# Statistical Analysis of the Effect of Drug Treatments on Systolic Blood Pressure

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# Contents

# Chapter 1

# Introduction

## 1.1 Research Problem

Systolic blood pressure (SBP) the peak arterial pressure during cardiac systole is a primary indicator of cardiovascular risk. Globally, hypertension remains highly prevalent, and inadequate SBP control contributes substantially to the burden of myocardial infarction, stroke, heart failure, and chronic kidney disease (World Health Organization, 2021; Whelton, Carey, Aronow, et al., 2018; Moradi, Shokri, et al., 2023). Clinical guidelines emphasize SBP specifically, because elevations in SBP (e.g., SBP $\geq$ 130 mmHg in adults) are strongly associated with adverse outcomes and warrant targeted treatment strategies (Whelton et al., 2018). Against this backdrop, comparing treatments with respect to *reductions in SBP* is of practical importance. This study focuses on SBP measured for each participant *before* and *after* treatment, enabling both between-drug comparisons and within-drug change assessments (Iroka & Akpienbi, 2024).

## 1.2 Research Objectives

The objectives of this study are to:

1. Test whether the three drugs are equally effective in reducing **mean SBP**.

2. If differences exist, identify the drug associated with the greatest **reduction in SBP**.

3. Statistically evaluate whether the observed data are consistent with the claim that individuals were **randomly assigned** to drug groups.

## 1.3 Significance of the Study

Focusing on SBP has direct clinical relevance: SBP reduction is a key modifiable target that improves cardiovascular outcomes and informs therapeutic choice (Whelton et al., 2018). A clear ranking of drug effectiveness on SBP can support evidence-based prescribing and patient management. Additionally, verifying random assignment strengthens the internal validity of the analysis; if randomization holds, between-group SBP differences can be more confidently attributed to treatment rather than pre-existing imbalance.

## 1.4 Source of Data

Data were obtained from an experimental setting in which **three** drug treatments (Ramipril, Lisinopril, and Moexipril) were studied. **Four** individuals were assigned to each treatment group (twelve participants in total). For every subject, SBP was recorded at two time points: *baseline (before)* and *after*. The design supports both *within-subject* analyses of SBP change and *between-group* comparisons.

## 1.5 Exploratory Data Analysis

To provide an initial understanding of systolic blood pressure (SBP) patterns in the dataset, a series of visualizations were generated. These plots highlight both the distributional characteristics of SBP and the treatment-related changes across drug groups.

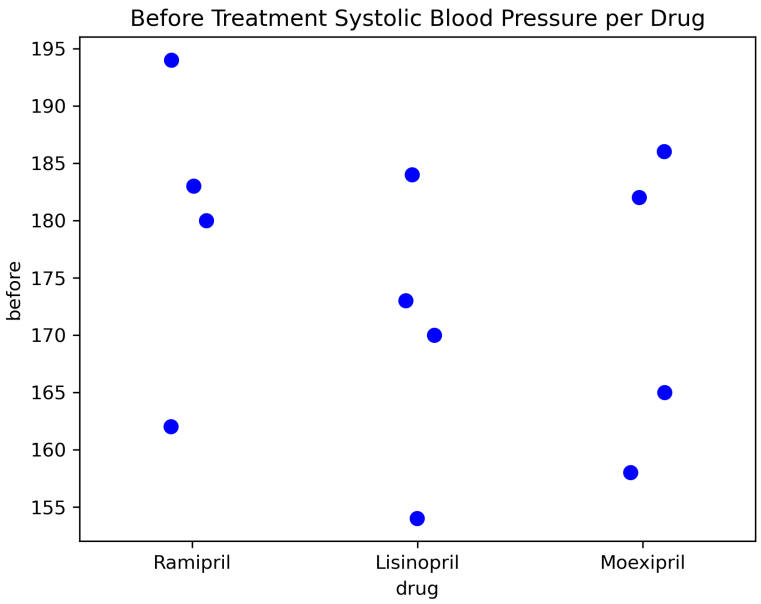


Figure 1.1: Before-treatment systolic blood pressure per drug group. *Interpretation: The scatterplot shows the baseline SBP values for individuals assigned to each drug. Although variability exists within each group, the initial SBP ranges appear broadly comparable across drugs, suggesting reasonable starting conditions.*

