Modeling Heart Attack Predictions With Patient Data

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Background

Heart attacks occupy a central role in the social consciousness as a commonly known medical condition with incredibly costly medical consequences as well as considerable financial implications for both the victims and their families. Therefore, our team has a powerful social motivation in exploring a data-driven approach to identifying a heart attack in cases of medical ambiguity, as well as predicting the incidence of a heart attack based on known symptoms and medical information about a given patient. We hope that our findings are applied, and improved upon, in medical applications to increase the quality of life.

In this report, we set the context of our findings as being applied to an individual for whom we have easily obtainable medical data – basic information such as age and sex, as well as information that could be determined through short-term medical procedures – resting heart rate, blood sugar, and indications of angina. We further frame the data question as follows:

> *"Given some set of available medical information about a patient, are they more likely than not to experience a heart attack?"*

In our investigation, we conclude that a logistic regression model provides the most robust predictive ability with an ROC of approximately 96.6% - this performance was the top out of all other models considered, which include KNN, SVM, NN, GBM, RF, NSC.

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Inventory of Resources

Data Source: The heart attack analysis prediction dataset from Kaggle posted by Rashik Rahman in March of 2021.

Software: Rstudio version 1.4.1106, R Version 4.04 – “Lost Library Book”

Terminology

1. Angina – a condition characterized by severe chest pain that spreads to the shoulders and arms caused by inadequate blood supply to the heart.
2. Resting Blood Pressure – titularly, refers to blood pressure when not doing any physical activity. Pressure below 120/80mm Hg is considered normal.

Objectives & Success Criteria

The primary goal of this project is a classification problem of predicting the incidence of heart attack where class 0 refers to no incidence and class 1 refers to an incidence of heart attack. In this instance, we focus on optimizing ROC and place equal weight on false positive and false negative results, as both result in medical treatment that may create suboptimal results. While we understand that the consequences of both are incommensurate, we lack the medical foreground to calculate the risks non-arbitrarily.

Exploratory Data Analysis

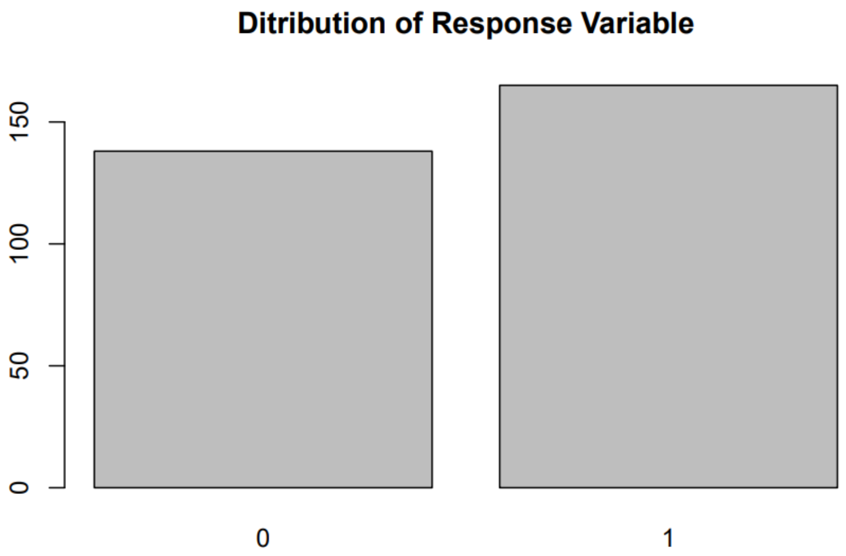
Our dataset consists of n=303 observations with a predictor space of 13 variables stored inside a single CSV file along with a response variable. The following descriptions were salvaged from the context provider by the dataset author:

Field	**Description**	**Type**
Output	Response where 1=heart attack	Nominal
Age	Age of patient	Ratio
Sex	0=Female, 1=Male	Nominal
CP	Chest Pain (Angina) 1=typical 2=atypical 3=non-anginal pain 4=asymptomatic	Nominal
Trtbps	Resting Blood Pressure	Ratio
Chol	Cholesterol levels	Ratio
Fbs	1 = fasting blood sugar > 120mg/dl	Nominal
Restecg	Resting electrocardiographic results: 0 = normal 1 = abnormal 2 = hypertrophy	Nominal
Thalachh	Maximum heart rate achieved	Ratio
Exng	1 = exercise induced angina	Nominal
Oldpeak	Previous peak – no context.	Ratio
Slp	Slope – no context.	Interval
Caa	Number of major vessels	Interval

Thal	“thal rate” – no context provided.	Discrete Ratio

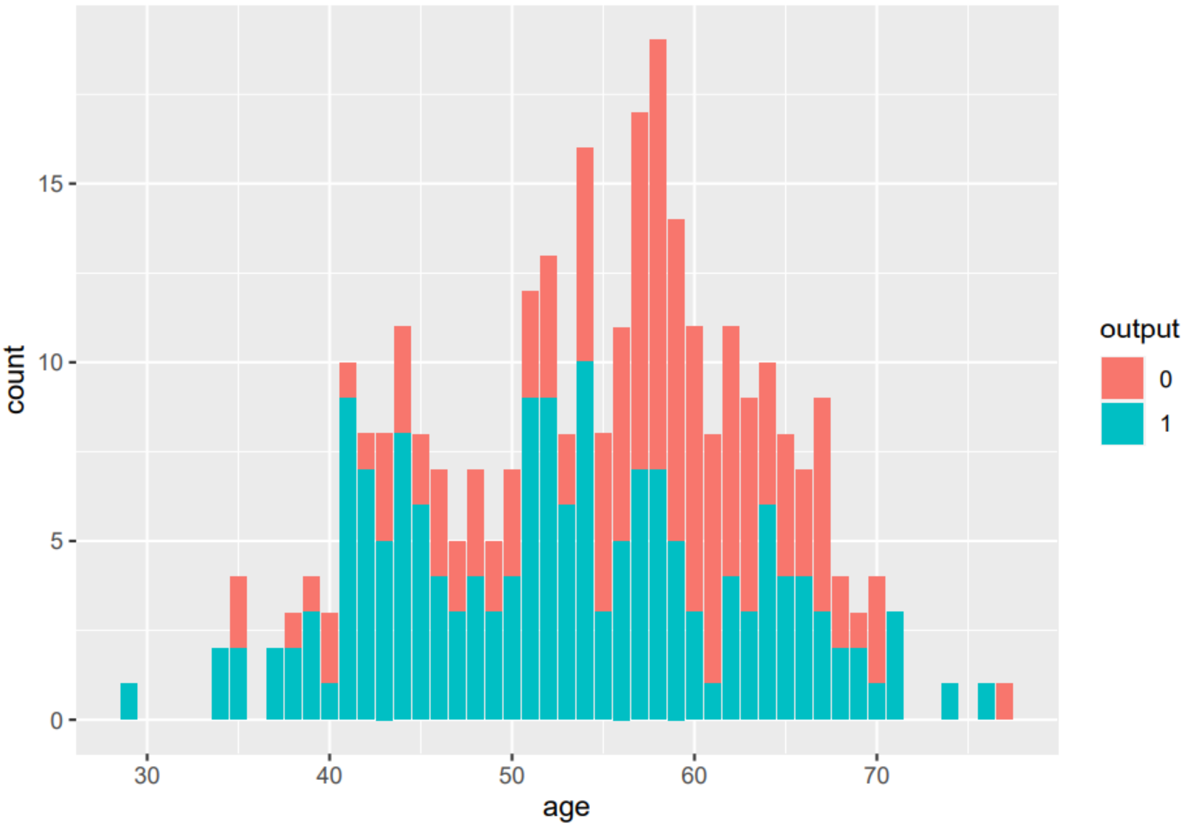
Data Description/ Observations

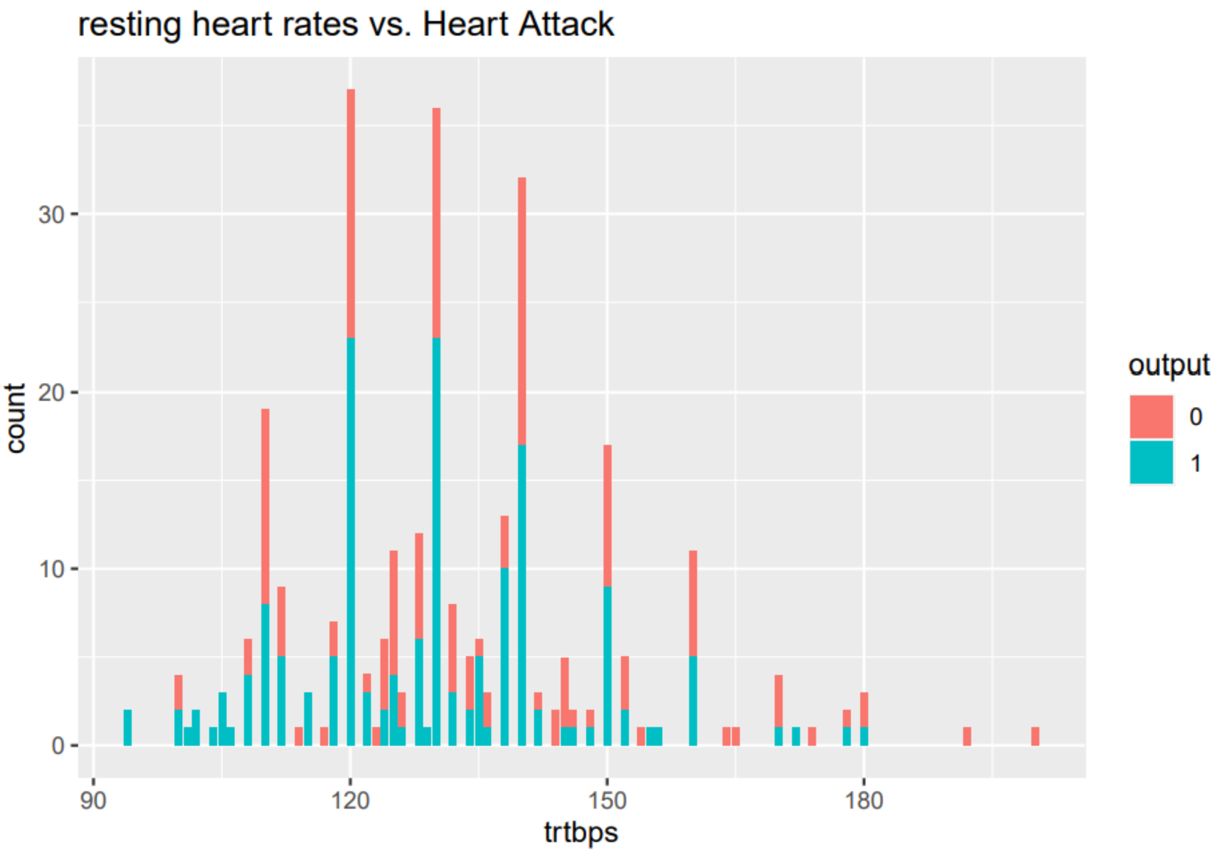
We begin by noting the absence of any missing values in our data, which makes cleaning and pre-processing considerably easier. We also find a near-perfect class balance, which allows us to sample randomly and without stratification:



Moving to analysing our predictor space, we find no significant correlations in our continuous space, and can begin observing the relationships between predictors and the response variable.

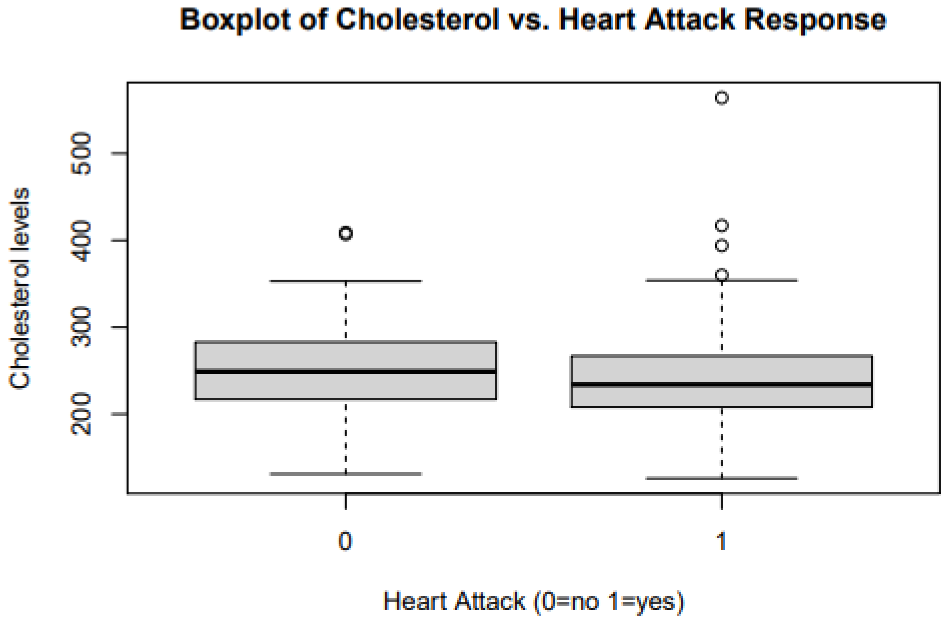
To begin with, we find a notable pattern in age, wherein lower ages are more disproportionately affected by the incidence of heart attack:


count
15
10
5
0
30
40
50
60
70
age
output
0
1



When we observe this relationship for resting heart rates (trtbps), the most obvious face-value observation is that resting heart rate values smaller than 120 bps are disproportionately associated with the risk of heart attack (output=1). This finding confirms the intuitions of our team, which presupposes that lower resting heart rates are linked to a higher incidence of risk.

