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Baseline Quality of Life and Risk of Stroke in the ALLHAT study

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Abstract

Background and Purpose—The visual analogue scale (VAS) is a self-reported, validated tool to measure quality of life (QoL). Our purpose was to determine whether baseline QoL predicted strokes in the ALLHAT study and evaluate determinants of post-stroke change in QoL. In the ALLHAT study, among the 33,357 patients randomized to treatment arms, 1,525 suffered strokes; 1,202 (79%) strokes were non-fatal. This study cohort includes 32,318 (97%) subjects who completed the baseline VAS QoL estimate.

Methods—QoL was measured on a VAS and adjusted using a Torrance transformation (TQoL). Kaplan-Meier curves and adjusted proportional hazards analyses were used to estimate the effect of TQoL on the risk of stroke, on a continuous scale (0–1) and by quartiles (≤0.81, >0.81 ≤0.89, >0.89≤0.95, >0.95). We analyzed the change from baseline to first post-stroke TQoL using adjusted linear regression.

Results—After adjusting for multiple stroke risk factors, the hazard ratio for stroke events for baseline TQoL was 0.93 (95% CI 0.89–0.98) per 0.1 unit increase. The lowest baseline TQoL quartile had a 20% increased stroke risk (HR =1.20 [CI 1.00–1.44]) compared to the reference

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highest quartile TQoL. Post-stroke TQoL change was significant within all treatment groups (p≤.001). Multivariate regression analysis revealed that baseline TQoL was the strongest predictor of post-stroke TQoL with similar results for the untransformed QoL.

Conclusions—The lowest baseline TQoL quartile had a 20% higher stroke risk than the highest quartile. Baseline TQoL was the only factor that predicted post-stroke change in TQoL.

Clinical Trials Registration—https://clinicaltrials.gov/ct2/show/NCT00000542?term=allhat&rank=3 Unique Identifier: NCT00000542

Keywords

Quality of Life; Stroke; Cerebrovascular disease; Risk Factors; Cardiovascular disease; Neurobehavior

Subject Codes

Lifestyle; Risk Factors; Cardiovascular disease; Quality and Outcomes; Cerebrovascular Disease/Stroke; Cognitive impairment

Introduction

In the United States, approximately 795,000 people experience new or recurrent stroke annually[1,2]. Quality of life (QoL) measures are frequently utilized tools for outcomes analysis post-stroke, but may also serve as an important independent predictor of major diseases. Health-related QoL has been reported to independently predict mortality following Coronary Artery Bypass Grafting (CABG)[3], Myocardial Infarction (MI)[4,5], heart failure[6], Diabetes Mellitus[78], and Hemodialysis [9]. Egido et al found that psychosocial stress predicted stroke in a small case-control study, implying that QoL is a significant causative role[1011]. Socioeconomic factors influencing QoL such as unemployment or multiple job losses, may be significant risk factors for acute MI and stroke[121314].

Since ample evidence suggests that strokes impact post-stroke QoL [1516], we seek to explore how QoL prior to stroke-events may impact stroke risk and outcomes. Despite major QoL burdens of stroke [17], there is no consensus stroke-specific QoL measurement tool in wide usage. The National Institute of Neurological Disorders and Stroke Common Data Elements (NINDS CDE)[18] and European Stroke Organization Outcomes Working Group[19] recommend standardized indices to monitor outcomes within clinical trials. The Barthel Index (BI) and the modified Rankin scale (mRS), commonly utilized stroke outcome scales, quantify activities of daily living (ADL) or the extent of functional disabilities, respectively. However, they each fail to assess the broad multi-dimensional factors contributing to QoL. Ali et al demonstrated correlation between mRS, BI, and QoL (generic and disease specific); self-reported QoL had a significantly stronger association with mRS while proxy QoL had stronger association with BI[2021,22].

The simple visual analogue scale (VAS), an easily used instrument to measure self-reported QoL, has been validated with excellent reliability in assessing global QoL, when compared to multi-item questionnaire tool [23], and serves as an appropriate tool to measure QoL in the

setting of stroke [242526]. To date, there has been no large-scale prospective studies analyzing the relationship between QoL and stroke[27]. We report relationship of baseline VAS QoL in Antihypertensive and Lipid Lowering Treatment to Prevent Heart Attack Trial (ALLHAT) with subsequent stroke risk and changes in QoL after stroke.

Methods

All clinical sites had local ethics board approvals. Written informed consent was obtained from all subjects or their legally authorized representatives.