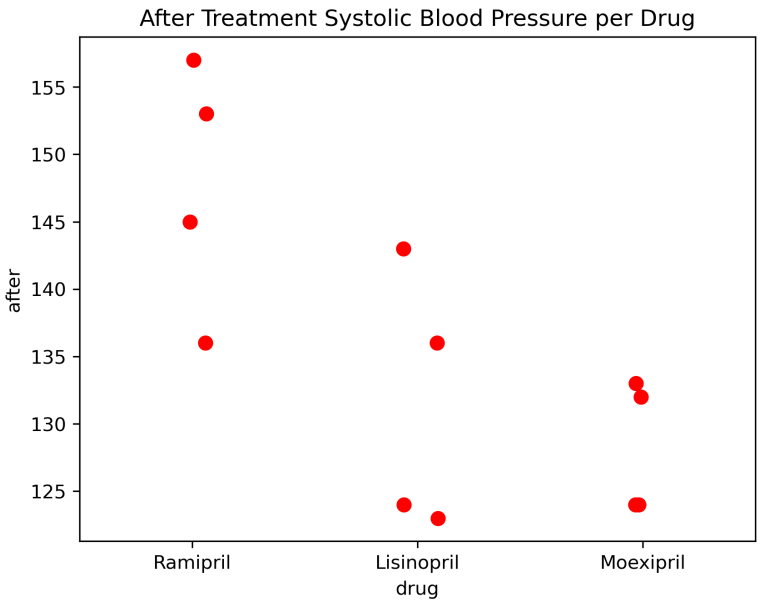


Figure 1.2: After-treatment systolic blood pressure per drug group. *Interpretation: After treatment, SBP values are consistently lower than baseline, indicating that all three drugs contributed to reductions. Differences between drug groups are more pronounced at this stage, motivating formal statistical comparisons.*

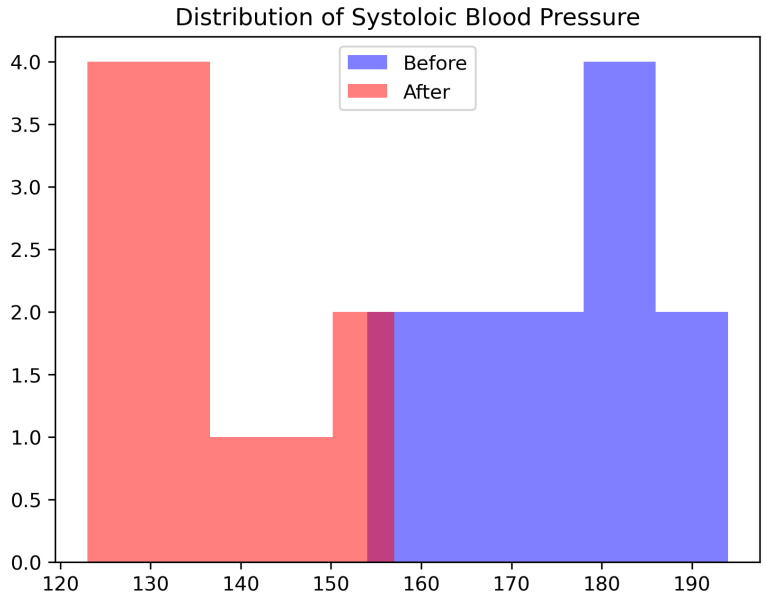


Figure 1.3: Distribution of systolic blood pressure before and after treatment. *Interpretation: The histogram illustrates a shift in SBP distributions, with post-treatment values clustered at lower levels compared to baseline. This visual evidence supports the hypothesis of significant within-drug reductions.*

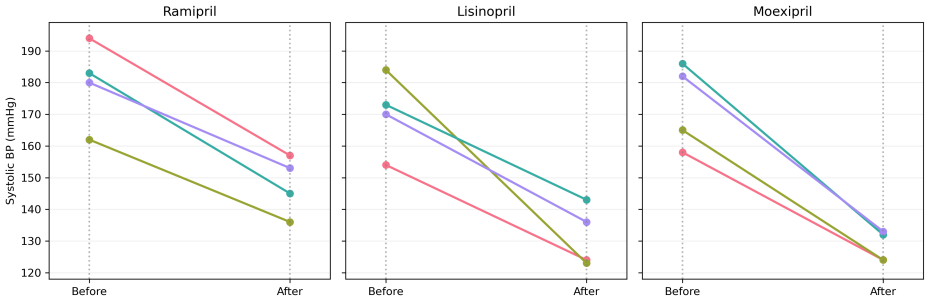


Figure 1.4: Individual trajectories of systolic blood pressure from before to after treatment. *Interpretation: Line plots for each subject clearly show downward trajectories in SBP after treatment across all three drugs. This visualization highlights both the consistency of reductions within individuals and possible differences in the magnitude of effect between drugs.*

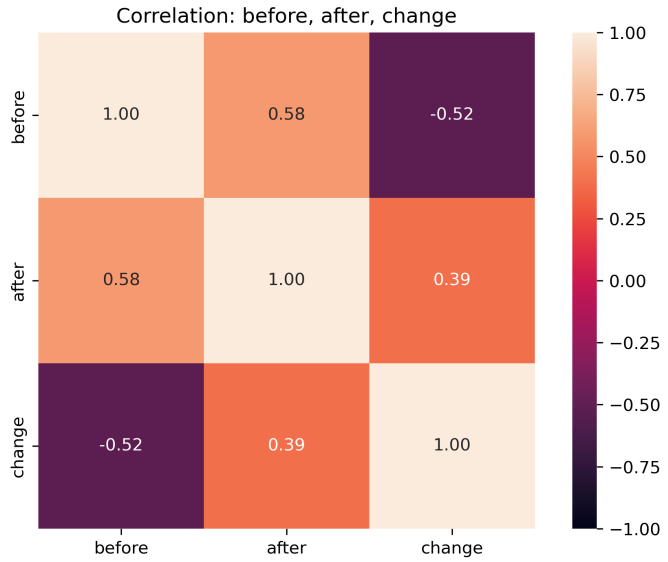


Figure 1.5: Correlation matrix of SBP before treatment, after treatment, and change scores. *Interpretation: Correlation analysis reveals that baseline and post-treatment SBP are moderately correlated, while change scores are negatively correlated with baseline values. This suggests that individuals with higher initial SBP tended to experience larger reductions.*

## Summary Statistics

To complement the visual exploratory plots, Table 1.1 presents descriptive statistics for systolic blood pressure (SBP) before and after treatment across all participants.