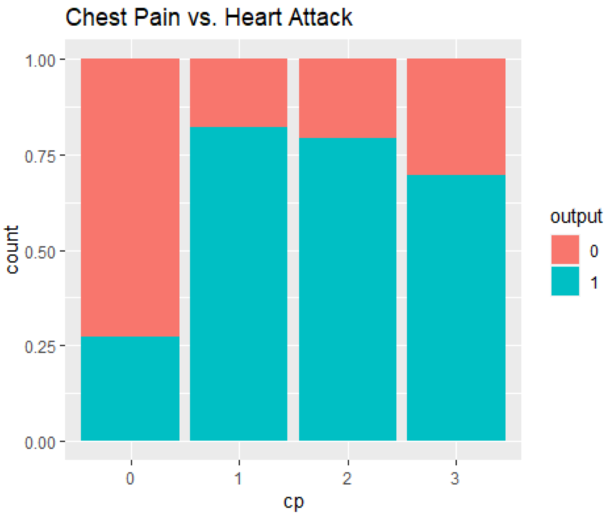
Although we preliminary suspected that cholesterol levels would have a significant bearing on heart attack risk, we found that there doesn’t seem to be a significant difference between response groups:

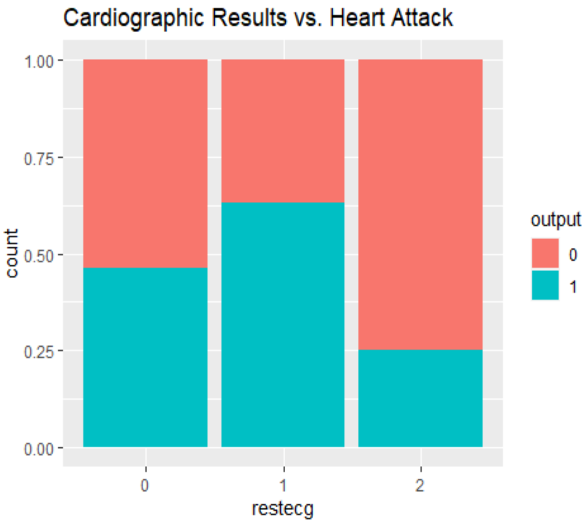


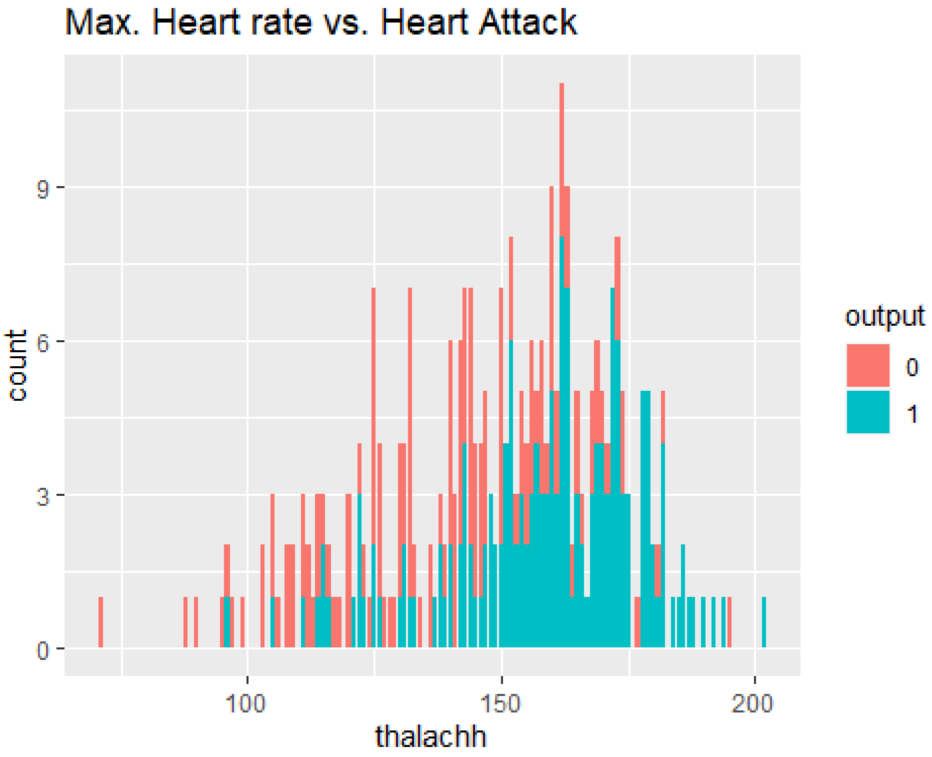
To further fortify the claim that cholesterol is not an informative predictor, a hypothesis test for the difference of means was conducted at an alpha = 0.05. Based on these findings, we do not have enough evidence to reject the null hypothesis that the difference of means between the two groups is zero. We did, however, identify a gender disparity with respect to the response variable:

	Heart Attack	No Heart Attack	**Total**
Male	114	93	207
Female	24	72	96

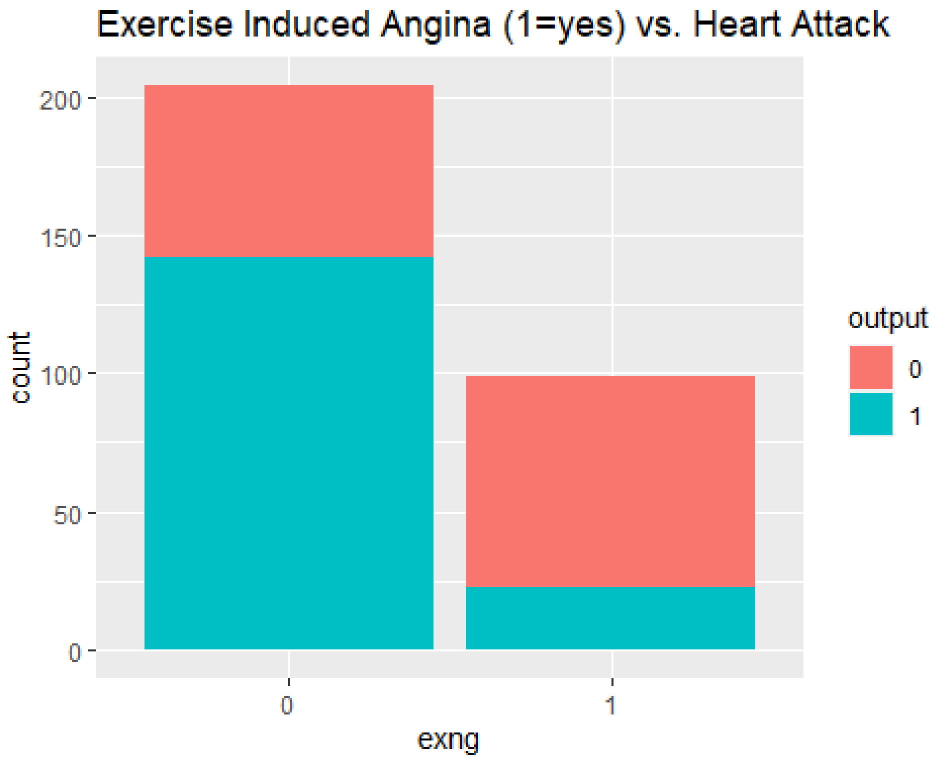
We also note the imbalance in gender between men and women, and recognize that this might pose a risk in representation, and would therefore welcome an opportunity to find more representative data. Other notable predictors that clearly demonstrate a separation between the two response groups include the incidence of chest pain (cp), cardiographic results (restecg), the maximum heart rate achieved (thalachh), the presence of exercise induced angina (exng), slope and thal, which had no interpretation per se, the number of major vessels, and fasting blood sugar:





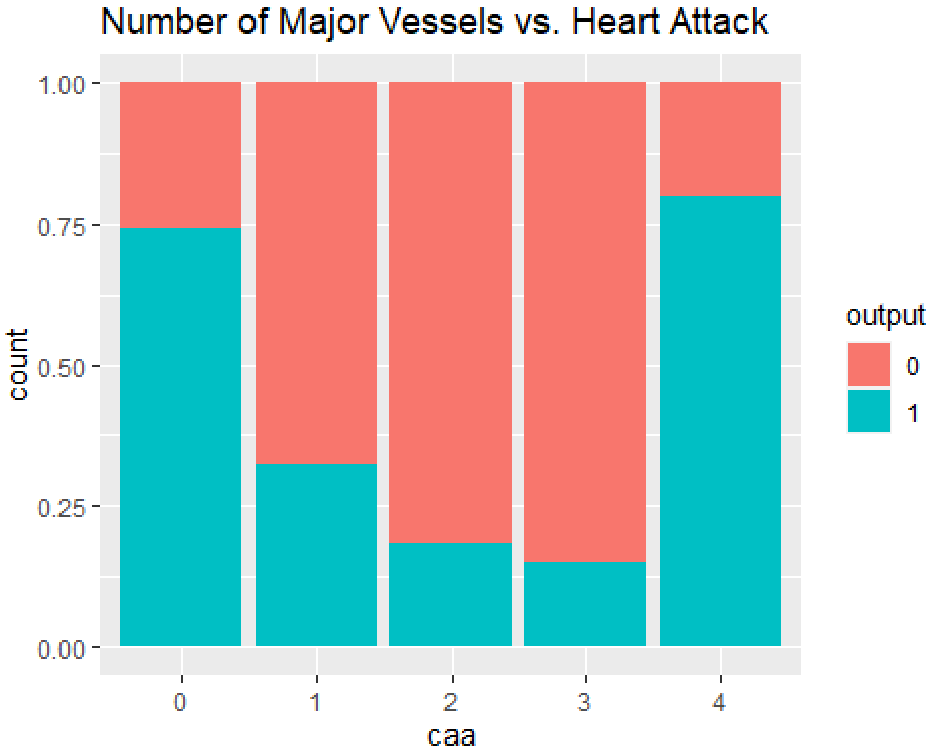


Here, we find the absence of chest pain is strongly associated with the absence of heart attack. Hypertrophy in cardiographic results is also strongly associated with a reduced risk of heart attack. Finally, we find that a higher maximum heart rate is associated with a higher incidence of heart attack, which was further confirmed by a hypothesis test at alpha=0.05, suggesting that we have enough evidence to reject the null hypothesis that the difference of means between the two response groups is zero.



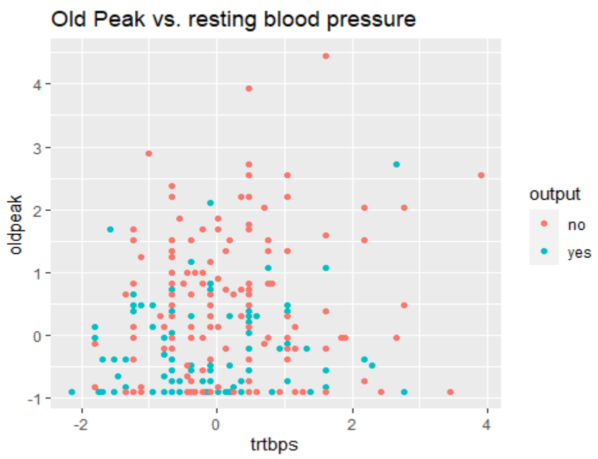
Curiously, we observe that individuals who suffered exercise induced angina are less likely to contract heart disease. From an intuitive point of view, the argument could be made that exercise decreases the risk of heart attack in many instances, and so the angina experienced by patients isn’t associated with a heart condition.

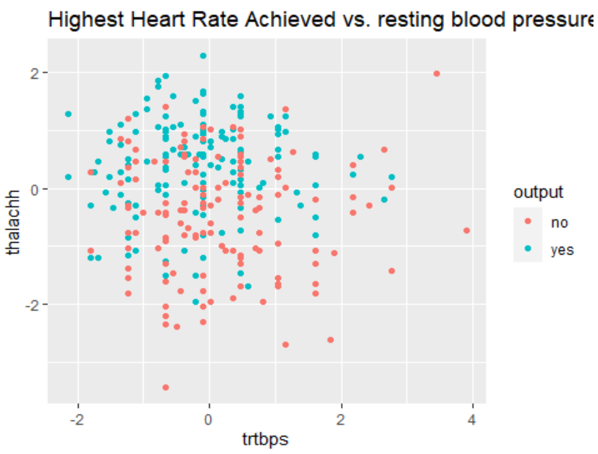
Finally, we find that individuals with 0 and 4 major vessels are considerably more associated with the risk of heart attack than all other values in between:

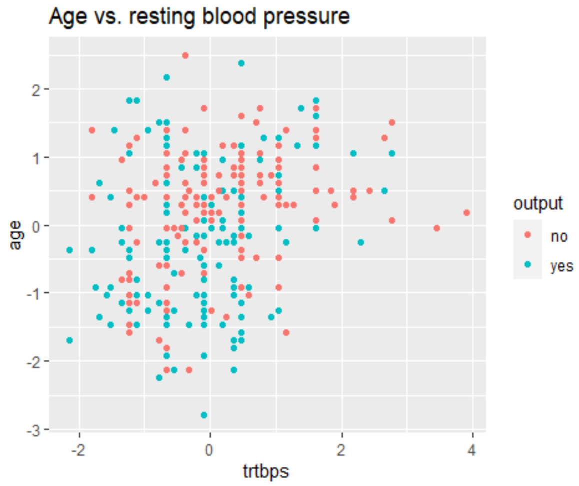


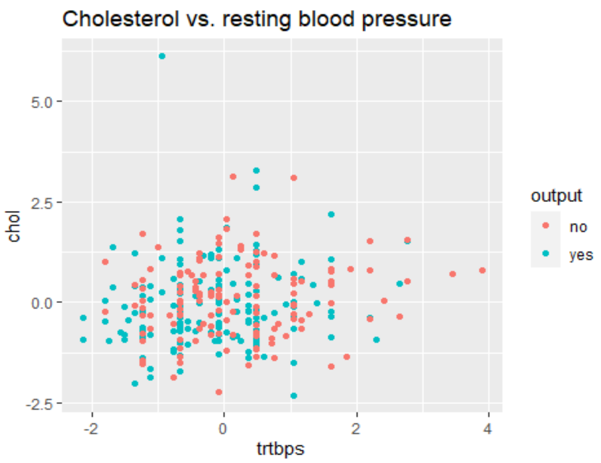
For this report, we choose not to visualize slope or thal, as we lack the interpretation for either of these variables, but still choose to include them during the modelling process.