ALLHAT Design

Rationale and design of ALLHAT, sponsored by the National Heart, Lung, and Blood Institute have been previously published[28]. ALLHAT was a randomized, double-blinded trial in 42,418 high-risk hypertensive patients. ALLHAT's main eligibility criteria were: 1) age ≥55 years 2) hypertension and 3) one or more additional risk factors for heart attack. Mean duration of treatment and follow-up was 4.9 years. Patients were assigned to 4 treatment groups: amlodipine, lisinopril, chlorthalidone, and doxazosin. Doxazosin arm was terminated early due to a significantly higher rate of cardiovascular disease and was not included in this analysis.

Outcomes

Primary endpoint of ALLHAT was the composite of fatal CHD and nonfatal MI. Four major protocol-defined pre-hypothesized secondary outcomes were: 1) all-cause mortality 2) combined CHD 3) strokes and 4) combined cardiovascular disease CVD. QoL was a prespecified secondary outcome[28].

Quality of Life

Of ALLHAT participants, 28,534 (86%) completed at least one bi-annual VAS estimate of QoL. The VAS scale ranged from 0–100. This value was transformed using the statistical Torrance transformation[29, 30]. We analyzed mean baseline QoL and transformed QoL (TQoL) before and after stroke for each participant.

Statistical Analysis

Baseline characteristics are described by stroke status: participants with no stroke versus fatal or nonfatal stroke. Characteristics are also described for participants with no stroke versus those with nonfatal stroke. Groups are compared by T-tests for continuous variables and chi-squared tests for categorical variables.

Mean baseline QoL and TQoL were analyzed by stroke status and by treatment groups. T-tests were used to compare between QoL and TQoL by stroke status. Cox regressions were used to evaluate the impact of baseline QoL and TQoL on stroke status, looking at the QoL variables both continuously and by quartile, both unadjusted and adjusted for selected baseline variables (antihypertensive treatment group, age, gender, race, history MI or stroke, diabetes, history of CHD, smoking status, and baseline SBP, DBP, atrial fibrillation, total

cholesterol, LDL, HDL, triglycerides, and aspirin use). Five-year cumulative stroke rates were estimated using the Kaplan-Meier method.

For participants with nonfatal stroke, QoL and TQol were compared pre-stroke and post-stroke. Two analyses were performed: (1) using baseline QoL or TQol and the first post-stroke QoL or TQoL, and (2) using average of all pre-stroke Qol or TQol measurements and the average of all post-stroke Qol or TQol measurements. The difference between pre-stroke and post-stroke values was calculated and T-tests were used to evaluate if changes differed from 0. Analyses were stratified by treatment group, age, race, and gender. Lastly, predictors of pre-stroke to post-stroke changes in QoL and TQol were evaluated using linear regression, both univariate and multivariate, by age subgroups and for the total group. These were also done in the manners described above, using both single and average pre- and post-stroke values. Results using QoL and TQoL were similar; therefore, only results for TQoL are presented. Similarly, pre-stroke vs post-stroke change results were similar using single and average values; therefore, only results using single pre- and post-stroke VAS are presented.

All analyses were done in STATA by the ALLHAT Coordinating Center.

Results

The consort diagram (Figure 1) outlines participants in ALLHAT randomized to each treatment arm (chlorthalidone, amlodipine, and lisinopril) and numbers of subjects no stroke versus with fatal and non-fatal in-trial strokes. Over mean 4.9 years of follow-up, 683 (4.5%), 382 (4.2%), 460 (5.1%) of 33,357 patients randomized to chlorthalidone (n=15,255), amlodipine (n=9,048), and lisinopril (n=9,054) groups experienced strokes, respectively. Mean times to stroke were similar across treatment arms at 2.67± 1.75 years for Chlorthalidone, 2.66±1.73 years for Amlodipine, and 2.54±1.73 years for Lisinopril arms. Stroke-related mortality across randomized groups were also similar between groups: Chlorthalidone (n=146;21%), Amlodipine (n=82;21%), and Lisinopril 22% (n=103;22%). Of all participants with strokes, 1475 (97%) had baseline VAS evaluation, and 559 (47%) with nonfatal stroke had at least one post-stroke VAS evaluation. Baseline characteristics of ALLHAT participants with nonfatal strokes with and without pre/post stroke QOL measurements (Supp. Table III) demonstrate no significant differences between each treatment groups (p=0.43).

Table 1 shows baseline characteristics of subjects with stroke compared to those without There are significant differences between groups, reflecting established risk factors such as age, race (Blacks>Non-Black), gender (male>female), educational attainment, aspirin use (higher in the stroke group), higher SBP, ASCVD, history of stroke or MI, baseline atrial fibrillation, type 2 diabetes, HDL (all p<.001).