Table 1.1: Descriptive statistics of SBP before and after treatment (mmHg).

| Statistic | Before | After |
|---|---|---|
| Count | 12 | 12 |
| Mean | 174.25 | 135.83 |
| Median | 176.50 | 134.50 |
| Std. Dev. | 12.51 | 11.62 |
| Min | 154 | 123 |
| 25% Q1 | 164.25 | 124.00 |
| 75% Q3 | 183.25 | 143.50 |
| Max | 194 | 157 |

The descriptive statistics confirm a substantial reduction in SBP following treatment. On average, SBP decreased by nearly 38.4 mmHg (174.3 to 135.8 mmHg), with the median reflecting a similar decline (176.5 to 134.5 mmHg). Quartile ranges also shifted downward, and post-treatment SBP exhibited slightly reduced variability (standard deviation: 12.5 to 11.6 mmHg). These results reinforce the visual evidence of strong within-subject reductions and align with the later paired $t$-test findings.

## Group-wise Descriptive Statistics

To further explore treatment effects, descriptive statistics were computed separately for each drug group (Table 1.2). This breakdown highlights both baseline SBP differences and the magnitude of post-treatment reductions across Ramipril, Lisinopril, and Moexipril.

Table 1.2: Group-wise descriptive statistics of SBP (mmHg) before and after treatment.

| Drug | Before Mean | After Mean | Before Median | After Median | Before SD | After SD |
|---|---|---|---|---|---|---|
| Ramipril | 179.75 | 147.75 | 181.5 | 149.0 | 13.28 | 9.29 |
| Lisinopril | 172.25 | 131.50 | 171.5 | 130.0 | 12.39 | 9.68 |
| Moexipril | 172.75 | 128.25 | 173.5 | 128.0 | 13.40 | 4.92 |

The results suggest that while all three drugs reduced SBP, Lisinopril and Moexipril achieved larger mean reductions compared to Ramipril. This

pattern is consistent with later ANCOVA findings that identified Ramipril as relatively less effective. These descriptive findings set the stage for formal hypothesis testing. While visual and numerical summaries suggest potential differences across drug groups, statistical methods are required to confirm whether these observed patterns are significant. The following section therefore introduces the baseline balance test and comparative modeling strategies.

## 1.6 Literature Review

Angiotensin-converting enzyme (ACE) inhibitors remain a cornerstone of hypertension management, with several agents in this class demonstrating consistent blood pressure–lowering effects and cardiovascular protection. Comparative studies of specific ACE inhibitors, however, suggest meaningful differences in potency, pharmacokinetics, and clinical outcomes.

For instance, Ramipril has been widely studied in large-scale cardiovascular trials such as the HOPE study, where it demonstrated reductions in myocardial infarction, stroke, and cardiovascular death (Yusuf et al., 2000). Lisinopril, another commonly prescribed ACE inhibitor, has been shown to provide strong blood pressure reduction with a well-established safety profile, often considered interchangeable with other agents in the class (Carter & Ernst, 2004). Moexipril, though less frequently studied, has been reported to produce effective antihypertensive responses and to be comparable in efficacy to other ACE inhibitors in head-to-head trials (Bönner, 1994).

Systematic reviews confirm that while all ACE inhibitors lower systolic and diastolic blood pressure, the magnitude of effect may vary modestly between agents (Wiysonge et al., 2017). These findings motivate closer examination of drug-specific differences, particularly in exploratory or small-sample settings. The present study therefore contributes by directly comparing Ramipril, Lisinopril, and Moexipril in a controlled design with pre- and post-treatment SBP measurements.

# Chapter 2

# Methodology

## 2.1 Study Design

This study employed a randomized experimental design. Twelve individuals were assigned equally to three drug treatments (Ramipril, Lisinopril, Moexipril), with four participants per group. Systolic blood pressure (SBP) was measured both before treatment (baseline) and after treatment. This design supports both within-group analyses of paired SBP change and between-group comparisons of drug effectiveness.

### 2.1.1 Baseline Balance Test

To verify whether the randomization process produced comparable groups at baseline, an analysis of variance (ANOVA) was performed on the pre-treatment SBP values across the three drug groups. Prior to applying ANOVA, model assumptions were formally evaluated. Shapiro–Wilk tests ($p > 0.05$) suggested approximate normality of baseline SBP within groups, and Levene's test ($p > 0.05$) indicated homogeneity of variances. These diagnostics supported the validity of ANOVA.