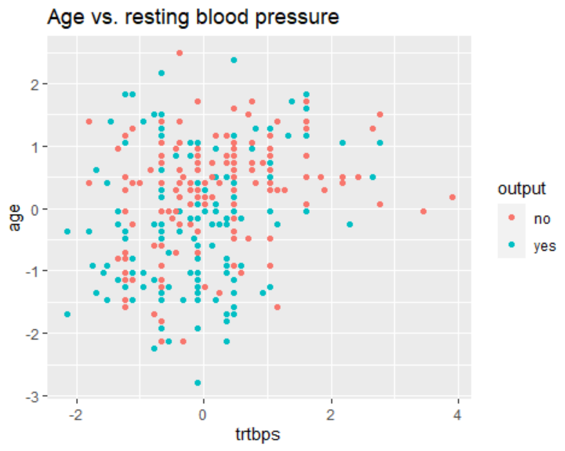
The last portion of EDA includes a discussion of whether or not this problem is linearly separable- as a preliminary attempt to understand the data environment, we visualize our continuous variables and attempt to find face-value evidence that the problem might be separated by a line in a two dimensional space:











Based on a preliminary assessment of the data, it's unclear that there's an opportunity to create a linear separation in the continuous predictor space, and so we're unlikely to use LDA or any other linear separators.

Data Pre-processing and Splitting

For the modelling portion of our investigation, predictors were scaled and centered. For the Neural Net portion of the modelling process, a spatial sign transformation was applied to continuous predictors where applicable. Since no missing values exist in the set, interpolation was not required. Data was split into a 90%-10% training/testing set due to the fact that n vastly outpaces the size of the predictor space. Of 303 samples, ~270 were used for the training set.

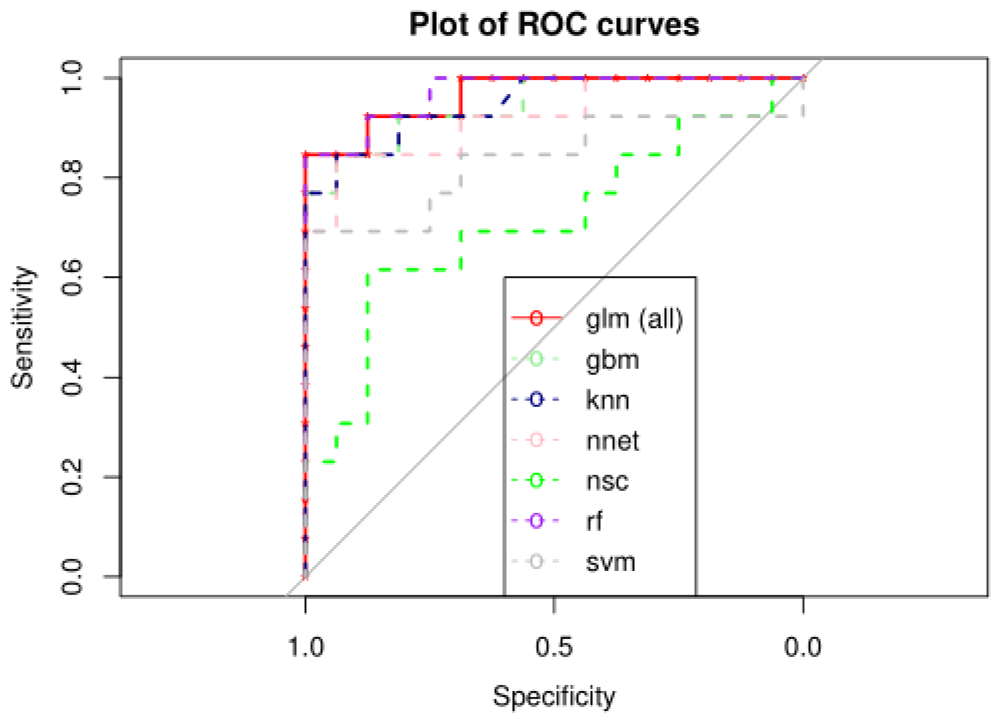
Modelling Strategy

Given that we have a combination of categorical and continuous data, one of the challenges we encountered during the process of creating a model building strategy was that some R packages were resistant to training a combination of both variables. For instance, while logistic regression / glm did end up outperforming all other models, the **train()** function from the caret package was harder to apply during the model building process than the regular glm() function. Because of this, we fielded a combination of tree-based models such as random forest and gbm, regression models like glm, and models of varying complexity such as SVM, Neural Networks, Nearest Shrunken Centroids, and K-Nearest Neighbours. By offering a broad range of classification models that all take fundamentally different approaches to the data problem, we hope to find a balance between ROC maximization and parsimony.

Model Performance and Hyperparameter Tuning

Of the seven models that were tested (KNN, NSC, SVM, NN, GLM, GBM, and RF), our neural net model performed the best, followed closely by random forest and gbm. Glm, svm, nsc, and knn all had below-90% ROC, of which KNN performed the best.

Model	ROC	Params	Model	ROC	Params
GLM	0.9663	NA	KNN	0.9495	K=40
GBM	0.9471	Trees=1000 depth =1, shrinkage=0.01, minobs=30	NNET	0.9231	Size=5, decay=1
NSC	0.7115	Thresh=0	RF	0.9712	Mtry=2
SVM	0.8365	Sigma=.054 C=.0625			



Final Model Selection / Results

In this instance, we find that Random Forest provides a robust ROC performance (+97%), which is comparable to the performance of the most parsimonious model, the logistic regression model. We observe the following variable importance values for RF below:

Variable	Overall
age	58.92492
sex	5.67629
cp	100
trtbps	44.00965
ochol	49.39511
fbs	10.1194
restecg	0
thalachh	90.27633
exng	22.08506
oldpeak	82.97035
slp	27.32064
caa	93.07395
thall	93.66576

Although we would normally consider parsimony a driving factor (ie model complexity) in deciding which model to move forward with, we make a special caveat for the given application, which is determining the incidence of a heart attack. In this particular instance, maximizing ROC is paramount because of the high social cost of misclassification in either direction – failing to identify a heart attack could result in potential death, while misdiagnosing a heart attack and applying defensive treatment might result in incredible financial expenses for the patient or possibly other secondary health detriments as a result of emergency treatment. Because of this, we choose to move forward with the random forest model.

References

Rashik Rahman, (March 2021). *Heart Attack Analysis & Prediction Dataset,* Kaggle, retrieved June, 2021 from https://www.kaggle.com/rashikrahmanpritom/heart-attack-analysis-prediction-dataset

Applied Predictive Modeling - Predicting Heart Disease

Filipp Krasovsky & Rudy Fasano

6/10/2021

This report focuses on analyzing a collection of anonymized medical records (n=303) with the intent of designing a model that can accurately predict the incidence of heart disease given an array of biological factors such as age, sex, blood cholestrol, etc. The modeling question before us stands as follows: "Given some combination of medical information about a patient, is a patient more likely than not to contract heart disease?"

Data was collected from Kaggle (citation needed here) and contains a total of 303 data points with 13 predictors and a single response variable (output), which serves as a binary indicator of whether or not an individual has contracted heart disease. Because of the nature of our response variable, we have no choice but to frame the problem as a classification challenge rather than pure regression - that is, OLS and similar models are excluded, but we're still able to utilize alternative classifiers such as logistic regression, SVM, KNN, etc. As a preliminary note, our data does not have any missing values.

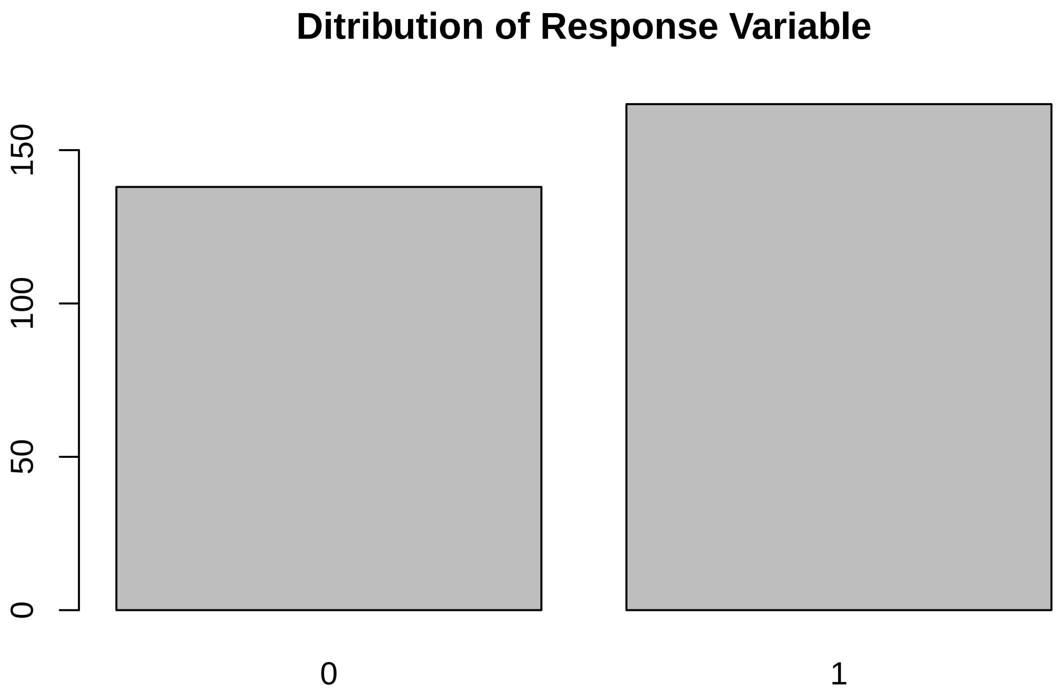
We begin with an overview of each variable's interpretation:

Age: A discrete ordinal variable which indicates the age of the patient. Sex: A binary categorical variable where 0=female and 1=male. cp: Chest Pain Type, categorical variable: 1 = typical angina, 2 = atypical angina, 3 = non-anginal pain, 4 = asymptomatic trtbps: resting blood pressure in mm Hg - continuous ratio variable. chol: cholestrol in mg/dl - continuous ratio variable fbs: a categorical variable where 1 indicates that fasting blood sugar > 120 mg/dl. restecg: resting electrocardiographic results where 0 = normal, 1 = wave abnormality, 2 = probably ventricular hypertrophy. thalachh: maximum heart rate achieved (ratio continuous variable) exng: an indicator variable where 1 indicates the presence of exercise induced angina. oldpeak: Previous Peak - a ratio continuous variable with no context available in the dataset. slp: slope - no context was available for this dataset. caa: number of major vessels. thall: the thal rate - a discrete ratio variable with no context provided.

```
#load in our dataset
require(caret)
require(pROC)
require(ggplot2)
df = read.csv('heart.csv',TRUE)
df$output = as.factor(df$output)
df$cp = as.factor(df$cp)
df$exng = as.factor(df$exng)
df$thall = as.factor(df$thall)
df$caa = as.factor(df$caa)
df$restecg = as.factor(df$restecg)
df$sex = as.factor(df$sex)
df$fbs = as.factor(df$sex)
df$slp = as.factor(df$slp)
df$caa = as.factor(df$caa)
```

We begin by noting that there is no class imbalance in our dataset:

```
#plot a histogram of the response variable
plot(df$output,main="Ditribution of Response Variable")
```