Mean baseline TQoLs (Supp. Table I) were compared by occurrence of fatal and non-fatal stroke events vs no strokes and found to be significantly lower for strokes compared with non-strokes in Chlorthalidone (0.84 vs 0.86, (p=<0.0001)) and amlodipine groups (0.84 vs. 0.86,(P=0.006) but not for lisinopril group (0.85 vs. 0.86; NS).

Table 2 and Figure 2 show the association between baseline TQoL and fatal/nonfatal stroke outcomes. Cox proportional hazards regression analyses were done with and without adjustments for treatment group, age, gender, race, history of MI or stroke, diabetes, history of CHD, smoking, and baseline BP values, atrial fibrillation, cholesterol, LDL, HDL, triglycerides, and aspirin use. In unadjusted model, hazard ratio (HR) of baseline TQoL per 0.1 unit increase, was significantly associated with in-trial strokes with HR 0.90 (95% CI 0.87 – 0.93). After adjustment, HR for stroke events for baseline TQoL per 0.1 unit increase was 0.93 (95% CI 0.89 – 0.98).

Figure 2 shows Kaplan-Meier event curves for first stroke up to 6 years after randomization, by baseline TQoL quartiles. The lowest quartile demonstrated a 36% higher stroke risk (HR=1.36 95% CI 1.18–1.58). In adjusted model, lowest quartile showed a HR of 1.20 (CI 1.00–1.44), equivalent to 20% higher stroke risk compared to highest quartile TQoL. The 2nd lowest quartile had significantly higher stroke risk in unadjusted models (HR=1.16 95% CI 1.00–1.35) but not after risk factor adjustment (HR=1.02, 95% CI 0.85–1.24).

Table 3 and Figure 3 show Pre-stroke and post-stroke TQoL in patients with nonfatal strokes, stratified by treatment arm, age, gender, and race were compared. 559 (47.1%) subjects with non-fatal stroke had pre- and post-stroke QoL scores (Chlorthalidone N=250; Amlodipine N=149; Lisinopril N=160). Post-stroke TQoLs were significantly lower than pre-stroke TQoLs cumulatively (−0.06 SD 0.19, p<0.0001) and when stratified according to treatment groups (−.04; SD .19 in Chlorthalidone, −0.08 SD 0.19 in Amlodipine, and −0.07 SD 0.21 in lisinopril), (p=0.12 for comparison across treatment groups). Mean changes in TQoL increased with age, −0.04, (p=0.08) in subjects 55–64 yrs, −0.06,(p=<0.0001) in 65–75 yrs, and −0.01 (p= <0.0001) in 75+ yrs, although these differences are not significantly different across age groups (p=.18). There were no consistent patterns when TQoL was stratified by gender and race.

The goal of our multivariate analyses, (Table 3 and Supp. Table II), was to determine predictors of change in TQoL in participants with nonfatal stroke, unadjusted (Table 3) and after adjusting for clinical risk variables (Supp. Table II). Table 3 presents unadjusted TQOL change by treatment group, age, sex, and race. Most of the changes were significantly different from 0. However, there were no significant differences across treatment groups, age at stroke, sex, or race. Multivariable analyses, including tests for interaction for age at stroke and baseline TQol and treatment group and baseline TQoL, are shown in Supplemental Table 2. Baseline TQOL was a significant predictor of post-stroke TQoL change. No other clinical risk factor significantly impacted TQOL change. Similarly multivariate regression analysis stratified by age revealed that baseline TQoL was strongest predictor of post-stroke change in TQol in all age groups.

DISCUSSION

The objective of this study was to determine effect of baseline QoL on stroke risk and outcomes in the ALLHAT study, demonstrating utility of simple VAS after previous work has demonstrated VAS and its transformation useful in cost-effectiveness studies in ALLHAT study population[31].

The major findings of this study indicate that baseline QoL is significantly lower in those who suffered a stroke during the average 4.9 years of ALLHAT follow-up, except in Lisinopril treatment group. Subjects in lowest quartile of baseline TQoL had a 20% higher stroke risk compared to reference group of highest quartile in a model adjusted for commonly known stroke risk factors.

Change in QoL showed a strong age effect from 50–64, 65–74 and 75+, which trended across all treatment arms. Amlodipine group had greatest change in QoL and TQoL, but biological and functional meaning of this is unclear. There was no consistent effect by gender or ethnicity.

We examined risk factors for change in TQoL in both univariate and multivariate models. Baseline TQoL was the strongest predictor of average post-stroke TQoL. A smaller effect was seen in stroke subjects less than 75 years of age. Greater TQoL change in older subjects is consistent with previous findings that stroke in older individuals is associated with less favorable outcomes[3233].