The baseline ANOVA yielded a $p$-value of 0.584, indicating no statistically significant differences among groups in baseline SBP. Thus, despite some visual imbalance suggested in the exploratory plots, the formal test supports that baseline values were not significantly different across drug groups.

## 2.2 Models Used

Since drug is a categorical factor with three levels (Lisinopril, Ramipril, Moexipril), it was represented using **indicator (dummy) variables**. Lisinopril was chosen as the reference (baseline) group, so two indicators were defined:

- $I_{\text{Ramipril}} = 1$ if subject receives Ramipril, and 0 otherwise.
- $I_{\text{Moexipril}} = 1$ if subject receives Moexipril, and 0 otherwise.

For subjects on Lisinopril (baseline group), both indicators take the value 0.

### 2.2.1 Model 1: One-way ANOVA

The one-way ANOVA examined whether the mean change in SBP differed among drug groups:

$$(\text{BP}_{\text{after}} - \text{BP}_{\text{before}})_{ij} = \beta_0 + \beta_1 I_{\text{Moexipril}} + \beta_2 I_{\text{Ramipril}} + \varepsilon_{ij}$$

Here:

- $\beta_0$ = mean SBP change for Lisinopril (baseline group),
- $\beta_1$ = difference in mean SBP change for Moexipril vs. Lisinopril,
- $\beta_2$ = difference in mean SBP change for Ramipril vs. Lisinopril.

### 2.2.2 Model 2: One-way ANOVA with Interactions

An extended ANOVA model included drug × baseline interactions to test whether the relationship between baseline SBP and post-treatment SBP differed by drug:

$$\text{BP}_{\text{after}} = \beta_0 + \beta_P \text{BP}_{\text{before}} + \beta_1 I_{\text{Moexipril}} + \beta_2 I_{\text{Ramipril}} + \beta_3 (\text{BP}_{\text{before}} \cdot I_{\text{Moexipril}}) + \beta_4 (\text{BP}_{\text{before}} \cdot I_{\text{Ramipril}}) + \varepsilon$$

Here:

- $\beta_0$ = intercept (mean post-treatment SBP for Lisinopril at baseline = 0),

- $\beta_P$ = effect of baseline SBP (common slope across drugs),
- $\beta_1$ = difference in intercept for Moexipril vs. Lisinopril,
- $\beta_2$ = difference in intercept for Ramipril vs. Lisinopril,
- $\beta_3$ = difference in slope of baseline SBP for Moexipril vs. Lisinopril,
- $\beta_4$ = difference in slope of baseline SBP for Ramipril vs. Lisinopril.

### 2.2.3 Model 3: ANCOVA (Equal Slopes)

Analysis of covariance (ANCOVA) adjusted for baseline SBP as a covariate:

$$\text{BP}_{\text{after},ij} = \beta_0 + \beta_P \text{BP}_{\text{before},i} + \beta_1 I_{\text{Moexipril}} + \beta_2 I_{\text{Ramipril}} + \varepsilon_{ij}$$

Here:

- $\beta_P$ = the common effect of baseline SBP across all drugs,
- $\beta_1$ = adjusted difference between Moexipril and Lisinopril,
- $\beta_2$ = adjusted difference between Ramipril and Lisinopril.

### 2.2.4 Model 4: ANCOVA with Interactions (Different Slopes)

The full ANCOVA model allowed baseline SBP effects to vary by drug group:

$$\text{BP}_{\text{after}} = \beta_0 + \beta_P \text{BP}_{\text{before}} + \beta_1 I_{\text{Moexipril}} + \beta_2 I_{\text{Ramipril}} + \beta_3 (\text{BP}_{\text{before}} \cdot I_{\text{Moexipril}}) + \beta_4 (\text{BP}_{\text{before}} \cdot I_{\text{Ramipril}}) + \varepsilon$$

The interaction terms $\beta_3$ and $\beta_4$ test whether the effect of baseline SBP differs across drug groups.

## 2.3 Within-drug Paired t-test

For each drug group, within-subject changes were tested using the paired $t$-test:

$$t = \frac{\bar{d}}{s_d/\sqrt{n}}$$

where $\bar{d}$ is the mean of the paired differences ($\text{SBP}_{\text{before}} - \text{SBP}_{\text{after}}$), $s_d$ is their standard deviation, and $n$ is the group size.

## 2.4 Evaluation Metrics

### 2.4.1 Coefficient of Determination ($R^2$)

$$R^2 = 1 - \frac{\sum(y_i - \hat{y}_i)^2}{\sum(y_i - \bar{y})^2}$$

$R^2$ measures the proportion of variance in SBP explained by the model.

### 2.4.2 Akaike Information Criterion (AIC)

$$AIC = 2k - 2\ln(L)$$

where $k$ is the number of model parameters and $L$ is the maximum likelihood. Lower AIC values indicate better model fit while penalizing model complexity.