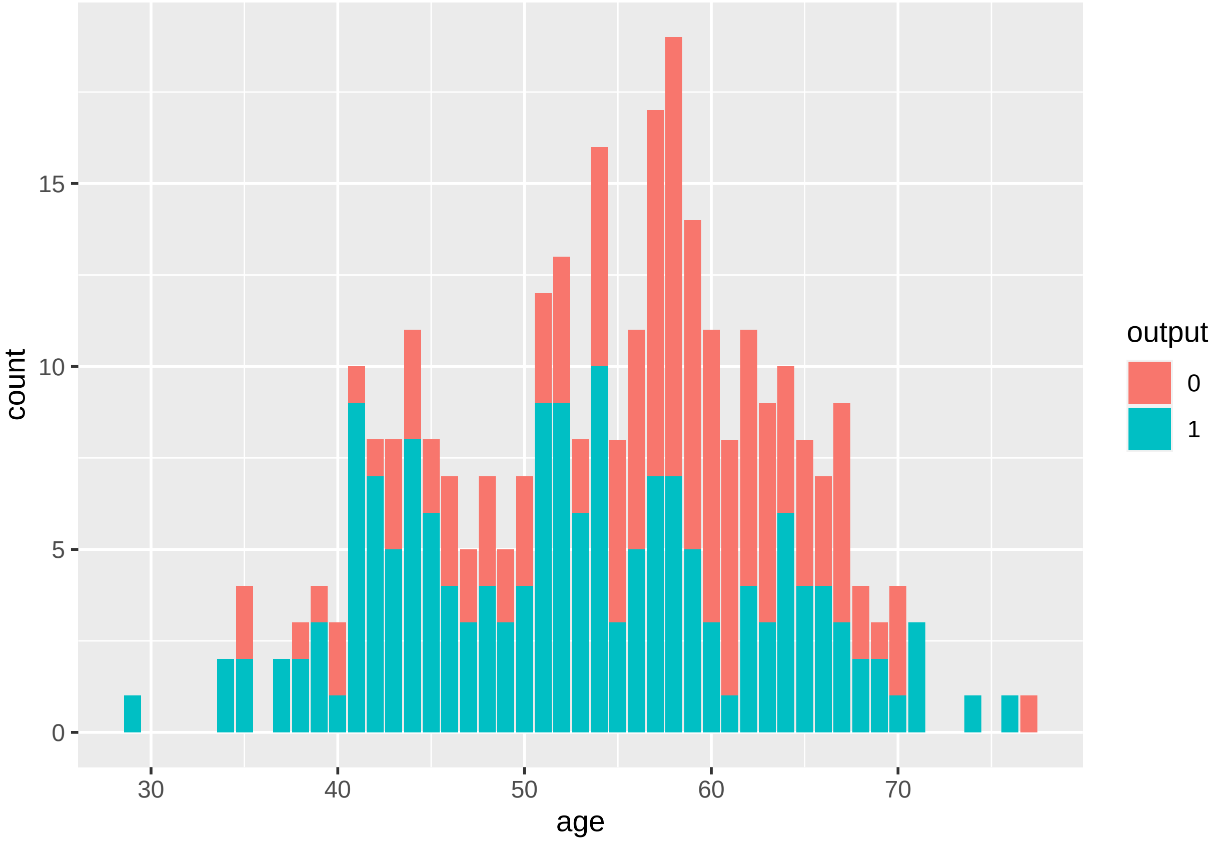


```
#print distribution by percentage
print((table(df$output)/nrow(df))*100)
```

```
##
##        0        1
## 45.54455 54.45545
```

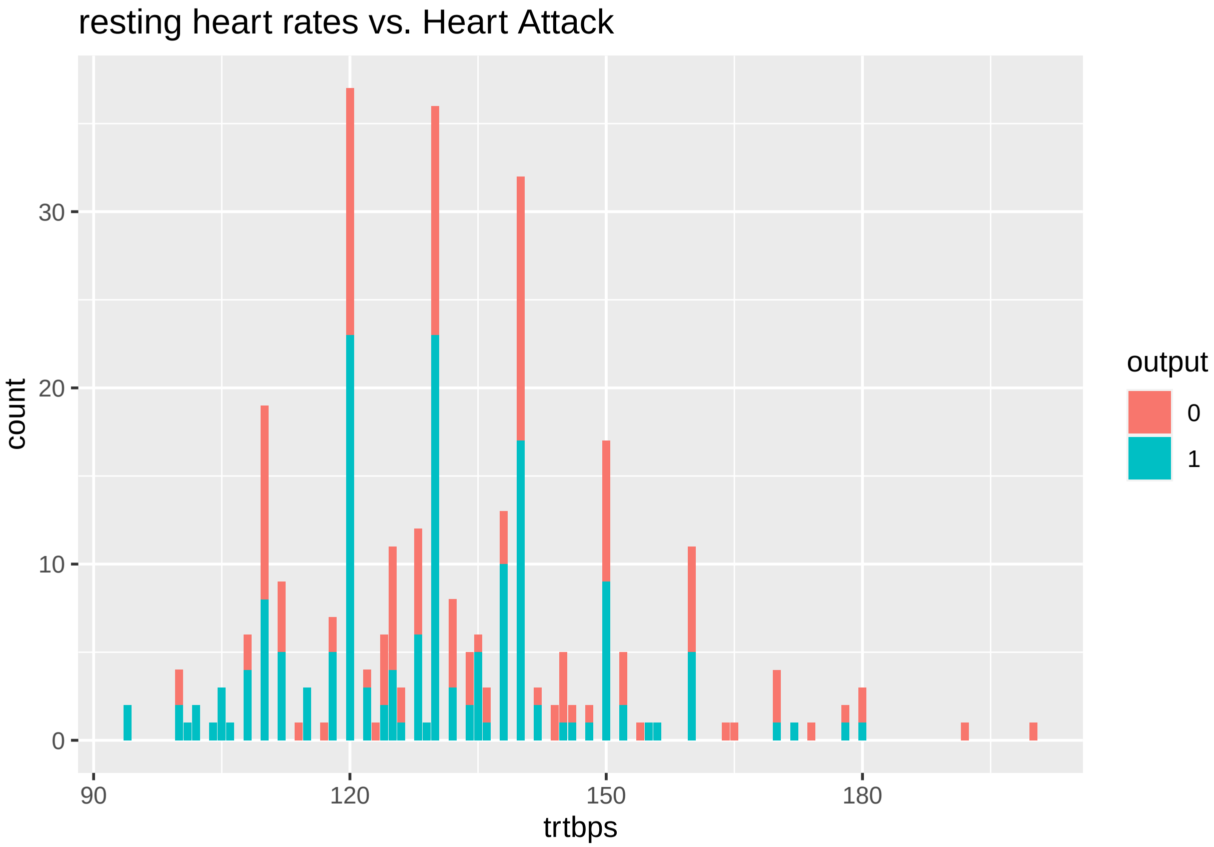
As demonstrated, we have a low class imbalance and probably will not need any sampling methods that require us to balance classes across samples, which allows us to move straight into k-fold cross validation, bagging, and bootstrapping. Of our predictor space, five variables are non-categorical, so we can proceed by examining their distributions relative to the response variable as well as any possible between-predictor relationships:

```
ggplot(df,aes(x=age,group=output,fill=output))+geom_bar()
```



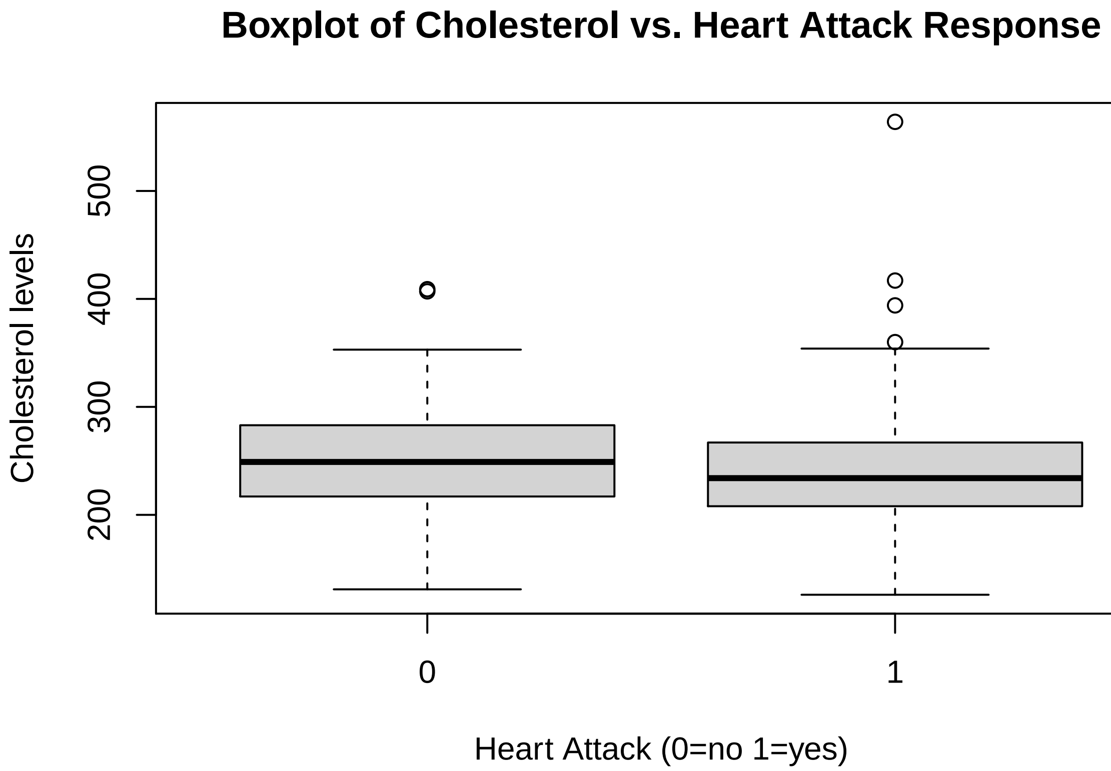
Starting with age, we observe that that the incidence of heart dominates samples with ages <= 55, while being more or less balanced for larger age values. When we observe this relationship for resting heart rates (trtbps), the most obvious face-value observation is that resting heart rate values smaller than 120 bps are disproportionately associated with the risk of heart attack (output=1):

```
ggplot(df,aes(x=trtbps,group=output,fill=output))+geom_bar(stat = 'count')+ggtitle("resting heart rates
```



Another intuitive hypothesis is that higher rates of cholesterol are strongly associated with a higher incidence of heart attack. For this examination, we can use a box and whisker plot. Our findings suggest that those samples with an incidence of heart attack had a smaller average cholesterol value but more outliers gravitating towards the higher end of cholesterol levels.

```
plot(df$output,df$chol,main="Boxplot of Cholesterol vs. Heart Attack Response",xlab="Heart Attack (0=no
```



However, given the motivation to explore this relationship further, we can consider a t-test to determine if the mean cholesterol levels are significantly different between those who did and did not have a heart attack:

```
response.1 <- which(df$output==1)
x1 = df[response.1,]$chol
x2 = df[-response.1,]$chol
#conduct a t test for difference of means
xtest = t.test(x1,x2)
print(xtest$p.value)
```

```
## [1] 0.1360182
```

Therefore, at a significance level of alpha = 0.05 that we do not have enough evidence to reject the null hypothesis that the difference in average cholesterol levels between patients who did and did not have a heart attack is zero.

Notable findings about the imbalance of heart attack patients, however, include the gender disparity. We first begin by observing the imbalance in data between men and women (0=women, 1=men):

```
tb.sex = (as.table(table(df$sex,df$output)))
print(tb.sex)
```

```
##
##        0   1
##   0   24  72
##   1  114  93
```

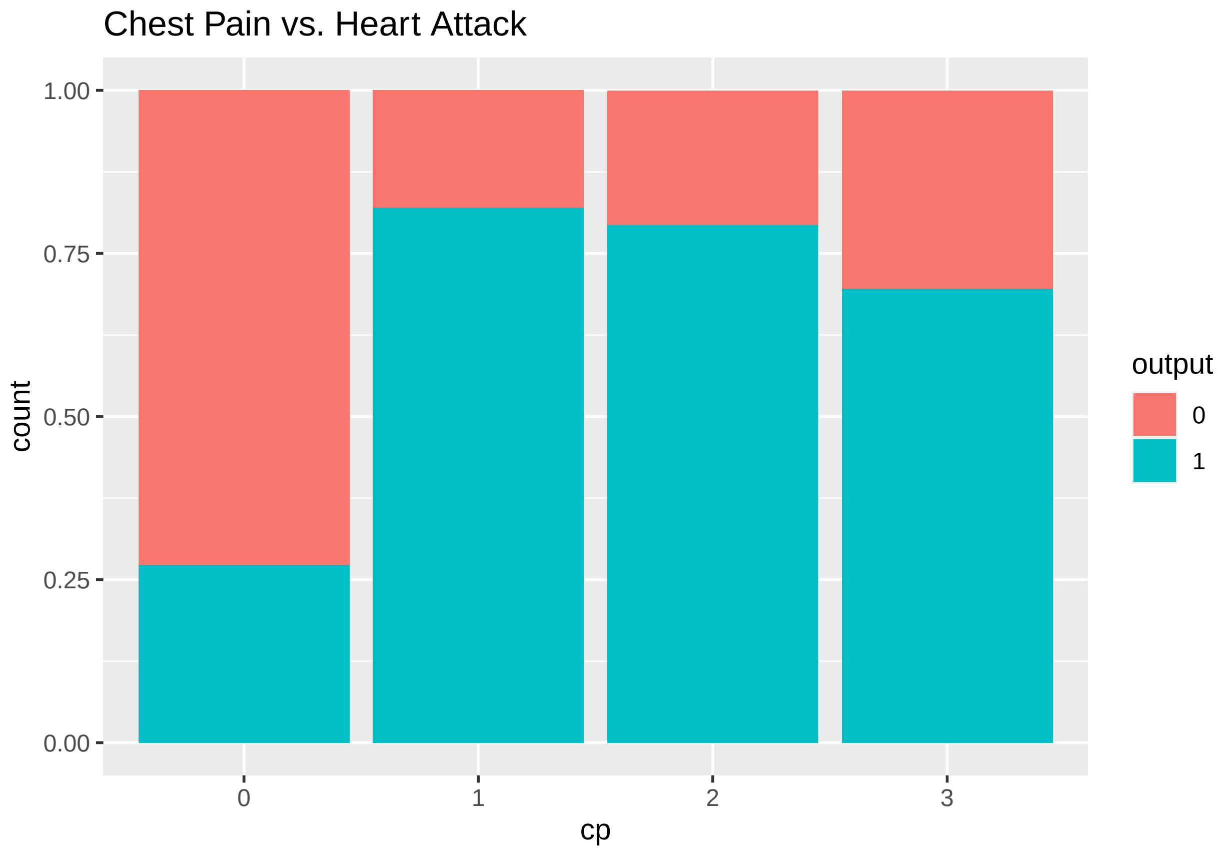
We find that while there are 96 female sample points, there are 207 male points, more than twice the number of female participants. Notwithstanding this, we find that the ratio of women who get heart attacks is much higher than that of men:

```
print(prop.table(tb.sex,margin=1))
```

```
##
##             0         1
##   0 0.2500000 0.7500000
##   1 0.5507246 0.4492754
```