The study has several substantial limitations which affect the conclusions and generalizability. ALLHAT remains the largest anti-hypertensive treatment trial, but was not designed as a stroke study. ALLHAT study was a parallel group study. We have created new sub-cohorts in our analysis of this secondary outcome measure, whose power to detect differences may be limited, although sample sizes remain quite large due to size of the complete ALLHAT study population. Conducted between 1994 and 2002, with an average of 4.9 years of follow up (3.2 years for the Doxazosin group) this study unfortunately did not collect data on type of stroke (nor prior stroke type), traditional Neurological outcomes, nor currently used functional outcome measures(BI and mRS). No cognitive outcomes are available, limiting attribution of cognitive change due to stroke post-stroke- QoL. Further, no clinical correlation with QoL and activities of daily living (ADLs) is available and warrants future investigation.

Special effort was made to recruit minorities, and 32% of final cohort were Black non-hispanic[34]. This group alone recorded 620 strokes (40% of total in-study strokes), which is a significant contribution to existing literature. While this minority recruitment effort contributed to groundbreaking utility of the primary outcome in changing hypertension treatment, ALLHAT is not a community based study, and data on Hispanics and Native Americans are not available to make additional generalizations regarding the overall population. Furthermore, individuals may enroll in clinical drug studies for many reasons, further skewing the population studied from the general population.

Prior stroke was not an exclusion criteria for subjects, and inclusion of this group can skew data on baseline QOL measures which correlate with outcome QOL. For this reason, our analysis included analysis of QOL change with in-study stroke, and there is no evidence that subjects with prior strokes were preferentially represented in any treatment arm nor other subgroup. Aspirin usage was higher in subjects with stroke (which includes infarcts and cerebral hemorrhages), and this may affect clinical outcomes [3536] and further analysis of the meaning of this effect cannot be considered. BI and mRS were not collected and comparison

with other stroke clinical trials is limited [1837] . The effect of lipid lowering was not included in our analyses, and is beyond the scope of this paper. Furthermore, generalization of our results to agents other than the treatment groups cannot be assessed.

In a study of ALLHAT size and breadth, it is inevitable that missing data will affect the analysis. There is no evidence of biased data ascertainment, and we acknowledge that missing follow up data on QoL/TQoL post-stroke might affect analytical conclusions, although robustness and face validity of findings remains. Lastly, conclusions derived from subgroup analysis (table 2, Figure 3), especially when stratified by treatment groups, may be underpowered.

QoL is a multiply determined integrative function [16,38], and seeming simplicity of VAS captures a global measure but does not distinguish which QoL component contributes to the results. The magnitude of stroke risk change between highest to lowest QoL group suggests possibility lifestyle change affecting global QoL could make a contribution to stroke risk reduction.

The VAS is a simply administered validated instrument. Its application in this study has revealed important outcomes seen in other conditions such as CABG, MI, hemodialysis, and DM. The use of a VAS tool for QoL assessment should be considered for inclusion in other stroke registries and clinical trials.

Supplementary Material

Refer to Web version on PubMed Central for supplementary material.