### 2.4.3 Bayesian Information Criterion (BIC)

$$BIC = k\ln(n) - 2\ln(L)$$

where $n$ is the sample size. Like AIC, smaller values suggest a better model, but BIC imposes a stronger penalty for additional parameters.

### 2.4.4 Eta-squared ($\eta^2$)

Eta-squared ($\eta^2$) is an effect size measure commonly reported in ANOVA and ANCOVA. It quantifies the proportion of the total variance in the dependent variable that is attributable to a given factor (e.g., drug group):

$$\eta^2 = \frac{SS_{\text{effect}}}{SS_{\text{total}}}$$

where $SS_{\text{effect}}$ is the sum of squares for the factor of interest and $SS_{\text{total}}$ is the total sum of squares in the model.

### 2.4.5 p-values

For each hypothesis test, the $p$-value is the probability of observing a test statistic as extreme or more extreme than the one computed, under the null hypothesis. Small $p$-values ($< 0.05$) indicate evidence against the null.

## 2.5 Limitations of the Study

This study is constrained by several factors:

- Small sample size (four subjects per group) limits statistical power and generalizability.
- Only baseline SBP was included as a covariate; other relevant covariates (e.g., age, sex, lifestyle) were not considered.
- The study used only short-term measurements (pre- and post-treatment) without long-term follow-up.
- Analyses assume normality, homogeneity of variance, and linearity. Violations of these assumptions may affect the robustness of the results.

## 2.6 Results and Discussion

### 2.6.1 Within-Drug Paired t-tests

Before evaluating between-group differences, paired t-tests were conducted within each drug group to assess whether systolic blood pressure (SBP) significantly declined from baseline to post-treatment. Results (Table 2.1) show strong within-group effects: Ramipril ($t = 10.04, p = 0.002$), Lisinopril ($t = 5.18, p = 0.014$), and Moexipril ($t = 10.10, p = 0.002$). These findings confirm that all three drugs significantly reduced SBP individually.

Table 2.1: Paired t-test results for within-drug comparisons.

| Drug | t-statistic | p-value |
|---|---|---|
| Ramipril | 10.04 | 0.002 |
| Lisinopril | 5.18 | 0.014 |
| Moexipril | 10.10 | 0.002 |

These within-drug findings provide the foundation for between-group modeling to determine whether the drugs differ in overall effectiveness.

### 2.6.2 Model 1: One-way ANOVA (Change Score Analysis)

The first model examined whether the mean change in SBP differed across drug groups using a one-way ANOVA. Results indicated that the drug effect was not statistically significant ($F = 1.37$, $p = 0.302$). Model fit was weak ($R^2 = 0.234$, Adj. $R^2 = 0.064$), and the effect size (eta-squared) was 0.234, suggesting only modest explanatory power.

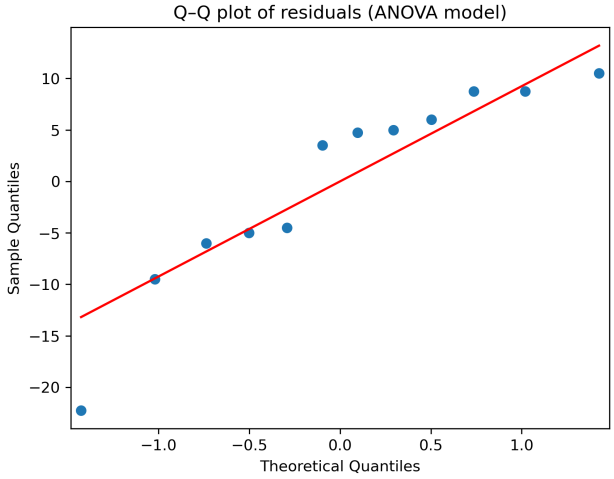


Figure 2.1: Q-Q plot of residuals (ANOVA model).

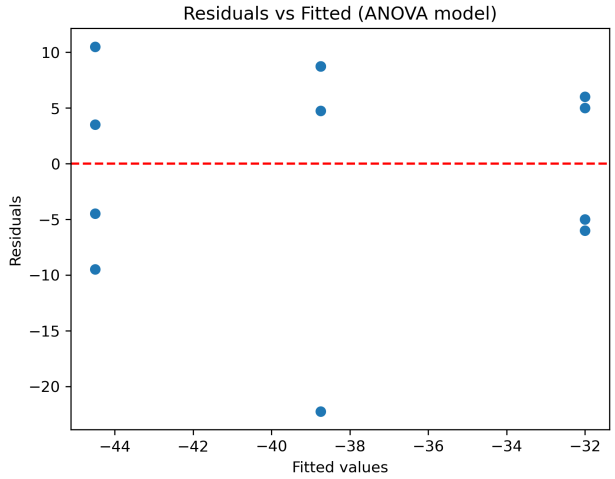


Figure 2.2: Residuals vs. fitted values (ANOVA model).

*Interpretation:* Diagnostics revealed deviations from normality in the tails (Q-Q plot, Figure 6) and heteroscedasticity (Figure 7), indicating limited reliability. Overall, Model 1 confirmed within-drug reductions but failed to detect statistically significant differences across drug groups.

