, even without underlying heart disease.[1-3]

LVH was initially thought to be a beneficial compensatory mechanism for maintaining normal wall stress in LV pressure and volume overload, epidemiological studies using electrocardiography (ECG) and recently echocardiography (ECHO) have elucidated the profound independent risk of LVH for congestive heart failure (CHF), coronary events, life threatening arrhythmias and cardiac mortality.

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Exercise testing has become standardized for the diagnostic and functional evaluation of patients with cardiovascular disease.

Some studies have shown that impaired exercise performance in Nigerian hypertensives occur with onset of LVH.[4-6]

Systolic BP at the end of maximal exercise has been more closely correlated than resting systolic blood pressure with LV mass.[7] It is also possible that the exaggerated BP response to exercise in normotensives with parental hypertension reflects the presence of LVH.

It has been suggested that the volta ge LVH on the ECG may be an early marker of impaired exercise capacity.[8]

While impaired exercise capacity has been demonstrated in Nigerian hypertensive patients with ECG-LVH and/or cardiomegaly on chest X-ray there is a paucity of data relating ECHO LVH and diastolic function to exercise capacity.

The purpose of this study therefore is to evaluate the association of LVH and dysfunction with abnormal exercise capacity among Nigerian hypertensives.

Materials and Methods

50 hypertensive patients with ECHO proven LVH attending the cardiology unit of OAUTHC, Ile-Ife who are between the ages of 30 – 65 years were studied after informed consent and ethical clearance have been obtained.

50 hypertensive patients without ECHO proven LVH and 50 normal subjects who were age and sex matched served as control groups. The control subjects were volunteers recruited from among hospital staff, medical and nursing students, and members of the local community.

For this study, the exclusion criteria for participants included the following: –
- Patients with diabetes mellitus
- Patients with chronic kidney disease with serum creatinine <176μmol/l in the last 3 months and free of structural kidney damage.[9,10]
- Patients with Chronic Obstructive Airway Disease
- Use of β - blockers or digoxin, atrial fibrillation, bundle branch block, and baseline ST – segment abnormalities.
- Patients with congestive heart failure and valvular heart disease.
- Severely obese patients.
- Patients with severe arthritic pains and other exercise limiting musculoskeletal diseases.

Procedure
All patients were examined clinically to evaluate their body mass index and cardiovascular status at rest and identify individuals with contraindications to exercise testing. Subjects were considered to be hypertensive if they had a resting SBP of $\geq$140mmHg or a resting DBP of $\geq$90mmHg or if they were on antihypertensive medication, excluding drugs such as methyl dopa and dihydropyridine calcium channel blockers which could influence heart rate.

All patients and control subjects underwent Chest X-ray, 12-lead ECG, M-mode, 2-D and Doppler ECHO-studies and the Bruce protocol treadmill exercise test. Complete blood count, serum chemistry screening and blood sugar were also performed

Echocardiographic study
Echocardiography was performed with a Sonoline G60 S ultrasound system with 4.2-6.2 MHz transducer.

Images of the heart were obtained in multiple cross-sectional planes with the use of standard transducer position with the subject in the left lateral decubitus position.

Measurements were in accordance with the recommendations of the American Society of Echocardiography.[11]

Except for LV systolic dimension (LVSD) that was taken at end systole, other wall thickness and cavity measurements, were done at end diastole. End systole was recognized as the nadir of the septal motion and/or the peak of the LV posterior wall motion.

In each subject, three (3) consecutive cardiac cycles were obtained for analysis.

Doppler echocardiography
Each subject was examined in the left lateral decubitus position with the transducer at or slightly to the left of the apical impulse to obtain apical four-chamber view of the heart, to visualize the LV cavity and obtain the maximal excursion of the mitral valve leaflets.

