Editorial

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Blood Pressure for Different Types of Patients

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Editorial

Hypertension is directly correlated with the different blood pressures. In practice, both the blood pressure and hypertension are a compound medical conditions. These two diseases are highly correlated, and they practically coexist [1,2]. Approximately, 30% of the adult human being are affected with hypertension [3]. Hypertension is directly correlated with stroke for 54%, and 47% of ischaemic cardiac disease [1,3]. Mainly pharmacotherapy is used to manage hypertension. There are numerous drugs available in the market, but the proper active rates of any specific drug are nearly 50%. It is well-known that one out of three hypertension patients have their controlled blood pressure (BP) to a specific target value [4]. Uncontrolled BP is due to many factors like as lifestyle, sleep apnoea, biochemical parameters, dietary habits, family history, etc. Also the intra-individual BP variation and the genetic impacts have a high effect on the drug response [5,6].

There are different types of blood pressures, namely, basal, systolic, diastolic, maximum, mean arterial pressure, and simply blood pressure. This report seeks answers of the following: Are the determinants of all these BP are same? Are the determinants of any specific BP are identical to every type of patients? What is the correlation of a determinant with the respective BP? Is there any relation among the different blood pressures? A little is known about the answers of these questions. These answers are examined here based on three different types of patients. The patients are the cardiac patients who underwent dobutamine stress echocardiography (DSE), shocked patients, and the chronic kidney patients.

In order to identify the BP determinants, earlier research articles have used some statistical techniques namely, *Chi-square* test, Z-test, odds ratio, logistic regression, regression analysis, classification and regression tree analysis, analysis of variance, etc. [7-11], which are not appropriate for the BP data sets [12,13]. Some earlier BP articles have treated the dependent variable as continuous with constant variance or dichotomous. But in practice, the dependent variable may be continuous with non-constant variance. Most of the articles have considered only the mean model based on constant variance. These analyses have derived erroneous findings [13-15]. Thus, the earlier findings welcome many debates and doubts. BP, a positive characteristic data set is mainly analyzed either by the gamma or log-normal model [12,16,17]. The variance of a positive data set is generally non-constant due its relationship with the mean. In this report, the answers are examined based on both the gamma and log-normal models [16], and the results of the better mode are presented herein.

First, dobutamine stress echocardiography (DSE) data set with 31 variables on 558 subjects (who underwent DSE) have been analyzed. DSE data set description, its collection, and patient population are given in ref [7]. The DSE study factors/variables are basal blood pressure (BBP), basal heart rate (HR) (BHR), double product (DP) of BBP and BHR (BDP), systolic BP (SBP), peak HR (PHR), DP of PHR and SBP (DPHS), dobutamine dose used (DOSE), maximum HR (MHR), percent maximum predicted HR (PMHR), maximum BP (MBP), DP of maximum DOSE and MBP (DPMDOBP), dobutamine dose at maximum double product (DOBDOSE), age (AGE), sex (SEX) (male=0, female=1), ejection fraction on dobutamine (DOBEF), baseline cardiac ejection fraction (BEF), chest pain (present (p)=0, absent (a)=1) (CHSTPN), positive stress on echocardiogram (ECOM) (p=0, a=1) (PSE), resting wall motion abnormality on ECOM (p=0, a=1) (RWMA), recent angioplasty (p=0, a=1) (NPTCA), new myocardial infraction (MI) (p=0, a=1) (NMI), death (p=0, a=1) (DEATH), recent bypass surgery (p=0, a=1) (NCABG), history of diabetes (p=0, a=1) (HODM), history of hypertension (p=0, a=1) (HOHT), history of MI (p=0, a=1) (HOMI), history of smoking (no=0, medium=1, high=2) (HOCIG), history of coronary artery bypass surgery (p=0, a=1) (HOCABG), history of angioplasty (p=0, a=1) (HOPTCA), baseline electrocardiogram diagnosis (normal=0, equivocal=1, MI=2) (ECG), death, NMI, NPTCA or NCABG (death=0, no=1) (EVENT). This data set contains basal, systolic and maximum blood pressures, and each of them is separately considered as the dependent variable, and the remaining others are considered as the independent variables. Each response has been modeled by the joint Gamma models [16] (as it provides better fit). The determinants of the BBP, SBP and MBP for the DSE data set are as follows.