Acknowledgments

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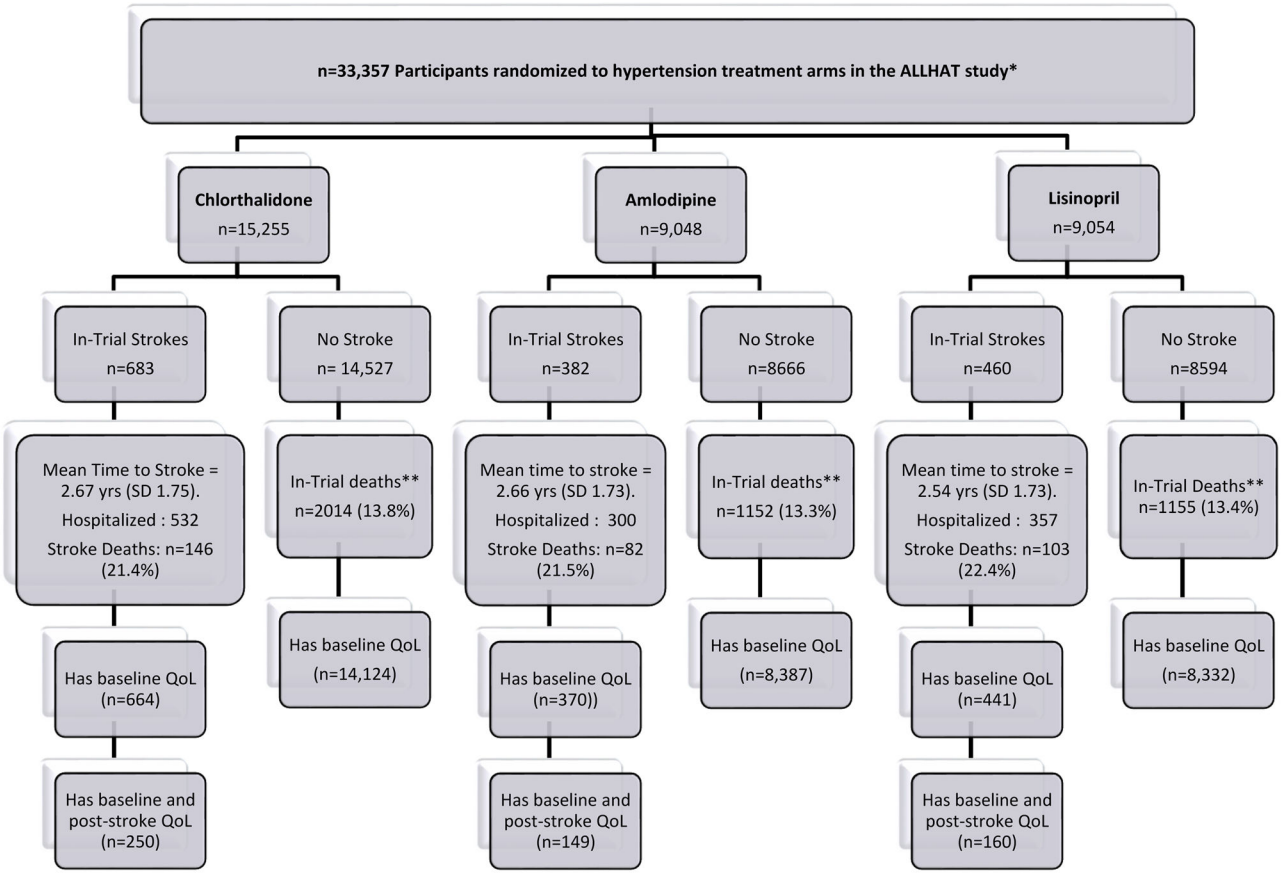

Figure 1.

Consort diagram for stroke outcomes among participants randomized to chlorthalidone, amlodipine, or Lisinopril treatment arms in the Antihypertensive and Lipid-Lowering treatment to prevent Heart Attack Trial (ALLHAT)[28]. **In-trial deaths separate from/does not include incident stroke deaths.

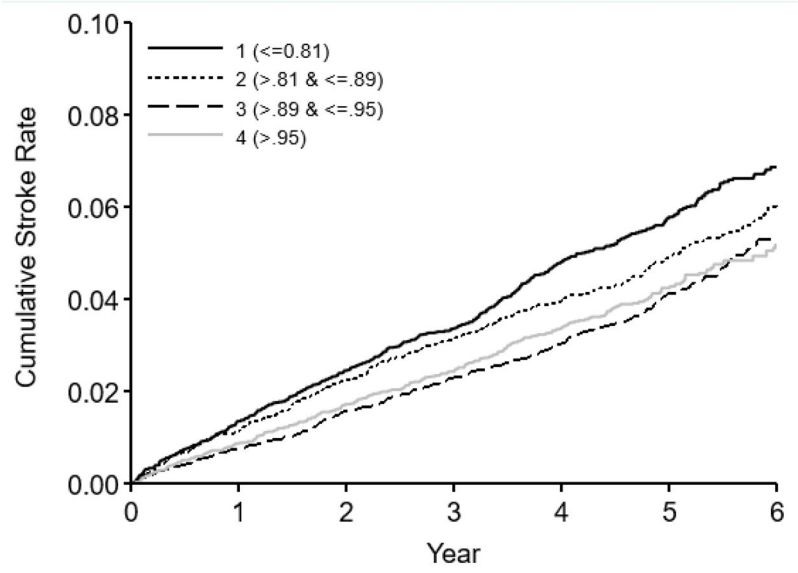

Figure 2. Stroke rates by quartile of baseline TQOL

Kaplan-Meier event curves for first stroke up to 6 years after randomization, by baseline TQoL quartiles. In the adjusted model, the lowest quartile showed a HR of 1.20 (CI 1.00–1.44), equivalent to 20% higher stroke risk compared to highest quartile TQoL.