When the transducer is slightly angulated anteriorly from the standard 4-chamber plane, a 5-chamber view was obtained which included the aortic valve, aortic root and the LV outflow tract. The Doppler mode was then selected on the scanner machine and simultaneously 2D and pulsed Doppler examination were performed. The sample volume was placed to determine the left and right ventricular inflow and outflow. The Doppler sample volume was positioned in the inflow area of the LV (and RV) between the tips of the mitral (and tricuspid) valves leaflets, as they open during diastole. The area was interrogated to obtain wave forms with the highest peaks of diastolic flow velocity and the optimal signal to noise ratio: these are usually identified within the LV cavity about 1cm below the mitral annulus

near the tips of the mitral leaflets. Tracing of at least three cardiac cycles were analyzed and averaged.

LV Diastolic function

The following Doppler indices of LV diastolic filling were obtained.[12,13]

- Peak early transmitral filling velocity (E)
- Peak late transmitral filling velocity (A)
- The ratio of early and late transmitral filling velocities (E/A)
- Deceleration time of the early transmitral filling velocity (Dct; time of peak early transmitral velocity to baseline).
- Isovolumic relaxation time (IVRT).

In each subject 3 consecutive cardiac cycles were chosen for analysis.

LV systolic function

The following measurements were used to assess the LV systolic function.

$$\text{Fractional shortening (FS)} = \frac{\text{LVDD - LVSD}}{\text{LVDD}} \times 100$$

$$\text{Ejection fraction (EF) in \%} = \frac{\text{LVEDV} - \text{LVESV}}{\text{LVEDV}} \times 100\%$$

LV mass (LVM) was estimated according to the American Society of Echocardiography convention (ASE)[14] whereby:-
$LVM = 0.80 (1.04 (LVIDD + PWT + IVST)^3 - (LVIDD)^3) + 0.6g$.
Where IVST = Interventricular Septal wall thickness
LVPWT = LV posterior wall thickness.
LV mass index was derived by dividing LVM by body height.
LVM Index = LVM/Height.
LVH was defined as a LV mass index greater than 143g/m for men and 102g/m for women[15]

Exercise testing

A standard multistage maximal exercise test was conducted on a motorized treadmill (Schiller MTM 1500) according to Bruce protocol.[16] The exercise testing began with subjects walking slowly for 3 min at 1.7m/h at a 10% grade; speed and grade then increased every 3 min until exhaustion.

Each subject was instructed to exercise to his maximum performance but was made to understand that he could stop voluntarily by indicating if he had any symptoms such as chest pain, muscle cramps, intolerable fatigue, low back ache, dizziness or breathlessness etc.

The electrocardiogram, heart rate and blood pressure were recorded during the last minute of each stage of exercise.

The electrocardiogram was continuously monitored on the Schiller CS-200 12 – channel electrocardiographic monitor. Leads II, aV$_F$ ad V5 were continuously displayed on the monitor throughout exercise. The subjects' electrocardiogram, blood pressure and heart rate were monitored and recorded during the post exercise period every 2 min for an average of 10 min.

The laboratory and equipment conformed to the specifications for exercise testing equipments[16]. The exercise laboratory was provided with an air-conditioner and room temperature maintained between 20°C and 25°C.

A defibrillator, cardio/Pak 936/S (Mennen Medical Inc, New York, USA) equipped with portable ECG monitor, and emergency care drugs were provided.

The following parameters were measured
- Duration of exercise (DOE) in seconds and from this maximal oxygen uptake was estimated
- Maximal (Exercise) heart rate (MHR) in beats per minutes.
- Metabolic equivalent (MET): As an index of exercise capacity estimated metabolic equivalent of workload was calculated from the exercise time as:-
- MET = 1.11 + 0.016 ¥.[16]
- Where ¥ is the exercise time in second.
- Rate Pressure product (or double product) is a measure of myocardial oxygen uptake during clinical exercise testing. It is estimated by the product of heart rate and systolic blood pressure.