The basal blood pressure (BBP) of DSE data set has been modeled based on the remaining other variables using the joint Gamma models. The mean BBP is inversely separately correlated with BHR ($P<0.01$), DPMDOBP ($P<0.01$), EVENT ($P=0.03$). BBP is high at low BHR or DPMDOBP. The heart patients under DSE who are near to death have high BBP. BBP is directly separately correlated with BDP ($P<0.01$), MHR ($P<0.01$), MBP ($P<0.01$), AGE ($P=0.01$). So, the BBP is high if BDP or MHR or MBP or AGE is high. Similarly, the SBP of DSE

data set has been modeled using the joint Gamma models. Mean SBP is inversely separately correlated with BHR (P<0.01), PHR (P<0.01), PMHR (P=0.03), DPMDOBP (P<0.01), HOHT (P=0.05). Therefore, SBP is high if BHR or PHR or PMHR or DPMDOBP is low. Also the SBP is higher for the DSE patients with HOHT. Again, SBP is directly separately correlated with BDP (P<0.01), DPHS (P<0.01), DOSE (P=0.03), MHR (P<0.01), MBP (P<0.01). Thus, SBP is high if BDP or DPHS or DOSE or MHR or MBP is high. Again the MBP of the DSE data set has been modeled by the joint Gamma models. It is observed that the mean MBP is directly separately correlated with the PHR (P<0.01), SBP (P<0.01), DPMDOBP (P<0.01), AGE (P=0.13), PSE (P=0.03). So, MBP is high if PHR or SBP or DPMDOBP or AGE is high. The patients under DSE with no PSE have high MBP. Mean MBP is inversely separately correlated with DPHS (P<0.01), MHR (P<0.01), NPTCA (P=0.02), HOMI (P=0.09), HOCABG (P=0.05). Thus, MBP is high if DPHS or MHR is low. The patients under DSE with NPTCA or HOMI or HOCABG have high MBP.

Second, a shock data set with 21 variables/factors on 113 subjects (obtained at the Shock Research Unit, University of Southern California, Los Angeles, California) has been analyzed. On each subject, there are two measurements, one at the admission time (initial measurement), and the other just before death or discharge time (final measurement). This data set has been obtained based on 113 critically ill patients. The patient population, data collection method, shock types are displayed in [18]. This particular data set can be obtained from the following links

http://www.umass.edu/statdata/statdata/data/shock.txt

https://www.statcrunch.com/app/index.php?dataid=1327401

The shock data contains the following factors/ variables. These are height (HEIGHT), age (AGE), gender (male=1, female=2) (GENDER), shock type (no shock=1, hypovolemic or cardiogenic=2, bacterial or neurogenic or other=3) (SHOCK), survival (survived=1, death=2) (SURVIVE), systolic BP (SBP), heart rate (HRT), mean arterial pressure (MAPR), diastolic BP (DBP), body surface index (BSIN), mean central venous pressure (MCVPR), cardiac index (CIN), mean circulation time (MCTI), appearance time (ATI), plasma volume index (PVIN), urinary output (UOU), hematocrit (HEMA), red cell index (RCIN), hemoglobin (HEMO), order of card (initial=1, final=2) (CARD). There are three blood pressures, namely SBP, DBP and MAPR. These three responses have been separately analyzed using both the joint Gamma and Log-normal models [16]. It is observed that the Gamma fitted model yields better results.