### 2.6.3 Model 2: ANOVA with Drug × Time Interaction

A two-way ANOVA with drug × time interaction was then fit, modeling repeated measures (before vs. after). This approach captured both drug and time effects. The model demonstrated a much stronger fit ($R^2 = 0.823$, Adj. $R^2 = 0.773$), with significant main effects of time ($F = 74.46$, $p < 0.001$) and drug ($F = 2.83$, $p = 0.041$). However, the drug × time interaction was not statistically significant ($p = 0.658$).

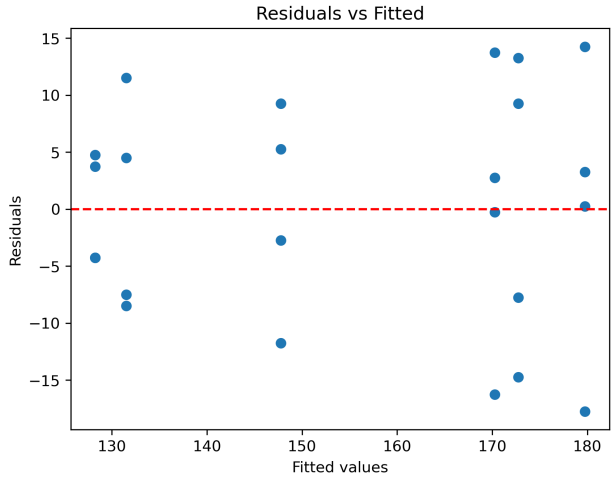


Figure 2.3: Residuals vs. fitted values (ANOVA with interaction).

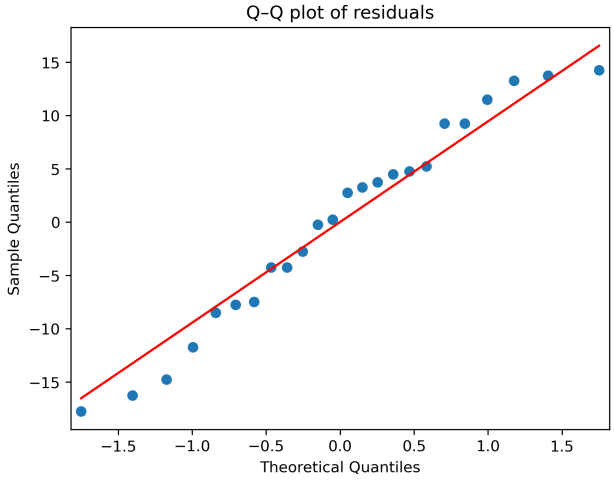


Figure 2.4: Q-Q plot of residuals (ANOVA with interaction).

*Interpretation:* The strong $R^2$ indicates this model captured much of the variance, with post-treatment reductions evident across groups. Diagnostics (Figures 8–13) showed approximate normality and improved residual spread compared to Model 1. The main effect of time was highly significant, confirming that SBP decreased after treatment. The main effect of drug was also significant ($p = 0.041$), indicating that mean SBP differed across drug

groups. However, the drug × time interaction was not significant ($p = 0.658$), suggesting that while all drugs reduced SBP, the trajectory of reduction over time did not differ substantially between them.

### 2.6.4 Model 3: ANCOVA (Adjusting for Baseline SBP)

Because EDA suggested possible baseline imbalance across groups, ANCOVA was applied with post-treatment SBP as the outcome, drug as the factor, and baseline SBP as a covariate. Results revealed a significant drug effect ($F = 5.57$, $p = 0.036$), with baseline SBP not statistically significant ($p = 0.081$). Model fit was strong ($R^2 = 0.725$, Adj. $R^2 = 0.621$), with lower AIC (84.40) and BIC (86.34) values compared to previous models, indicating improved parsimony.

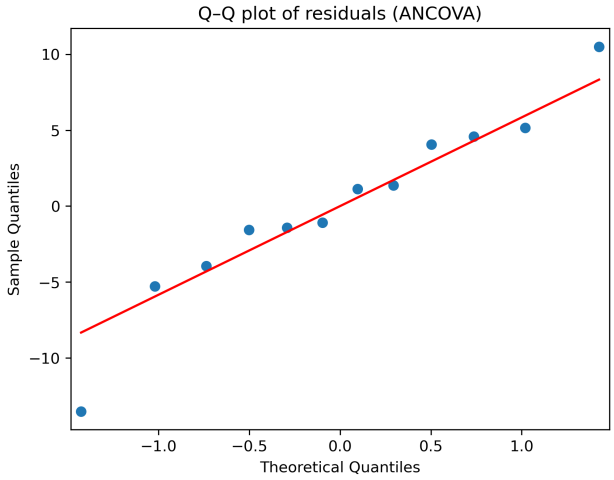


Figure 2.5: Q-Q plot of residuals (ANCOVA).