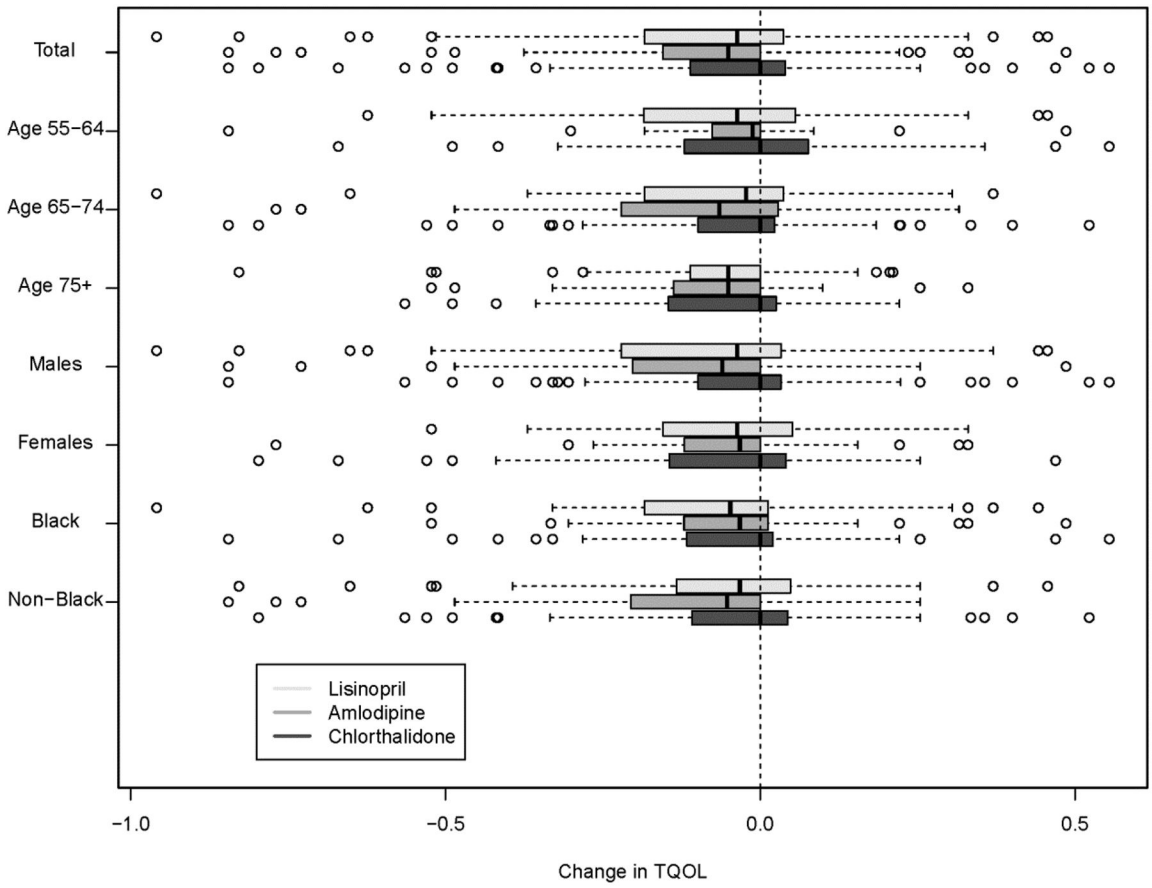

Figure 3.

Box-and-whisker plot of changes in TQOL from pre-stroke to post-stroke (line in shaded boxes indicates median)

Table 1

Baseline characteristics of ALLHAT participants without in-trial stroke vs. in-trial nonfatal stroke

Baseline Characteristic	No in-trial stroke N=31,832	In-trial stroke N=1,525	P-value
Age (years), mean (SD)	66.7 (7.7)	69.8 (7.8)	<0.0001
Age group, n (%)			<0.001
55–64	13,767 (43.3)	417 (27.3)	
>65	18,065 (56.8)	1,108 (72.7)	
Race, n (%)			<0.001
Black	11,172 (35.1)	620 (40.7)	
Non-Black	20,660 (64.9)	905 (59.3)	
Men, n (%)	16,828 (52.9)	891 (58.4)	<0.001
Education in years, mean (SD)	11.0 (4.0)	10.6 (3.9)	0.0001
Medication use, n (%)			
Antihypertensive treatment	28,691 (90.1)	1,398 (91.7)	0.049
Aspirin	11,333 (35.6)	619 (40.6)	<0.001
Lipid medications	7,447 (23.4)	326 (21.4)	0.07
Blood pressure (mm Hg), mean (SD)			
SBP	146.2 (15.6)	148.9 (15.4)	<0.001
DBP	84.1 (10.0)	83.5 (10.5)	0.048
Eligibility risk factors, n (%) *			
Current smoker	6,977 (21.9)	326 (21.4)	0.62
ASCVD †	16,291 (51.2)	907 (59.5)	<0.001
History Myocardial infarction or stroke	7,189 (22.6)	548 (35.9)	<0.001
History coronary revascularization	4,073 (12.8)	237 (15.5)	0.002
Other ASCVD	7,514 (23.6)	387 (25.4)	0.11
Major ST depression or T-wave inversion	3,259 (10.3)	161 (10.7)	0.64
Baseline Atrial fibrillation	287 (1.0)	47 (3.3)	<0.001
Type 2 diabetes	12,436 (39.1)	732 (48.0)	<0.001
Left ventricular Hypertrophy	6,273 (19.7)	343 (22.5)	0.008