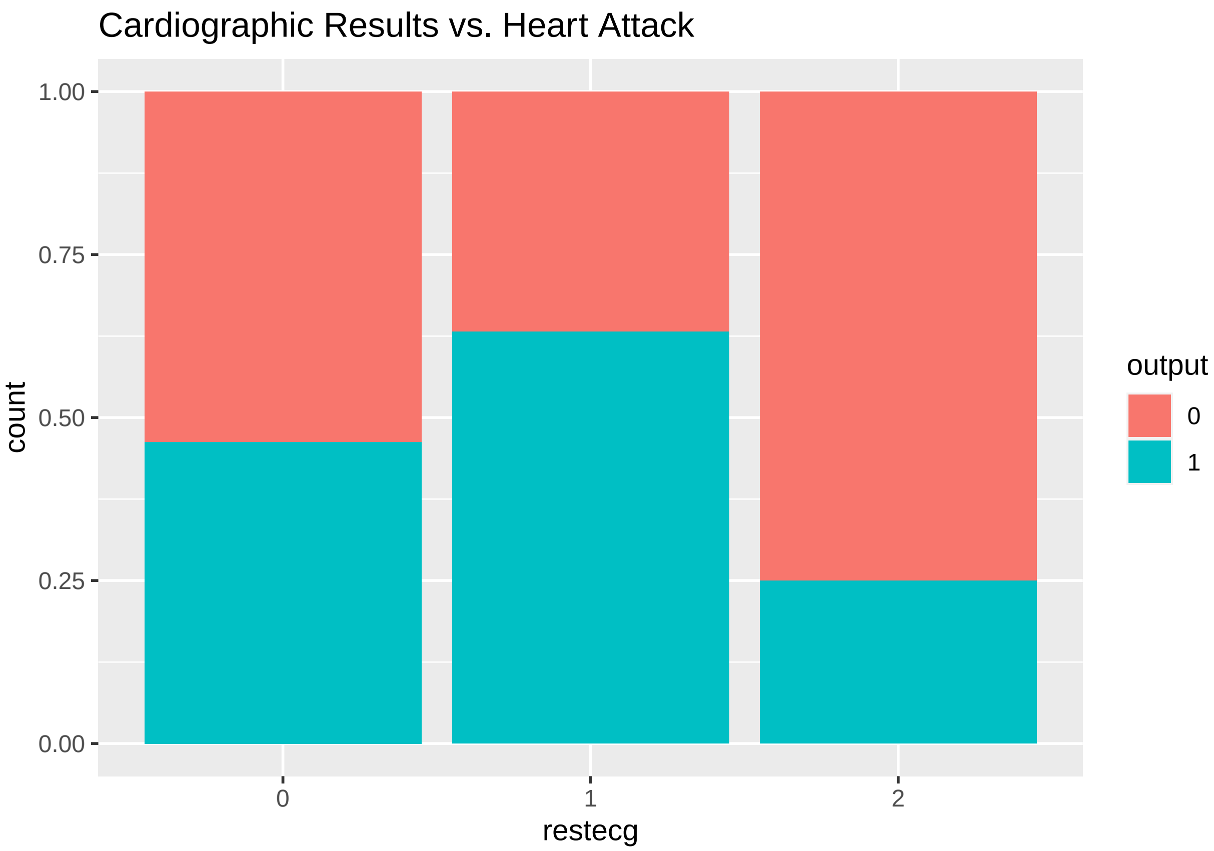
Other notable findings include the observation that heart attacks are significantly less likely for the lowest possible cp level. Although the description of this dataset labels data values 1-4 as increasing levels of angina pain, there doesn't seem to be any presence of values higher than 3. Consequently, we have to assume that the lowest level of the cp column is either the absence of angina entirely or an indicator of the presence of normal angina. Either way, the intuition still stands that non-acute/non-irregular levels of Angina (as well as the absence of angina entirely) are less likely to be associated with the incidence of a heart attack:

```
ggplot(df,aes(x=cp,group=output,fill=output))+geom_bar(stat='count', position = 'fill')+ggtitle("Chest 
```



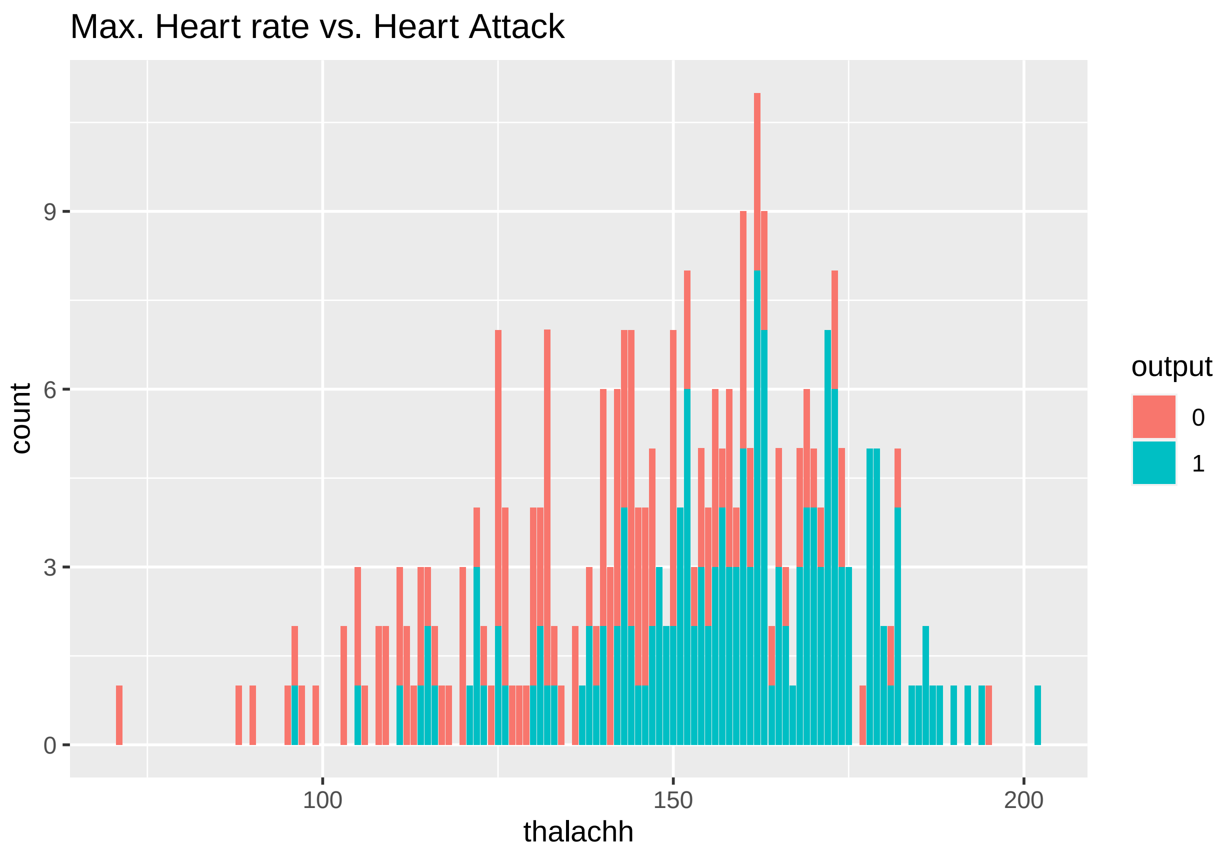
Detectiong of cardiographic results from the restecg variable suggest that the incidence of wave abnormality is significantly more associated with the risk of heart attack than normal results, and significantly moreso compared to incidences of ventricular hypertrophy. While this teams lacks the medical background to understand the implications of hypertrophy or wave abnormality, we can still understand that these conditions are abnormal, and so, intuitively affect heart functions.

```
ggplot(df,aes(x=restecg,group=output,fill=output))+geom_bar(stat='count', position = 'fill')+ggtitle("Ca
```



Carrying on in the same vein, our team also identified an intuitive relationship between the response variable and the thalachh variable, which corresponds to the maximum heart rate achieved. We find that higher maximum heart rates correspond more frequently to a higher incidence of heart attack:

```
ggplot(df,aes(x=thalachh,group=output,fill=output))+geom_bar()+ggtitle("Max. Heart rate vs. Heart Attack
```



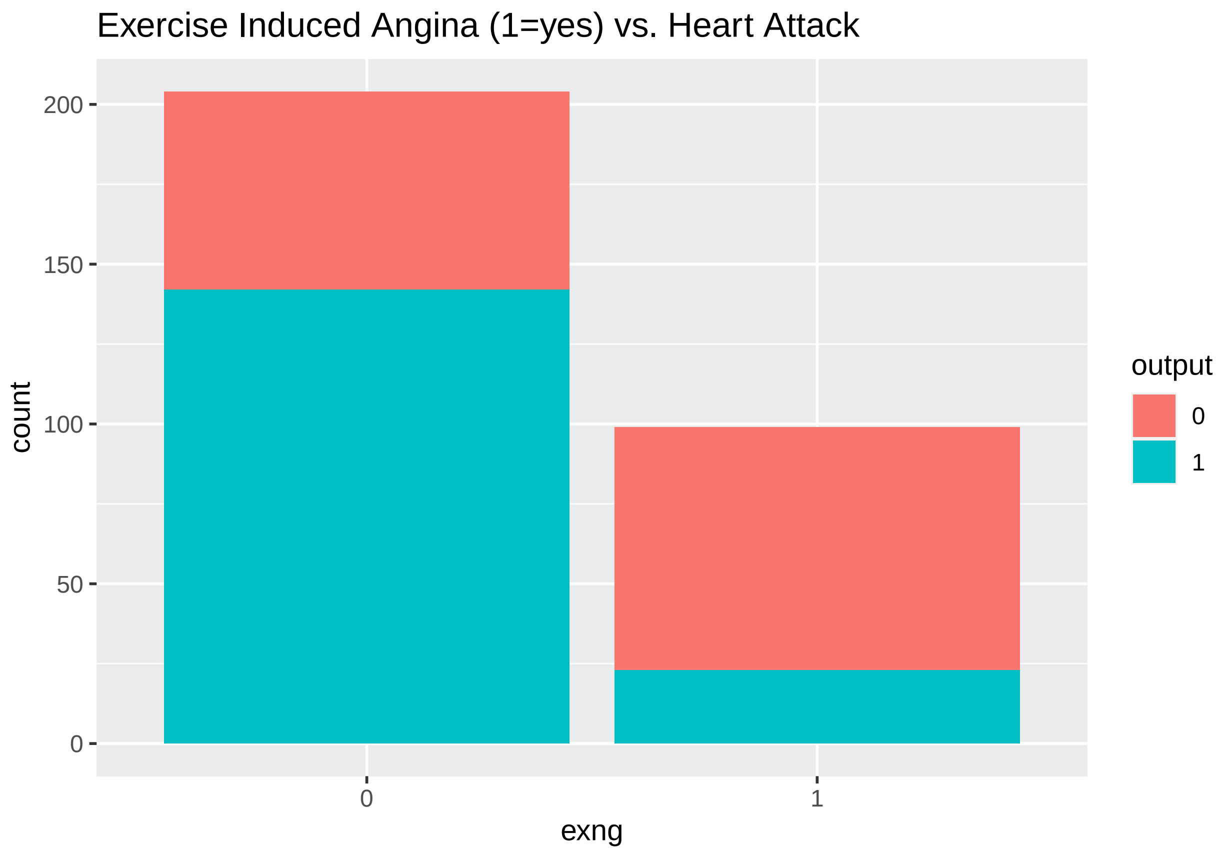
We further confirm this intuition with a t-test at a significance level of 0.05:

```
x = subset(df,output==1)$thalachh
y = subset(df,output==0)$thalachh
thaltest = t.test(x,y)
print(thaltest)
```

```
##
##  Welch Two Sample t-test
##
## data:  x and y
## t = 7.953, df = 269.9, p-value = 5.019e-14
## alternative hypothesis: true difference in means is not equal to 0
## 95 percent confidence interval:
##  14.57132 24.15912
## sample estimates:
## mean of x mean of y
##  158.4667  139.1014
```