Statistical analysis

The results were expressed as mean ± standard deviation. Statistical analysis was by analysis of variance and further comparisons were carried out by t-test. The relationship between variables was determined by simple and multiple linear regressions. Pearson correlation coefficient was used to determine correlation among variables. Ninety nine percent confidence interval was quoted where appropriate and null hypotheses was rejected at $P<0.05$.

Data was analyzed on a Pentium M computer with an SPSS 13.0 version statistical package.

Results

General

Hypertensives with LVH were well matched for age, sex and body mass index with hypertensives without LVH and normotensive controls [Table 1].

Serum creatinine values were significantly different, among the groups ($P = 0.020$) although they were within normal limits.

The resting systolic and diastolic blood pressures were significantly higher in hypertensive with LVH than normotensive controls; 143.6 (± 8.2) mmHg vs 121.4 (± 7.2) mmHg (*P*< 0.001) and 87.2 (± 6.4) mmHg vs 76.4 (± 4.8)mmHg (*P*< 0.001) respectively.

However, the SBP and DBP values at rest were higher among the hypertensives with LVH than the hypertensives without LVH but only the SBP 143.6 (± 8.2) mmHg vs 139.4 (± 10.3) mmHg was statistically significant (*P*< 0.05).

In comparison with normotensive controls both the systolic and diastolic BP of hypertensives without LVH were significantly higher (*P*<0.001)

Exercise testing

Exercise duration was significantly lower in hypertensives with LVH compared to both the hypertensives without LVH and normotensive controls 315.8(±75.6) sec vs 411.6(±82.3) sec (*P*<0.001) for hypertensives without LVH and 315.8(± 75.6) s vs 455.4 (± 79.1) sec (*P*<0.001) for normotensive subjects. [Table 2]

The exercise duration in normal subjects was also significantly higher than the hypertensive without LVH group *P*< 0.001.

The normotensive subjects have higher metabolic equivalent (MET) than the hypertensive with LVH patients 8.39 (± 1.26) mets vs 6.27(± 0.99) mets (*P*< 0.001).

Hypertensive without LVH patients also have significantly higher MET than the hypertensive with LVH patients 7.62(± 1.33) mets vs 6.27 (± 0.99) mets (*P*< 0.001).

Maximal heart rate was significantly lower in hypertensives with LVH when compared to both the hypertensives without LVH and normotensive controls 175.2(± 19.4) beats/min vs 179.9 (± 7.8) beats/min *P*< 0.05 for hypertensives without LVH and 175.2 (± 19.4) beats/min vs 184.9(± 12.6) beats/min *P*< 0.05 for normotensive controls.

The SBP at maximum exercise was significantly higher among hypertensives with LVH when compared with normotensive controls 210.3(± 17.9) mmHg vs 184.2 (± 10.2)mmHg *P*< 0.001.

Table 1: Clinical and demographic details of patients and normotensive subjects

	Normotensive controls (*n* = 50)	Hypertensive without LVH (*n* = 50)	Hypertensive with LVH (*n* = 50)	P value (anova)
Age (years)	46.6 ± 8.45	47.2 ± 7.7	48.3 ± 5.8	0.177 (NS)
Sex (M/F)	25/25	27/23	24/26	0.142 (NS)
BMI (kg/m²)	24.3 ± 2.0	24.5 ± 2.5	25.4 ± 1.9	0.160 (NS)
CTR	0.47 ± 0.01	0.48 ± 0.02	0.48 ± 0.03	0.160 (NS)
SBP at rest (mmHg)	121.4 ± 7.2	139.4 ± 10.3	143.6 ± 8.2	0.000 (S)
DBP at rest (mmHg)	76.4 ± 4.8	86.4 ± 5.2	87.2 ± 6.4	0.000 (S)
HR at rest (beats/min)	83.8 ± 6.6	85.5 ± 8.0	88.6 ± 9.4	0.014 (S)
Serum creatinine (um/l)	69.1 ± 29.1	96.3 ± 23.4	102.7 ± 26.5	0.020 (S)
Serum cholesterol (mmol/l)	4.63 ± 0.32	4.77 ± 0.46	4.81 ± 0.29	0.129 (NS)