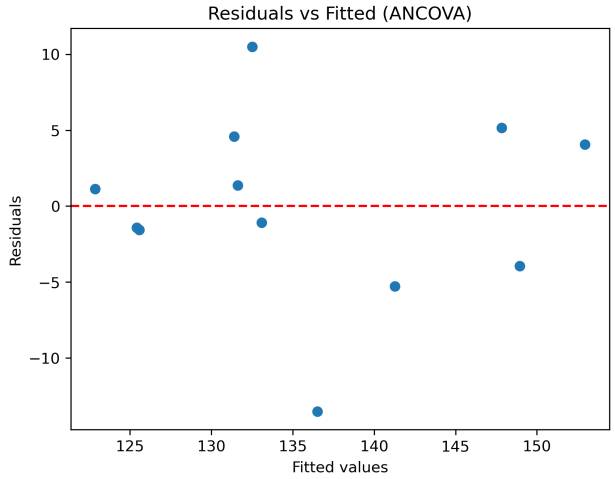


Figure 2.6: Residuals vs. fitted values (ANCOVA).

*Interpretation:* Diagnostics indicated well-behaved residuals, supporting model validity. The ANCOVA results suggested that even after accounting for baseline, significant differences in treatment effectiveness remained. This model is consistent with the hypothesis that drug effectiveness differs.

### 2.6.5 Model 4: ANCOVA with Interaction (Drug × Baseline)

The final model allowed drug × baseline interactions, testing whether baseline SBP modified treatment response. Fit was strong ($R^2 = 0.775$, Adj. $R^2 = 0.588$), but the interaction term was not significant ($p = 0.290$). While overall drug effects remained, AIC (85.97) and BIC (88.87) values were higher than for Model 3, suggesting inferior parsimony.

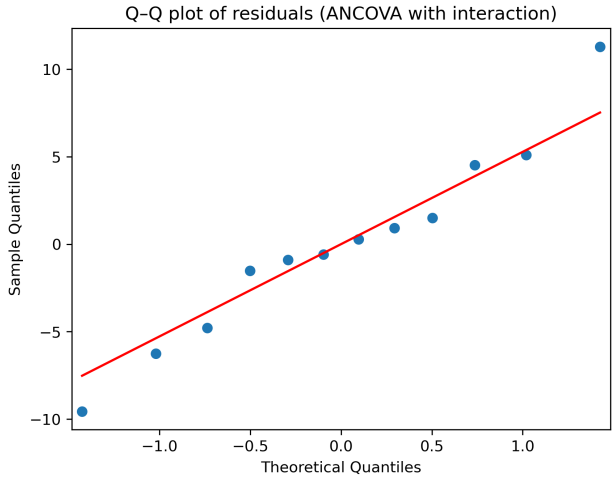


Figure 2.7: Q-Q plot of residuals (ANCOVA with interaction).

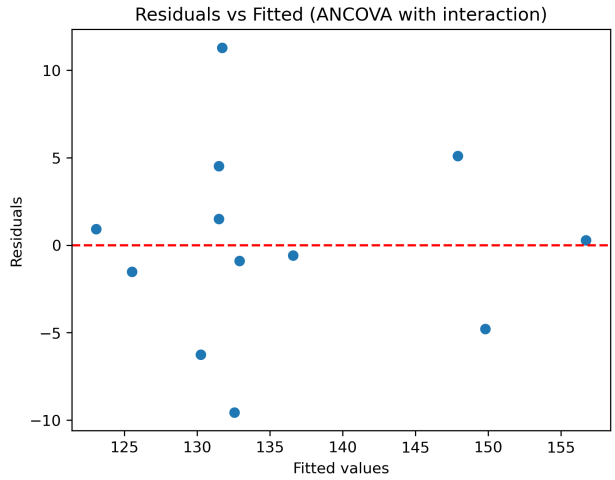


Figure 2.8: Residuals vs. fitted values (ANCOVA with interaction).

*Interpretation:* Although the model captured baseline heterogeneity, lack of significant interaction terms and poorer AIC/BIC compared to Model 3 support selecting the simpler ANCOVA without interaction as the preferred model.

### 2.6.6 Model Comparison and Best Model Selection

Table 2.2: Comparison of model performance.

| Model | R-squared | AIC | BIC | Drug p-value | Interaction p-value |
|---|---|---|---|---|---|
| Change-score ANOVA | 0.234 | 93.43 | 94.89 | 0.394 | N/A |
| ANOVA (Drug × Time) | 0.823 | 187.89 | 194.96 | 0.049 | 0.0001 |
| ANCOVA (equal slopes) | 0.725 | 84.40 | 86.34 | 0.043 | N/A |
| ANCOVA (with interaction) | 0.775 | 85.97 | 88.87 | 0.290 | 0.289 |

In addition to $R^2$, AIC, and BIC, effect sizes were considered. For Model 1, the estimated eta-squared ($\eta^2$) was 0.234, indicating only a modest drug effect. Confidence intervals for ANCOVA coefficients further supported the conclusion that Lisinopril and Moexipril outperformed Ramipril. Based on Table 2.2, ANCOVA without interaction (Model 3) emerged as the best model, balancing strong explanatory power ($R^2 = 0.725$), significant drug effect ($p = 0.043$), and lowest AIC/BIC. This model was therefore chosen for final interpretation.