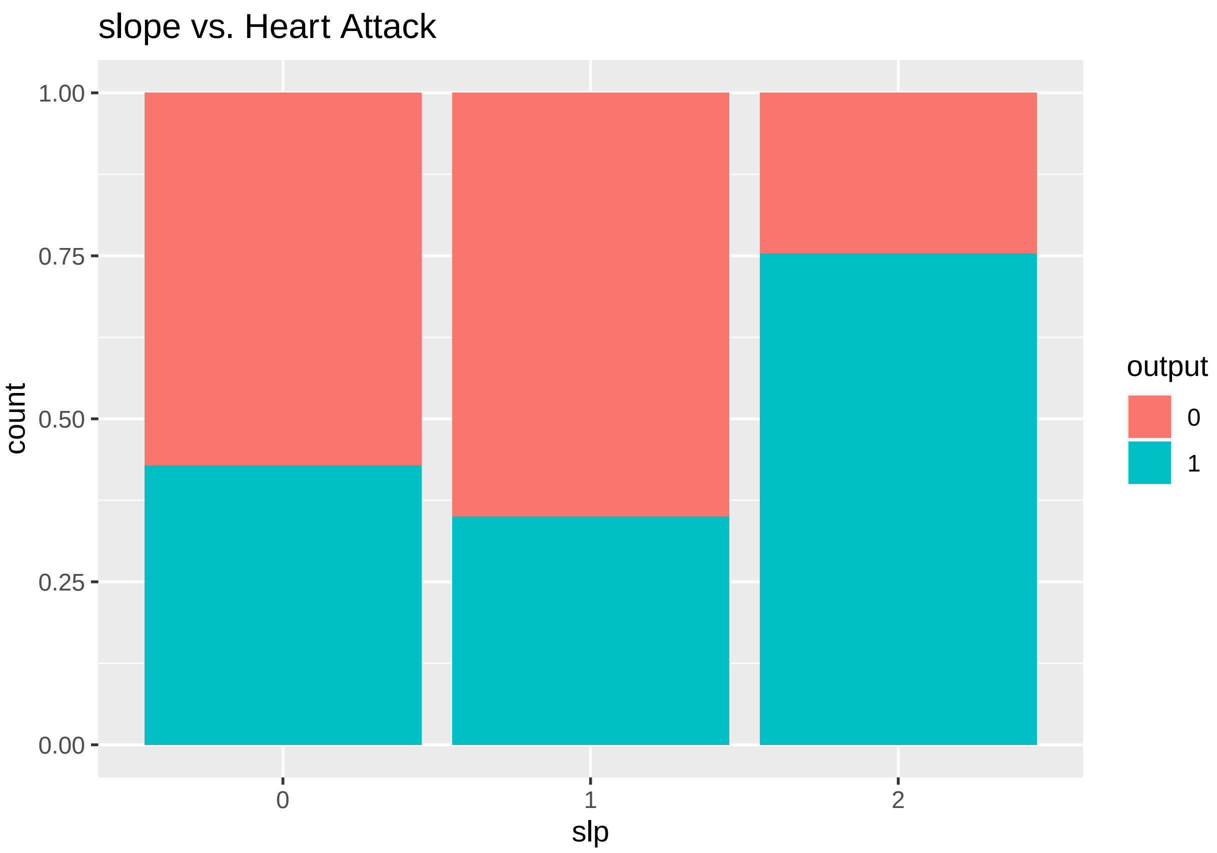
The mean max. heart rate for those who experienced a heart attack is significantly higher than their counterparts, confirming our intuition. Conversely, the data supports the claim that individuals who suffer from exercise induced angina are less likely to suffer a heart attack. While this was largely surprising during EDA, an intuitive explanation might include that individuals suffering from an exercise-induced condition may be more resistant to a heart attack based on their athletic activity:

```
ggplot(df,aes(x=exng,group=output,fill=output))+geom_bar()+ggtitle("Exercise Induced Angina (1=yes) vs.
```



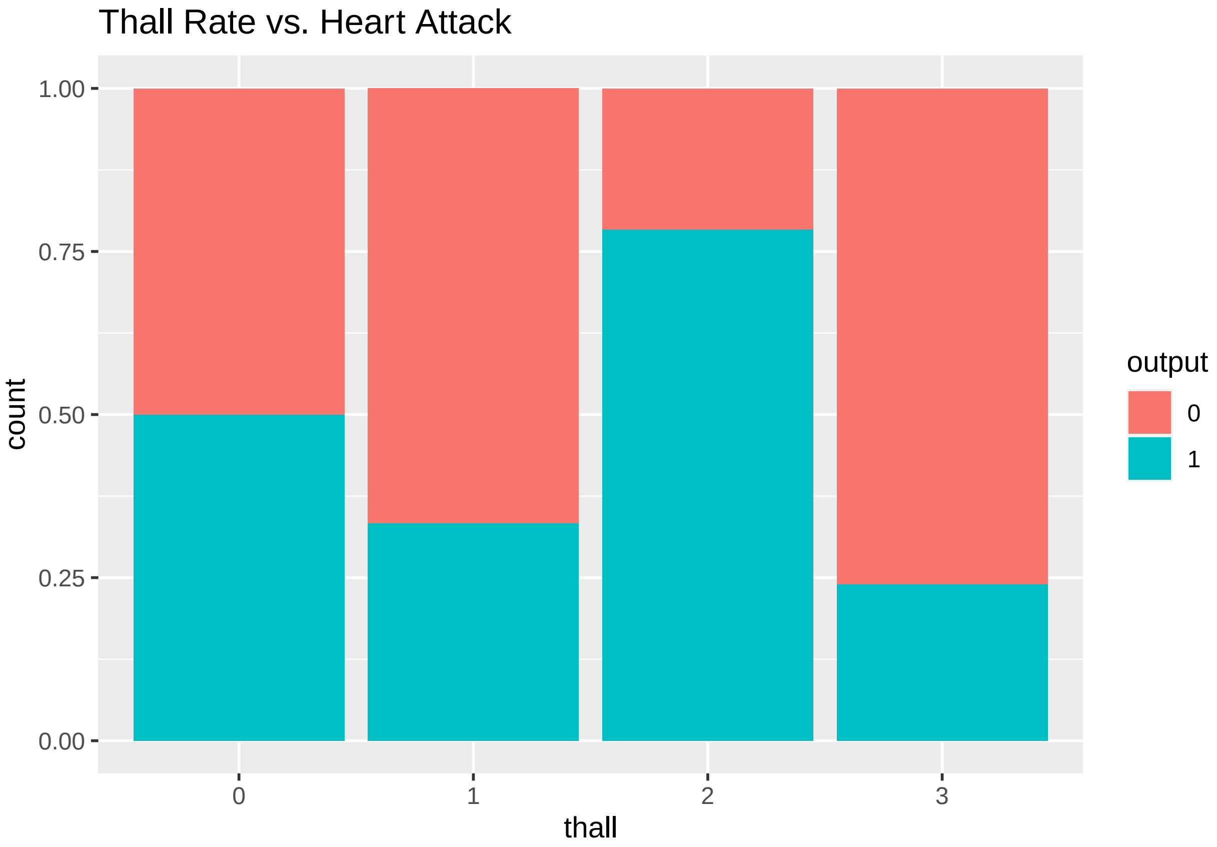
Furthermore, while we were unable to determine the context for the slope variable (slp) in the dataset, we did find that it significantly separates heart attack incidence based on its three unique values (0-2). In particular, we find that patients with a slope of 2 are significantly more likely to experience a heart attack:

```
df$slp = as.factor(df$slp)
ggplot(df,aes(x=slp,group=output,fill=output))+geom_bar(stat='count',position='fill')+ggtitle("slope vs
```



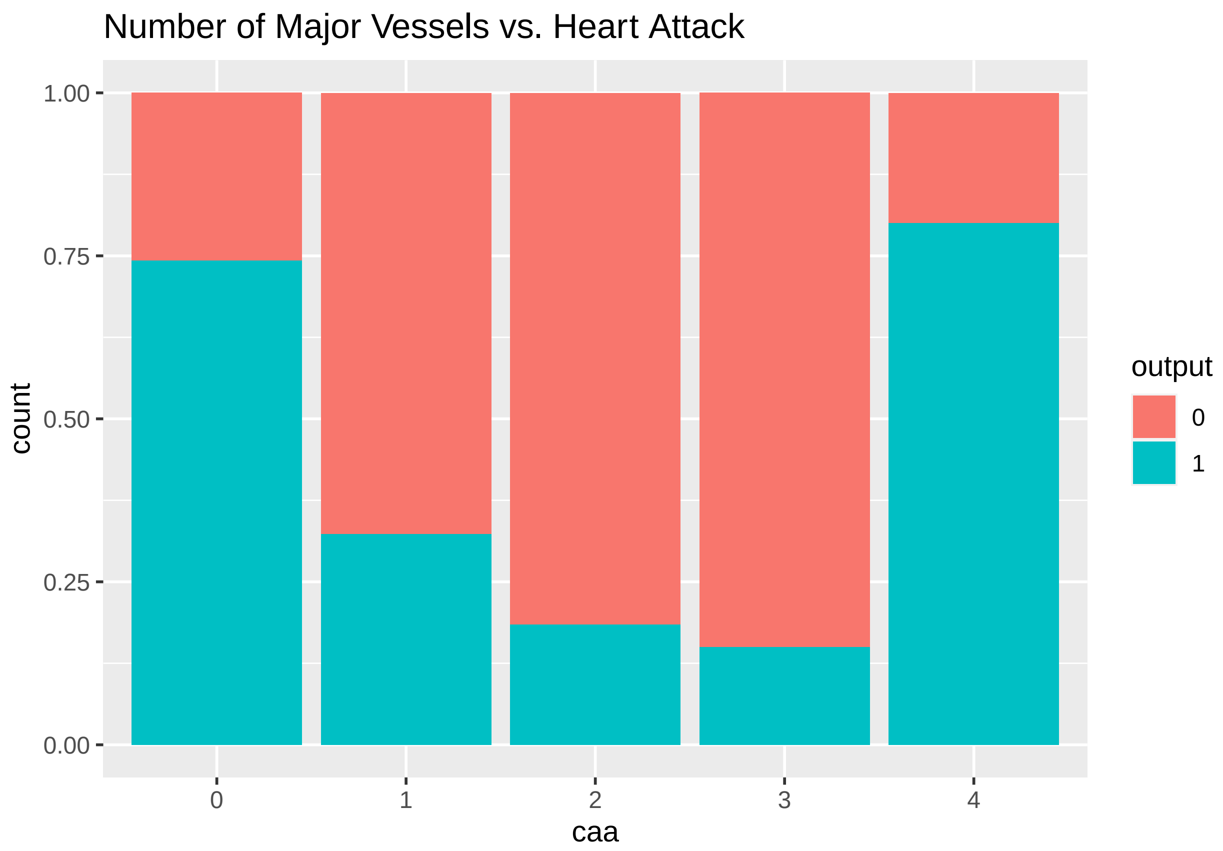
Similarly, while we found no background explanation on how to interpret the thall rate, we did find that those patients with a value of 0 and 2 had a significantly higher association with heart attack than values of 1 and 3:

```
ggplot(df,aes(x=thall,group=output,fill=output))+geom_bar(stat='count',position='fill')+ggtitle("Thall
```



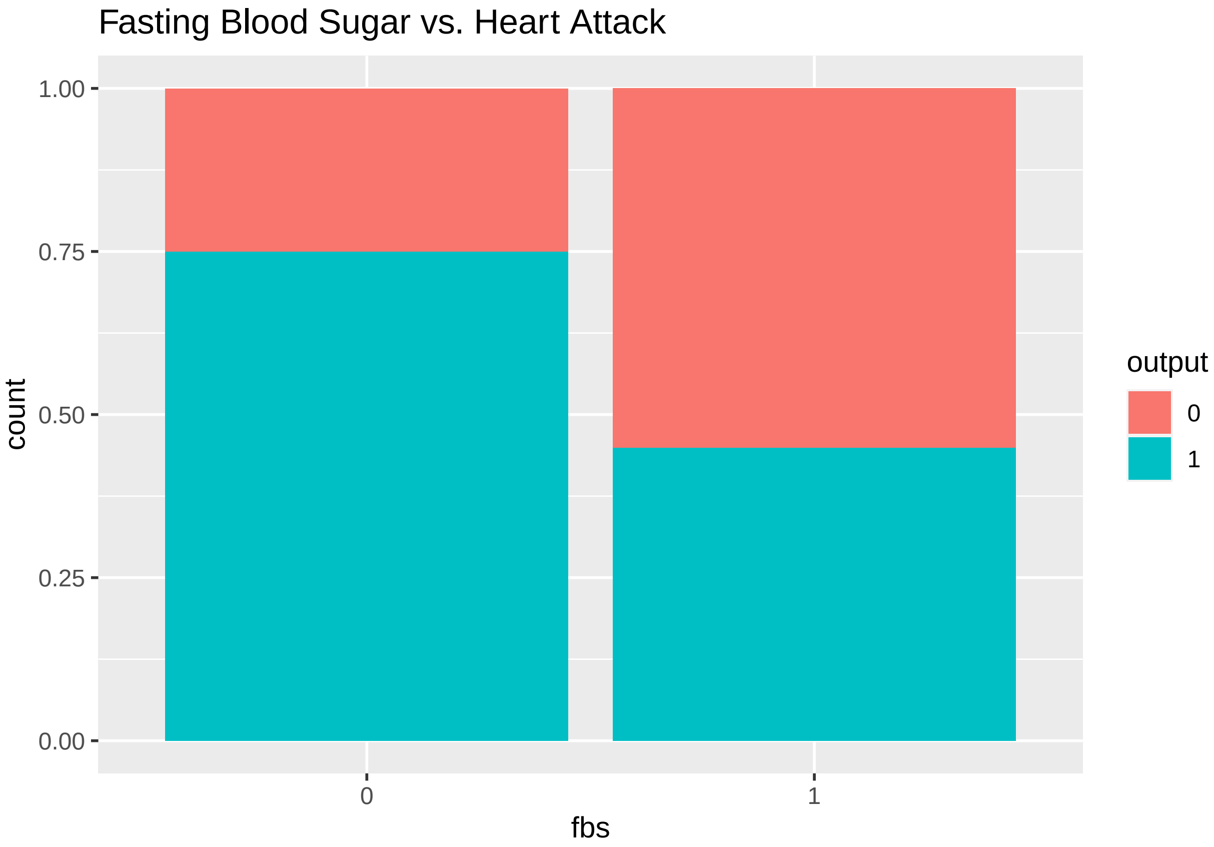
The number of major vessels also seems to have a significant effect on separating our response variable - individuals with zero and four major vessels are disproportionately associated with the incidence of heart attack:

```
ggplot(df,aes(x=caa,group=output,fill=output))+geom_bar(stat='count',position='fill')+ggtitle("Number o
```



Finally, we find that individuals with a lower resting blood sugar seem to have a higher incidence of heart attacks.

```
ggplot(df,aes(x=fbs,group=output,fill=output))+geom_bar(stat='count', position = 'fill')+ggtitle("Fastin
```



Moving forward with data pre-processing, we can center and scale our continuous predictors: age, trtbps, chol, thalachh, and oldpeak.