S = Statistically significant; NS = Not statistically significant; CTR = Cardiothoracic ratio; BMI = Body mass index; SBP = Systolic blood pressure; DBP = Diastolic blood pressure; HR = Heart rate

Table 2: Treadmill exercise response in Patients and Normotensive subjects

	Normotensive controls (*n* = 50)	Hypertensive without LVH (*n* = 50)	Hypertensive with LVH (*n* = 50)	P value (anova)
SBP at rest (mmHg)	121.4 ± 7.2	139.4 ± 10.3	143.6 ± 8.2	0.001 (S)
SBP max (mmHg)	184.2 ± 10.2	204.6 ± 23.1	210.3 ± 17	0.001 (S)
DBP at rest (mmHg)	76.4 ± 4.8	86.4 ± 5.25	87.2 ± 6.4	0.001 (S)
DBP max (mmHg)	89.2 ± 6.9	99.4 ± 6.51	100.6 ± 7.1	0.001 (S)
HR at rest (beats/m)	83.8 ± 6.6	85.5 ± 8.0	88.6 ± 9.40	0.014 (S)
HR max (beats/m)	184.9 ± 12.6	179.9 ± 7.8	175.2 ± 19	0.003 (S)
RPP at rest (mmHg b/m)	10218 ± 1213	11918 ± 1480	12722 ± 1833	0.001 (S)
RPP max (mmHg b/m)	34058 ± 2314	36807 ± 3894	36844 ± 4706	0.001 (S)
Stage	3.5 ± 0.3	3.16 ± 0.48	2.72 ± 0.34	0.001 (S)
DOE (s)	455.4 ± 79.1	411.6 ± 82.3	315.8 ± 75.6	0.001 (S)
MET	8.39 ± 1.26	7.62 ± 1.33	6.27 ± 0.99	0.001 (S)

S = Statistically significant; SBP = Systolic blood pressure; DBP = Diastolic blood pressure; HR = Heart rate; RPP = Rate pressure product; DOE = Duration of exercise; MET = Metabolic equivalent

The rate pressure product which is a measure of myocardial oxygen demand was significantly higher among hypertensives with LVH when compared with both hypertensives and nomotensive controls 36844(± 4706)mmHg beats/min vs 36807(±3894)mmHg beats/min for the hypertensive controls (P< 0.05) and 36844(± 4706)mmHg beats/min vs 34058(± 2314)mmHg beats/min for the normotensives P< 0.001.

Echocardiographic parameters
There are statistically significant difference in the value of left ventricular mass, left ventricular mass index, and left ventricular diastolic and systolic dimension between the hypertensives with LVH , hypertensive and normotensive controls. [Table 3]

Hypertensives with LVH have a LV mass of 240.6 ± 50.4g while hypertensive controls have a LV mass of 154.5 ± 40.0g (P< 0.001) and normotensives have a LV mass of 152.8 ± 37.6g (P< 0.001).

There is no statistically significant difference between the LV mass of hypertensives without LVH and normotensive controls (P = 0.82). The left ventricular diastolic dimension for hypertensives with LVH was 4.70 (± 0.40)cm vs 4.58 ± 0.36cm for hypertensive patients (P<0.001) and 4.19 ± 0.54cm for normotensive controls (P<0.001).

Univariate analysis of Echocardiographic parameters that correlate with metabolic equivalent
There is a significant correlation between the fractional shortening, ejection fraction and MET in hypertensives with LVH r = -0.38 and – 0.34 respectively; P = 0.006 and 0.017 respectively. [Table 4]

There is also a significant correlation between their IVRT and metabolic equivalent r = -0.42 (P = 0.002).

There is a significant positive correlation between mitral E velocity, mitral E/A ratio and MET r = 0.39 and 0.30 respectively; P = 0.004 and 0.03 respectively.