Baseline Characteristic	No in-trial stroke N=31,832	In-trial stroke N=1,525	P-value
History CHD, n (%)	7,962 (25.2)	453 (30.1)	<0.001
Body mass index, mean (SD)	29.8 (6.2)	29.2 (6.0)	0.0005
Antihypertensive treatment group, n (%)			0.02
Chlorthalidone	14,572 (45.8)	683 (44.8)	
Amlodipine	8,666 (27.2)	382 (25.1)	
Lisinopril	8,594 (27.0)	460 (30.2)	
Baseline QOL, mean (SD)	0.74 (0.16)	0.72 (0.17)	<0.001
****Transformed baselineQOL, mean (SD)**	0.86 (0.13)	0.84 (0.14)	<0.001
****Transformed baselineQOL quantiles, n (%)**			<0.001
1 (<=0.81)	8,109 (26.3)	463 (31.4)	
2 (>0.81 & <=0.89)	7,470 (24.2)	373 (25.3)	
3 (>0.89 & <=0.95)	8,370 (27.1)	340 (23.1)	
4 (>0.95)	6,894 (22.4)	299 (20.3)	
Biochemical measures, mean (SD)			
Total cholesterol (mg/dL)	216.0 (43.2)	218.6 (48.3)	0.03
LDL cholesterol (mg/dL)	135.7 (37.0)	139.2 (39.7)	0.0006
HDL cholesterol (mg/dL)	46.9 (14.8)	45.4 (14.1)	0.0001
Triglycerides	172.8 (133.6)	171.1 (132.0)	0.68

SBP/DBP=systolic/diastolic blood pressure; ASCVD=atherosclerotic cardiovascular disease; CHD=coronary heart disease; QOL = Quality of Life.

*For trial eligibility, participants had to have at least 1 other risk factor in addition to hypertension.

†History of MI or stroke; history of coronary revascularization; major ST segment depression on T wave inversion on any ECG in the past 2 years; other ASCVD (please consult original ALLHAT trial design for detailed inclusions).

**Transformed Quality of Life – The Torrance transformation was used so that the distribution of QoL utilities better matched the standard utility values such as the time tradeoff or standard gamble.[31,32]

Table 2

Baseline TQOL as Predictors of In-Trial Stroke

	All TQOL	TQoL Quartile 1 (Lowest)	TQoL Quartile 2	TQoL Quartile 3	TQoL Quartile 4 (Highest)
HR for stroke per 0.1 unit (95% CI)					
Unadjusted	0.90 (0.87 – 0.93)	---	---	---	---
Adjusted *	0.93 (0.89 – 0.98)	---	---	---	---
HR by quartile *, **unadjusted**	---	1.36 (1.18 – 1.58)	1.16 (1.00 – 1.35)	0.96 (0.82 – 1.12)	1.00 (ref)
HR by quartile, adjusted **	---	1.20 (1.00 – 1.44)	1.02 (0.85 – 1.24)	0.97 (0.80 – 1.17)	1.00 (ref)
5-year cumulative stroke rates by quartile	0.048 (0.045 – 0.050)	0.060 (0.053 – 0.064)	0.050 (0.044 – 0.055)	0.041 (0.037 – 0.046)	0.042 (0.038 – 0.048)
Number of events by quartile (% of events)	1312	418 (0.31)	333 (0.25)	297 (0.23)	264 (0.20)

Abbreviations: TQOL=transformed quality of life; HR=hazard ratio

*Range of VAS within each quartile of Qol - Highest Quartile >−.85, 3rd Quartile 0.75–0.85, 2nd Quartile 0.65–0.75, Lowest Quartile <0.65.

**Adjusted for antihypertensive treatment group, age, gender, race, prior MI or stroke, diabetes, history of CHD, smoking status, and baseline SBP, DBP, atrial fibrillation, total cholesterol, LDL, HDL, triglycerides, and aspirin use.