Because our predictor space is only 13 variables and n = 303, we can afford a 90-10 split between our training and testing set as well. We will also engage in a 10-fold cross validation for model training.

```
cont_vars = c("age","trtbps","chol","thalachh","oldpeak")
df.cont <- df[,cont_vars]
print(findCorrelation(cor(df.cont))) #no correlated predictors for our continuous variables.
```

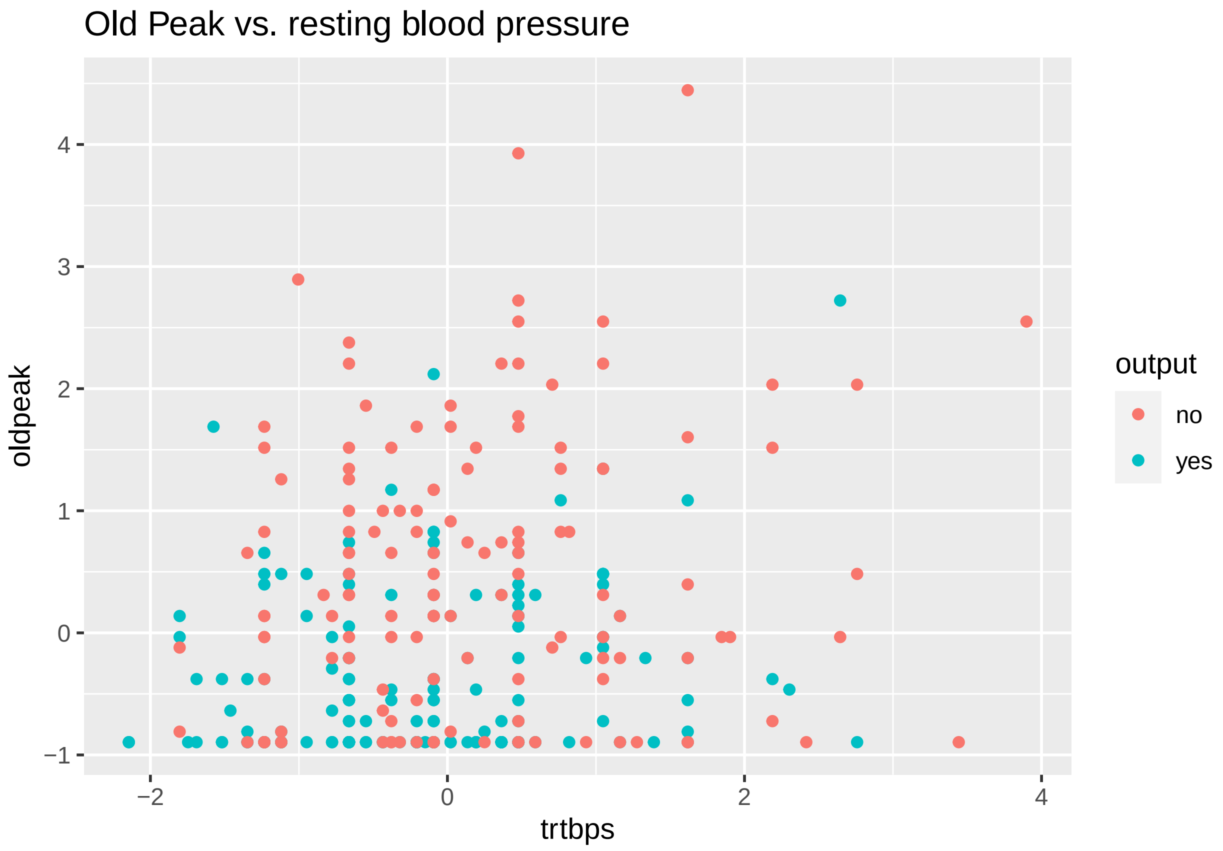
```
## integer(0)
```

```
df$output = ifelse(df$output==1,"yes","no")

#center and scale the continuous variables
df = predict(preProcess(df,method=c("center","scale")),df)

#partition 90-10
set.seed(10)
trainingRows <- createDataPartition(df$output,p=0.9,list=FALSE)

predictors = subset(df,select=-c(output))
response   = data.frame(df$output)
names(response) = "response"

predictors.train = predictors[trainingRows,]
```

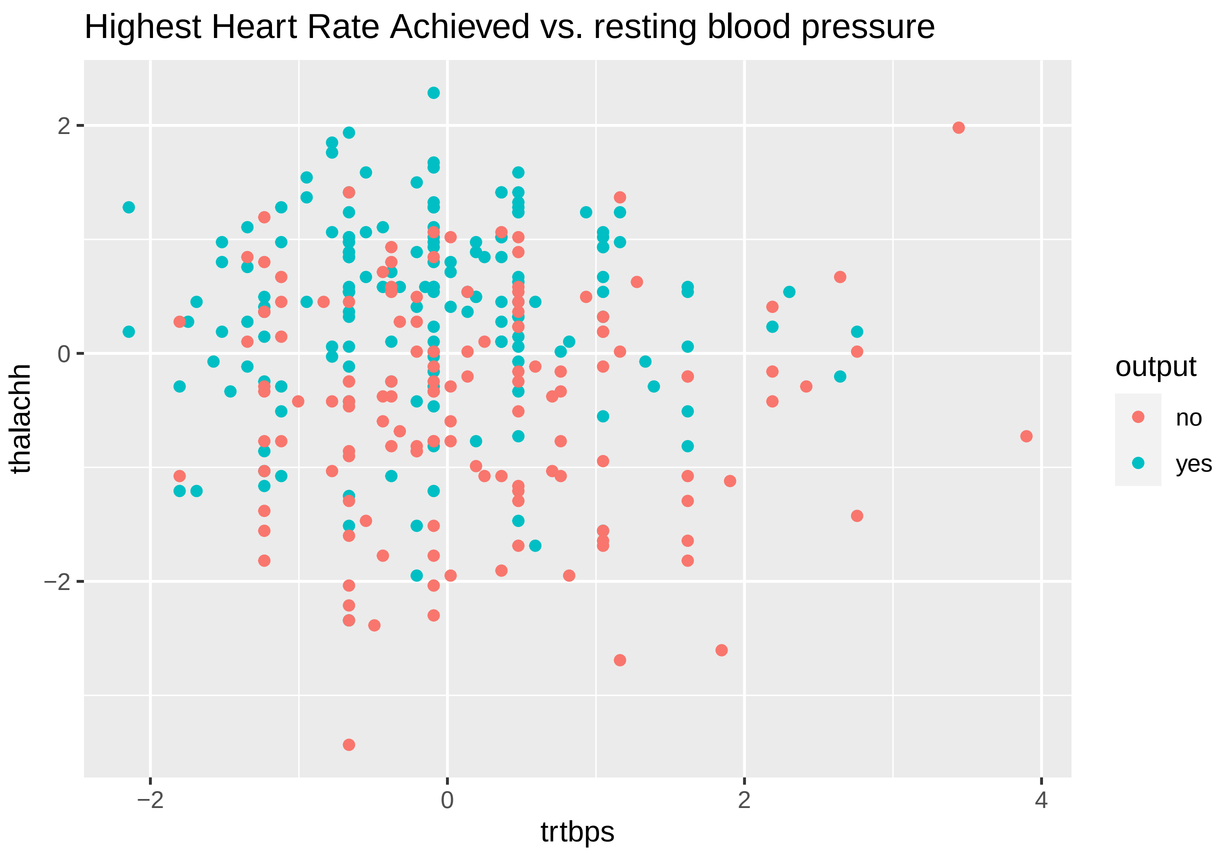
```
response.train   = response[trainingRows]

predictors.test  = predictors[-trainingRows,]
response.test    = response[-trainingRows]

reduced.train    = df.cont[trainingRows,]
reduced.test     = df.cont[-trainingRows,]
```

Because this is a classification challenge, we can consider classification models only (ie Logistc regression, QDA/LDA, Knn, etc.) We can create a hunch for whether or not the business problem is linearly separable by looking at scatter plots of our continuous data:

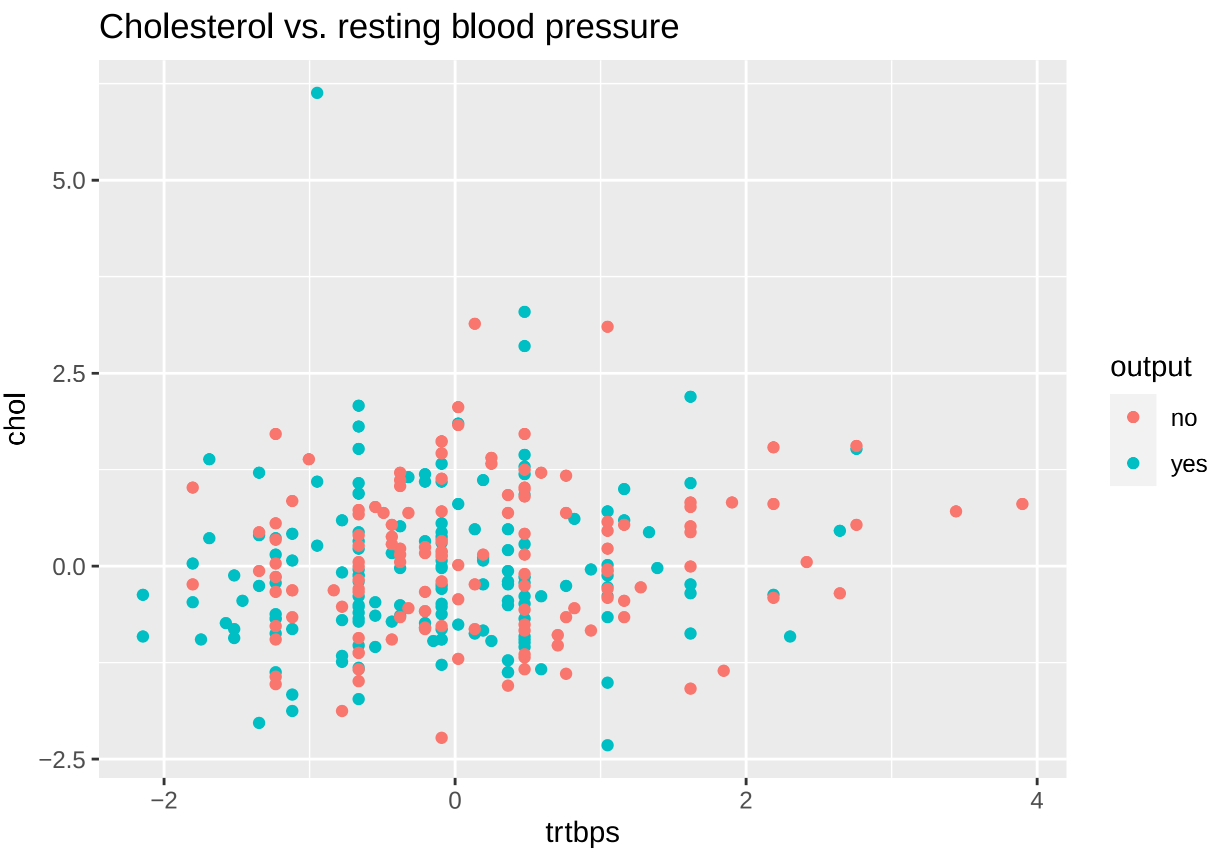
```
ggplot(df,aes(x=trtbps,y=oldpeak,group=output))+geom_point(aes(color=output))+ggtitle("Old Peak vs. res
```