Multiple regression analysis of ECHO parameters that correlate with Metabolic equivalent
Multivariate analysis was performed with all univariate variables (P < 0.05). The result showed that all these variables have 17% contribution to the prediction of metabolic equivalent. The multivariate independent predictors for a higher metabolic equivalent were a lower IVRT and lower LVMI. [Table 5]

Discussion

This study demonstrated significant impairment of exercise capacity in hypertensives with or without LVH compared to normotensive subjects. This is in keeping with previous studies.[4,5,17] Balogun *et al.*[4] studied the influence of hypertension on the exercise performance in normotensives, hypertensives and patients with hypertensive heart failure and found a progressive reduction of the systolic rise in blood pressure during exercise, exercise induced tachycardia, exercise time, and maximal oxygen uptake.

Since the subjects were well matched for age, sex and habitual physical activity, the differences observed may be attributable to the effect of the disease on the functional capacity of the patients. Maximal heart rate was significantly lower in hypertensives with LVH than hypertensive and normotensive controls.

Reduced maximal heart rate values in hypertensives compared with nomotensives have been observed in other studies with increasing severity of hypertension[17,18]. Previous groups have reported abnormal chronotropic responses in healthy subjects with increased LV mass and increased LV cavity size[19]. This might be due to the neurohormonal abnormalities associated with left ventricular hypertrophy and dilatation, most especially reduced sympathetic reserve. The reduction in maximal heart rate in this study may in part contribute to the impairment of exercise capacity by limiting cardiac output and thus exercise capacity.

Table 3: M – Mode / 2 D – Echocardiographic Data of Patients and Normotensive subjects

	Normotensive controls (*n* = 50)	Hypertensive without LVH (*n* = 50)	Hypertensives with LVH (*n* = 50)	*P* value (anova)
LVDd (cm)	4.19 ± 0.54	4.58 ± 0.36	4.70 ± 0.40	0.001 (S)
LVDs (cm)	2.6 ± 0.53	2.94 ± 0.41	3.04 ± 0.45	0.001 (S)
FS (%)	37.7 ± 6.88	37.1 ± 5.77	35.3 ± 7.5	0.194 (NS)
EF (%)	74.0 ± 8.6	74.3 ± 8.0	71.7 ± 10.4	0.348 (NS)
IVST (cm)	0.96 ± 0.14	1.11 ± 0.2	1.35 ± 0.18	0.001 (S)
PWT (cm)	0.94 ± 0.15	1.02 ± 0.18	1.22 ± 0.16	0.001 (S)
LV Mass (g)	152.8 ± 37.6	154.5 ± 40.0	240.6 ± 50.4	0.001 (S)
LV Mass index (g/m)	91.5 ± 21.0	92.1 ± 23.1	145.8 ± 28.3	0.001 (S)

S = Statistically significant; NS = Not statistically significant; LVDd = Left ventricular dimension in diastole; LVDs = Left ventricular dimension in systole; FS = Fractional shortening; EF = Ejection fraction; IVST = Interventricular septal thickness; PWT = Posterior wall thickness; RVD = Right ventricular dimension; AOD = Aortic root dimension; ACS = Aortic cusp separation; LAD = Left atrial dimension; LV = Left ventricular

Table 4: Univariate analysis of ECHO parameters that correlate with metabolic equivalent

	Correlation coefficient	P value
IVRT vs Met	-0.42	0.002 (S)
LVDs vs Met	0.41	0.003 (S)
Mitral E velocity	0.39	0.004 (S)
FS vs Met	-0.38	0.006 (S)
EF vs Met	-0.34	0.017 (S)
Mitral E/A ratio vs Met	0.30	0.033 (S)
LVDd vs Met	0.15	0.272 (NS)
LVM index vs Met	0.05	0.691 (NS)

S = Statistically significant; NS = Not statistically significant;
MET = Metabolic equivalent

Table 5 : Multiple regression analysis of ECHO parameters that correlate with Metabolic equivalent