Table 3

Comparison of Prestroke and Poststroke TQoL in Patients With Nonfatal Stroke

	Chlorthalidone	Amlodipine	Lisinopril	Total
Total				
Total participants with stroke and TQoL	250	149	160	559
BL TQoL, mean (SD)	0.84 (0.14)	0.85 (0.13)	0.86 (0.13)	0.85 (0.14)
First poststroke TQoL, mean (SD)	0.81 (0.17)	0.78 (0.18)	0.79 (0.19)	0.79 (0.18)
Change in TQoL, mean (SD)	−0.04 (0.19)	−0.08 (0.19)	−0.07 (0.21)	−0.06 (0.19)
P value	0.0011	<0.0001	0.0001	<0.0001
Age 55–64 y				
Total participants with stroke and TQoL	50	27	44	121
BL TQoL, mean (SD)	0.82 (0.16)	0.81 (0.16)	0.85 (0.14)	0.83 (0.15)
First poststroke TQoL, mean (SD)	0.81 (0.17)	0.77 (0.19)	0.80 (0.18)	0.80 (0.18)
Change in TQoL, mean (SD)	−0.02 (0.22)	−0.04 (0.21)	−0.05 (0.22)	−0.04 (0.22)
P value	0.5763	0.3103	0.1270	0.0754
Age 65–74 y				
Total participants with stroke and TQoL	116	74	75	265
BL TQoL, mean (SD)	0.84 (0.14)	0.85 (0.14)	0.85 (0.13)	0.85 (0.14)
First poststroke TQoL, mean (SD)	0.81 (0.18)	0.77 (0.18)	0.79 (0.18)	0.80 (0.18)
Change in TQoL, mean (SD)	−0.04 (0.19)	−0.09 (0.20)	−0.07 (0.21)	−0.06 (0.20)
P value	0.0411	0.0005	0.0079	<0.0001
Age 75+ y				
Total participants with stroke and TQoL	84	48	41	173
BL TQoL, mean (SD)	0.86 (0.13)	0.88 (0.11)	0.88 (0.12)	0.87 (0.12)
First poststroke TQoL, mean (SD)	0.81 (0.17)	0.80 (0.16)	0.79 (0.21)	0.80 (0.17)
Change in TQoL, mean (SD)	−0.05 (0.15)	−0.08 (0.16)	−0.09 (0.20)	−0.07 (0.17)
P value	0.0015	0.0014	0.0089	<0.0001
Men				
Total participants with stroke and TQoL	154	92	102	348
BL TQoL, mean (SD)	0.85 (0.15)	0.85 (0.14)	0.86 (0.14)	0.85 (0.15)
First poststroke TQoL, mean (SD)	0.82 (0.15)	0.76 (0.18)	0.78 (0.20)	0.79 (0.18)
Change in TQoL, mean (SD)	−0.03 (0.17)	−0.09 (0.21)	−0.08 (0.23)	−0.06 (0.20)
P value	0.0686	0.0001	0.0006	<0.0001
women				
Total participants with stroke and TQoL	96	57	58	211
BL TQoL, mean (SD)	0.84 (0.13)	0.86 (0.12)	0.85 (0.12)	0.85 (0.12)
First poststroke TQoL, mean (SD)	0.78 (0.20)	0.81 (0.16)	0.81 (0.16)	0.79 (0.18)
Change in TQoL, mean (SD)	−0.06 (0.20)	−0.05 (0.16)	−0.05 (0.17)	−0.05 (0.18)
P value	0.0046	0.0145	0.0490	<0.0001
Blacks				

	Chlorthalidone	Amlodipine	Lisinopril	Total
Total participants with stroke and TQoL	95	51	68	214
BL TQoL, mean (SD)	0.84 (0.16)	0.85 (0.14)	0.86 (0.14)	0.85 (0.15)
First poststroke TQoL, mean (SD)	0.79 (0.19)	0.81 (0.13)	0.79 (0.19)	0.80 (0.18)
Change in TQoL, mean (SD)	−0.05 (0.20)	−0.04 (0.17)	−0.07 (0.22)	−0.07 (0.23)
P value	0.0162	0.0925	0.0085	0.0001
Non-blacks				
Total participants with stroke and TQoL	155	98	92	345
BL TQoL, mean (SD)	0.85 (0.13)	0.86 (0.13)	0.86 (0.12)	0.85 (0.13)
First poststroke TQoL, mean (SD)	0.81 (0.16)	0.76 (0.19)	0.79 (0.19)	0.79 (0.18)
Change in TQoL, mean (SD)	−0.03 (0.18)	−0.09 (0.20)	−0.06 (0.20)	−0.06 (0.19)
P value	0.0259	<0.0001	0.0033	<0.0001

BL indicates baseline; and TQoL, transformed quality of life.