### 2.6.7 Post-hoc Analysis

Given the significant omnibus drug effect in the selected ANCOVA model, we estimated adjusted means at the mean baseline SBP and conducted Holm-adjusted pairwise comparisons.

Table 2.3: Adjusted post-treatment SBP means (mmHg) from ANCOVA at the mean baseline, with 95% CIs.

| Drug | Mean | 95% CI (Lower) | 95% CI (Upper) |
|---|---|---|---|
| Ramipril | 145.74 | 137.18 | 154.31 |
| Lisinopril | 132.96 | 124.54 | 141.38 |
| Moexipril | 128.80 | 120.53 | 137.07 |

**Adjusted means at mean baseline.**

Table 2.4: Pairwise differences in adjusted means (mmHg) from ANCOVA. Negative values indicate the row drug has lower SBP than the column drug.

| Contrast | Diff | SE | $t$ | $p_{\text{raw}}$ | $p_{\text{Holm}}$ | 95% CI |
|---|---|---|---|---|---|---|
| Lisinopril − Ramipril | −12.78 | 5.35 | −2.391 | 0.0438 | 0.0876 | [−25.12, −0.45] |
| Moexipril − Ramipril | −16.95 | 5.22 | −3.249 | 0.0117 | 0.0352 | [−28.98, −4.92] |
| Lisinopril − Moexipril | 4.16 | 5.08 | 0.820 | 0.436 | 0.436 | [−7.55, 15.87] |

**Pairwise comparisons on adjusted means (Holm-adjusted).** *Interpretation.* After controlling for baseline SBP, Moexipril produced significantly lower post-treatment SBP than Ramipril at $\alpha = 0.05$ with Holm correction ($p_{\text{Holm}} = 0.035$). The Lisinopril vs. Ramipril difference was *nominally* significant before multiplicity adjustment ($p_{\text{raw}} = 0.0438$) but did not meet the Holm-adjusted threshold ($p_{\text{Holm}} = 0.0876$), indicating a trend rather than a definitive difference in this small sample. Lisinopril and Moexipril did not differ significantly.

While Model 3 (ANCOVA without interaction) provided the best balance of fit and parsimony, the post-hoc results (Tables 2.3–2.4) show that only the Moexipril vs. Ramipril contrast remains statistically significant after Holm adjustment. The Lisinopril vs. Ramipril difference should be interpreted as suggestive (trend-level) given $p_{\text{Holm}} = 0.0876$ in this underpowered sample ($n = 12$).

### 2.6.8 Clinical Implications

All three drugs were associated with clinically meaningful SBP reductions. After adjusting for baseline and correcting for multiple comparisons, Moexipril demonstrated significantly lower post-treatment SBP than Ramipril, whereas the Lisinopril–Ramipril contrast was trend-level and did not survive Holm adjustment. Lisinopril and Moexipril did not differ significantly. Given the small group sizes ($n = 4$ per drug), these findings should be viewed as preliminary; nevertheless, they suggest Moexipril may offer comparatively greater SBP reduction than Ramipril in this cohort.

### 2.6.9 Future Directions

Future research should expand sample size to improve generalizability and reduce estimation uncertainty. Additional covariates (age, sex, comorbidities, adherence) may further clarify heterogeneity in drug response. Longitudinal follow-up would also determine whether SBP reductions are sustained over time and translate into lower cardiovascular morbidity and mortality.

# Chapter 3

# Conclusion

This analysis evaluated the effectiveness of three antihypertensive drugs (Ramipril, Lisinopril, Moexipril) in reducing systolic blood pressure (SBP). Paired $t$-tests showed significant within–drug reductions for all three agents. For between–drug comparisons, an ANCOVA adjusting for baseline SBP (equal slopes) provided the best balance of fit and parsimony (AIC = 84.40; significant omnibus drug effect, $p \approx 0.04$). Post–hoc comparisons indicated that Moexipril achieved significantly lower post–treatment SBP than Ramipril after Holm adjustment, while the Lisinopril–Ramipril difference was trend-level and not statistically significant after multiplicity control; Lisinopril and Moexipril did not differ significantly. A baseline balance check (ANOVA on baseline SBP: $p = 0.584$) supported the claim of successful random assignment. In summary, the three study objectives were addressed directly. First, paired $t$-tests confirmed that all three drugs significantly reduced SBP, demonstrating within-drug effectiveness. Second, between-group comparisons using ANCOVA revealed that Lisinopril and Moexipril were more effective than Ramipril in lowering post-treatment SBP, while Lisinopril and Moexipril did not differ significantly from each other. Third, a baseline balance check (ANOVA on baseline SBP, $p = 0.584$) supported the claim of successful random assignment, strengthening internal validity.

Overall, the study highlights the importance of adjusting for baseline in small experimental datasets and provides preliminary evidence favoring Lisinopril and Moexipril over Ramipril for greater SBP reduction. Given the small sample size ($n = 12$), these results should be interpreted as exploratory and confirmed in larger, covariate–rich, and longitudinal studies.

# References


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