	Standardized regression coefficient	P value
(Constant)		0.159
IVRT	−0.336	0.014 (S)
LVDs	0.359	0.123 (NS)
FS	−0.661	0.396 (NS)
EF	0.532	0.483 (NS)
Mitral E velocity	0.342	0.044 (S)
Mitral E/A ratio	−0.170	0.340 (NS)

S = Statistically significant; NS = Not statistically significant

The blood pressure response to exercise was significantly higher in hypertensives with LVH than hypertensive and normotensive controls in agreement with other studies.[20]

The peak exercise diastolic blood pressure compared with rest values, was expectedly reduced in normal controls, a finding consistent with earlier studies.[17] This tends to support the observation that hypertensives maintain a higher total peripheral resistance at all levels of exercise as shown from invasive measurement[21] than normal subjects.

The parameters of systolic function e.g. fractional shortening, ejection fraction were comparable among hypertensives with LVH and normotensive controls. This is in agreement with previous studies.[6,22] Ajayi and Akinwusi[6] had found comparable left ventricular contractility indices among normotensive and mildly hypertensive Nigerians. Similarly Lawal *et al.*[22] had previously demonstrated that the ejection fraction, mean velocity of circumferential shortening and fractional shortening in mild hypertensive Nigerians are similar to those of the normotensives.

This study showed that ejection fraction, fractional shortening, isovolumic relaxation time and Doppler transmitral flow velocity pattern correlate with metabolic equivalent in hypertensives with LVH. These results are in agreement with that of Ajayi *et al.*[6] who reported

correlation between systolic parameters and indices of exercise capacity among heart failure patients during treadmill exercise testing.

However, several other studies have shown that measures of left ventricular systolic function are poor predictors of exercise capacity.[23] Lewis *et al.*[24] reported that exercise capacity after myocardial infarction did not correlate with left ventricular systolic function but did with diastolic function. In their study exercise capacity was inversely related to transmitral A velocity and directly to the E/A.

Echocardiographic Doppler indices of left ventricular diastolic function have been reported to be related to exercise capacity in patients with LV systolic function.[25]

In this study, exercise capacity was directly related to the mitral E/A ratio. Since the introduction of the Doppler transmitral flow pattern analysis, non-invasive routine assessment of left ventricular diastolic function has become available.[26] Some workers have reported on the association of left ventricular diastolic function and exercise capacity in normal individuals.[27,28] Vanoverschelde *et al.*[27] studied 66 healthy individuals and found that the mitral E/A ratio was the strongest independent predictor of exercise capacity. Genovesi-Ebert *et al.*[28] studied 20 healthy individuals and 34 patients with hypertension and showed that Echocardiographic Doppler indices of left ventricular filling were associated with exercise capacity.

Several previous authors have reported some possible reasons for the impaired exercise capacity associated with diastolic dysfunction.

Coucolo *et al.*[29] studied patients with essential hypertension and LVH and reported that an abnormal LVEF response during exercise in patients with impaired early diastolic filling was related to their decreased exercise capacity. They found a significant correlation between left ventricular mass and diastolic indices in patients with hypertension. However, Vanoverschelde *et al.*[27] and this study showed no significant correlation between LV mass and exercise capacity in hypertensives and normotensive controls.

Thus, it is supposed that the presence of diastolic dysfunction with or without increased left ventricular mass may limit LV stroke volume to increase despite the elevated left ventricular filling pressure during exercise. In addition, compensatory tachycardia to increase cardiac output would cause a further deterioration in left ventricular filling.

Conclusion

The results demonstrated significant impairment of exercise

capacity in hypertensives with or without LVH compared to normotensive subjects. The hypertensive with LVH patients showed worse cardiovascular responses to exercise when compared to hypertensives without LVH.

Finally the study showed that ejection fraction, fractional shortening and Doppler transmitral flow velocity patterns correlate with treadmill exercise capacity in hypertensives with LVH.

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