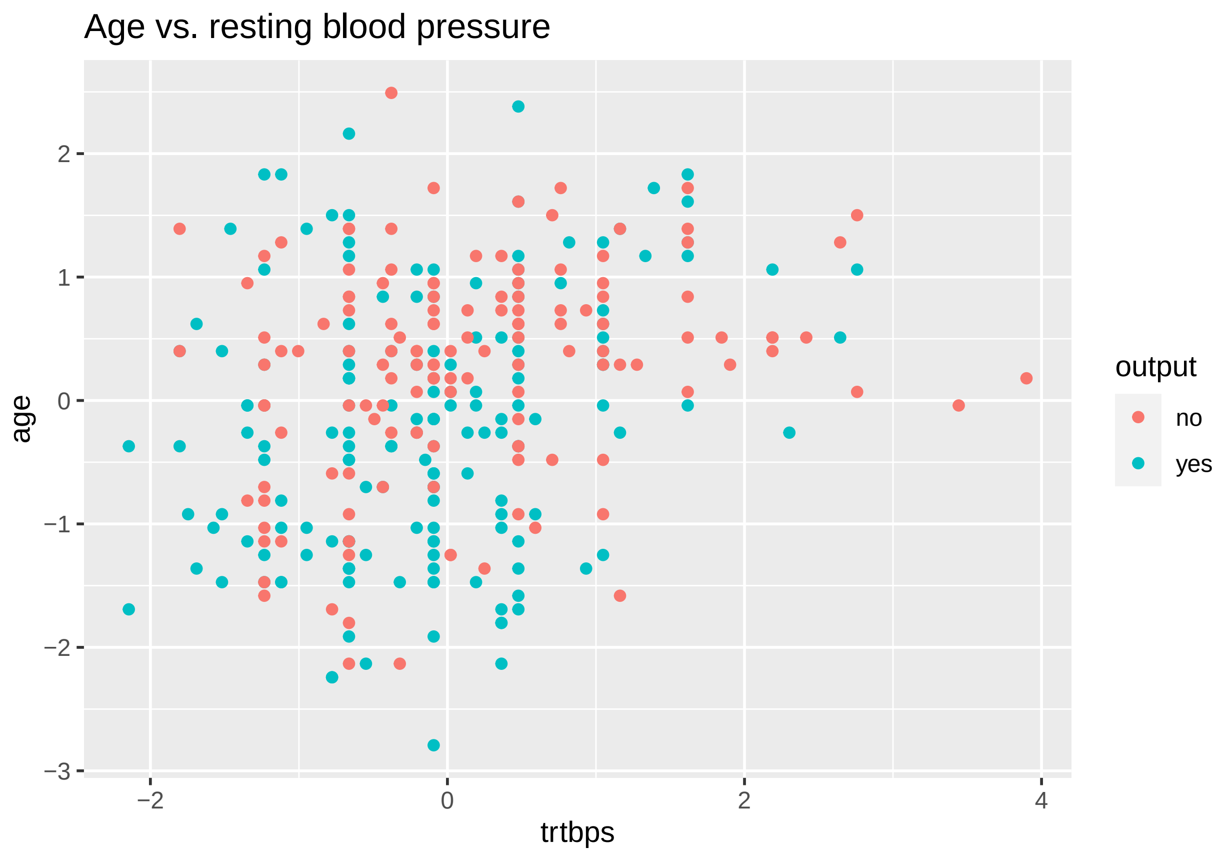
```
ggplot(df,aes(x=trtbps,y=thalachh,group=output))+geom_point(aes(color=output))+ggtitle("Highest Heart Ra
```



```
ggplot(df,aes(x=trtbps,y=chol,group=output))+geom_point(aes(color=output))+ggtitle("Cholesterol vs. res
```



```
ggplot(df,aes(x=trtbps,y=age,group=output))+geom_point(aes(color=output))+ggtitle("Age vs. resting blood
```



Based on a preliminary assessment, it seems unlikely that the problem would be linearly separable between the two response values. From here, we would favor nonlinear separation methods like logistic regression, QDA, NN, SVM, KNN, NSC, and Random Forests. Given that only five our predictors are numeric, we would have to limit the application of qda/svm/etc to only those predictors.

```
ctrl <- trainControl(summaryFunction = twoClassSummary, classProbs = TRUE)
knngrid <- data.frame(.k=c(1:40))

response.train = as.factor(response.train)
response.test =  as.factor(response.test)

#KNN
set.seed(476)
knnfit <- train(predictors.train, as.factor(response.train),
method = "knn",
metric = "ROC",
tuneGrid = knngrid,
trControl = ctrl)

knn.predict <- predict(knnfit,predictors.test,type="prob")
knn.roc <- roc(predictor = knn.predict$yes,response=response.test,levels=rev(levels(response.test)))
print(knn.roc)
```

```
##
## Call:
## roc.default(response = response.test, predictor = knn.predict$yes,     levels = rev(levels(response.
##
## Data: knn.predict$yes in 16 controls (response.test yes) > 13 cases (response.test no).
## Area under the curve: 0.9495
```

Next, we move on to our SVM model.

```
#SVM - set tuning param grid
set.seed(600)
library(kernlab)
sigmarange <- sigest(as.matrix(reduced.train))
svmgrid    <- expand.grid(.sigma = sigmarange[1],.C = 2 ** (seq(-4,4)))

#randomize, train, predict
set.seed(601)
svmfit <- train(x=reduced.train,y=response.train,method="svmRadial",tuneGrid = svmgrid,metric="ROC", fi
svm.predict <- predict(svmfit,reduced.test,type='prob')
svm.roc <-  roc(predictor = svm.predict$yes,response=response.test,levels=rev(levels(response.test)))
print(svm.roc)
```

```
##
## Call:
## roc.default(response = response.test, predictor = svm.predict$yes,     levels = rev(levels(response.
##
## Data: svm.predict$yes in 16 controls (response.test yes) > 13 cases (response.test no).
## Area under the curve: 0.8365
```

Given our heavy dependence on categorical variable, it's intuitive that our SVM model did not perform as well as expected.

```
#NSC
set.seed(529)
nscGrid <- data.frame(.threshold = 0:25)
nscTuned <- train(x = reduced.train,y = response.train,method = "pam",tuneGrid = nscGrid,metric= "ROC",
```

```
## 11111111111111111111111111
```

```
nsc.predict <- predict(nscTuned,reduced.test,type='prob')
nsc.roc <- roc(predictor=nsc.predict$yes,response=response.test,levels=rev(levels(response.test)))
print(nsc.roc)
```

```
##
## Call:
## roc.default(response = response.test, predictor = nsc.predict$yes,     levels = rev(levels(response.
##
## Data: nsc.predict$yes in 16 controls (response.test yes) > 13 cases (response.test no).
## Area under the curve: 0.7115
```

Next, we consider logistic regression both with continuous only predictors and categorical predictors.

```
#glm using only continuous vars
set.seed(289)
glmTuned <- train(x=reduced.train,y=response.train,method="glm",metric="ROC",trControl=ctrl)
glm.predict <- predict(glmTuned,reduced.test,type='prob')
glm.roc <- roc(predictor=glm.predict$yes,response=response.test,levels=rev(levels(response.test)))
print(glm.roc)
```

```
##
## Call:
## roc.default(response = response.test, predictor = glm.predict$yes,     levels = rev(levels(response.t
##
## Data: glm.predict$yes in 16 controls (response.test yes) > 13 cases (response.test no).
## Area under the curve: 0.8365
```

```
#glm using the entire set
set.seed(628)
train <- cbind(predictors.train,response.train)
train$response.train=as.factor(train$response.train)
glm2 <- glm(response.train ~ .,data=train,family=binomial)
sucProb <-1 - predict(glm2,newdata=predictors.test,type="response")
glm2.roc = roc(predictor = sucProb,response=response.test,levels=rev(levels(response.test)))
print(glm2.roc)
```

```
##
## Call:
## roc.default(response = response.test, predictor = sucProb, levels = rev(levels(response.test)))
##
## Data: sucProb in 16 controls (response.test yes) < 13 cases (response.test no).
## Area under the curve: 0.9663
```

We now explore NN:

```
#NN
set.seed(1028)
nnetgrid = expand.grid(.size = 5:10, .decay  = c(0,.1,1,2))
maxsize = max(nnetgrid$.size)
numwts   = (maxsize *(length(predictors.train)+1) + maxsize + 1)
nnetfit = train(x=predictors.train,y=response.train,method="nnet",metric="ROC",preProcess = c("spatialSi

#predict, calculate roc
nnetfit.pred = predict(nnetfit,predictors.test,type='prob')
nnetfit.roc = roc(predictor=nnetfit.pred$yes,response=response.test,levels=rev(levels(response.test)))
print(nnetfit.roc)
```

```
##
## Call:
## roc.default(response = response.test, predictor = nnetfit.pred$yes,     levels = rev(levels(response
##
## Data: nnetfit.pred$yes in 16 controls (response.test yes) > 13 cases (response.test no).
## Area under the curve: 0.9231
```

Our Neural Net also performs very well, but faces technical limitations in an R setting. We now consider RF implementation:

```
#RF
set.seed(972)
library(randomForest)
rfmodel<- train(x=predictors.train,y=response.train,method="rf",metric="ROC",trace=FALSE,trControl=ctrl
rfpredict <- predict(rfmodel,predictors.test,type='prob')
rf.roc <- roc(predictor=rfpredict$yes,response=response.test,levels=rev(levels(response.test)))
print(rf.roc)
```

```
##
## Call:
## roc.default(response = response.test, predictor = rfpredict$yes,     levels = rev(levels(response.te
##
## Data: rfpredict$yes in 16 controls (response.test yes) > 13 cases (response.test no).
## Area under the curve: 0.9712
```

Along the same vein of decision tree use, we can also implement gbm:

```
#Boosted trees
set.seed(2055)
gbmGrid <- expand.grid(.interaction.depth = seq(1, 7, by = 2), .n.trees = seq(100, 1000, by = 100), .sh
gbmTune <- train(predictors.train, response.train, method = "gbm", metric="ROC",tuneGrid = gbmGrid, ver
gbm.predict = predict(gbmTune,predictors.test,type='prob')
gbm.roc <- roc(predictor=gbm.predict$yes,response=response.test,levels=rev(levels(response.test)))
print(gbm.roc)
```

```
##
## Call:
## roc.default(response = response.test, predictor = gbm.predict$yes,     levels = rev(levels(response.
##
## Data: gbm.predict$yes in 16 controls (response.test yes) > 13 cases (response.test no).
## Area under the curve: 0.9471
```

Finally, we print the optimal tuning for each model:

```
#print tuning params for each model except glm
print(gbmTune$bestTune)
```

```
##    n.trees interaction.depth shrinkage n.minobsinnode
## 10    1000                 1      0.01             30
```

```
print(knnfit$bestTune)
```

```
##     k
## 40 40
```

```
print(nnetfit$bestTune)
```

```
##   size decay
## 3    5     1
```

```
print(nscTuned$bestTune)
```

```
##   threshold
## 1         0
```

```
print(rfmodel$bestTune)
```

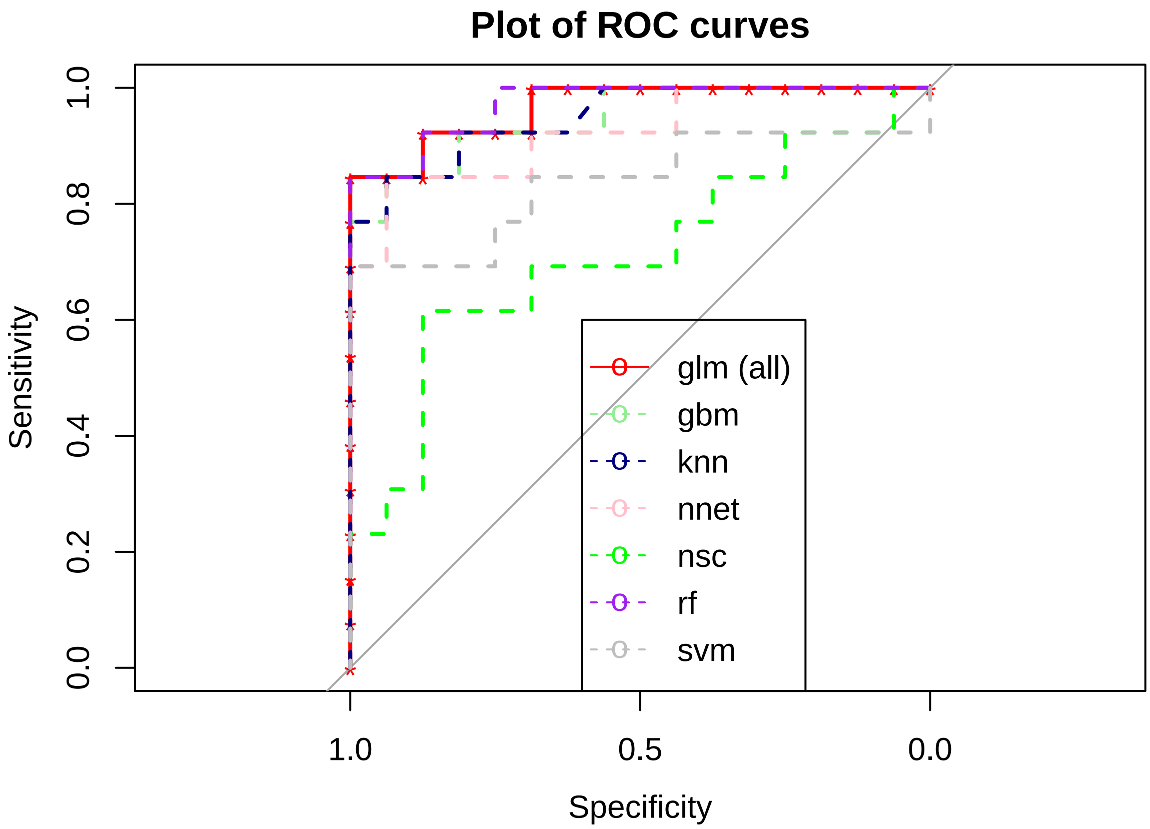
```
##   mtry
## 1    2
```

```
print(svmfit$bestTune)
```

```
##       sigma      C
## 1 0.0541278 0.0625
```

We plot the ROC curves for our models together to determine which model best satisfied our ROC expectations:

```
#plot ROC curves for each model
plot(glm2.roc,col='red',main="Plot of ROC curves",lty=1,type="o",pch="*")
lines(gbm.roc,col='lightgreen',lty=2)
lines(knn.roc,col='navyblue',lty=2)
lines(nnetfit.roc,col='pink',lty=2)
lines(nsc.roc,col='green',lty=2)
lines(rf.roc,col='purple',lty=2)
lines(svm.roc,col='gray',lty=2)
legend(0.6,0.6,legend=c("glm (all)","gbm","knn","nnet","nsc","rf","svm"), col=c("red","lightgreen","navy
```


Plot of ROC curves
Sensitivity
0.0
0.2
0.4
0.6
0.8
1.0
Specificity
1.0
0.5
0.0
glm (all)
gbm
knn
nnet
nsc
rf
svm