Gamma model fit of SBP on the other factors/ variables of shock data reveals the following results. It is observed that the mean SBP is inversely separately correlated with HEIGHT (P<0.01), DBP (P<0.01), SURVIVE (P=0.02) and HEMO (P=0.01). It implies that the shock patients have higher SBP if their HEIGHT is smaller or DBP is low or HEMO is low. Also the mean SBP is directly separately correlated with GENDER (P=0.01), MAPR (P<0.01), HRT (P=0.04), BSIN (P<0.01), and ATI (P<0.01). Thus, the shock patients have higher SBP if their MAPR or HRT or BSIN or ATI is high. Also the SBP is lower for male shock patients than the female. The DBP has been fitted using the joint Gamma models on the other factors/variables. The fitted model yields the following. The mean DBP is inversely separately correlated with SBP (P<0.01), SURVIVE (P<0.01), PVIN (P=0.07), and CARD (P<0.01). So, the DBP is higher for the shock patients if their SBP or PVIN is low. The shock patients have higher DBP at the initial stage than the final. Also the survived shock patients have higher DBP than who were near to death. The mean DBP is directly separately correlated with MAPR (P<0.01), AGE (P=0.08), HRT (P<0.01), ATI (P=0.04), and HEMO (P=0.08). Thus, the DBP is higher of the shock patients who have high value of MAPR or HRT or ATI or HEMO or at older age. The MAPR has been fitted using the joint Gamma models on the other factors/ variables. The fitted model gives the following. The MAPR is inversely separately correlated with HRT (P<0.01), SURVIVE (P<0.01), BSIN (P<0.01), ATI (P=0.01), and RCIN (P=0.06). So, the MAPR is higher for the shock patients who have low HRT or BSIN or ATI or RCIN. The survived shock patients have higher MAPR than who were near to death. Also the MAPR is directly separately correlated with SHOCK (P=0.05), AGE (P=0.02), SBP (P<0.01), MCVPR (P=0.023), DBP (P<0.01), CIN (P<0.01), UOU (P=0.04). HEMA (P=0.08), and CARD (P=0.05). Thus, the MAPR is higher for the shock patients who have older age or high value of SBP or MCVPR or DBP or CIN or UOU or HEMA. Again the MAPR is higher for the shock patients with shock levels at bacterial or neurogenic or other than the non-shock or hypovolemic or cardiogenic. Also, the MAPR is higher of the shock patients at the final stage than the initial.

Third, we have considered the chronic kidney patients data which have been collected under the supervision of Dr. P. Soundarapandian, in Apollo Hospitals, Managiri, Madurai, Tamilnadu, India, and the data set has been created by Jerlin Rubini, Alagappa University (E-mail: jel.jerlin@gmail.com). This study contains factors/variables as follows: 1. Age (years) (coded as AGE), 2. Blood pressure (mmHg) (coded as BP), 3. Specific gravity (nominal) (coded as SG), 4. Albumin (nominal) (coded as AL), 5. Sugar (nominal) (coded as SU), 6. Red Blood Cells (nominal) (abnormal =1, normal=2) (coded as RBC), 7. Pus Cell (nominal) (abnormal=1, normal=2) (coded as PC), 8. Pus Cell clumps (nominal) (not present=1, present=2) (coded as PCC), 9. Bacteria (nominal) (not present=1, present=2) (coded as BA), 10. Blood Glucose Random (numerical) (mgs/dl) (coded as BGR), 11. Blood Urea (numerical) (mgs/dl) (coded as BU), 12. Serum Creatinine (numerical) (mgs/dl) (coded as SC), 13. Sodium (numerical) (mEq/L) (coded as SOD), 14. Potassium (numerical) (mEq/L) (coded as POT), 15. Hemoglobin (numerical) (gms) (coded as HEMO), 16. Packed Cell Volume (numerical) (coded as PCV), 17. White Blood Cell Count (numerical) (cells/cumm) (coded as WBCC), 18. Red Blood Cell Count (numerical) (millions/cmm) (coded as RBCC), 19. Hypertension (nominal) (no=1, yes=2) (coded as HTN), 20. Diabetes Mellitus (nominal) (no=1, yes=2) (coded as DM), 21. Coronary Artery Disease (nominal) (no=1, yes=2) (coded as CAD), 22. Appetite (nominal) (good=1, poor=2) (coded as APPET), 23. Pedal Edema (nominal) (no=1, yes=2) (coded as PED), 24. Anemia (nominal) (no=1, yes=2) (coded as ANE), 25.

Class (nominal) (ckd=1, notckd=2) (coded as CLASS). We have analyzed this data set using both the joint Gamma and Log-normal models [16]. It is observed that the Log-normal fit gives better results. There is only one blood pressure (coded as BP) which is treated as the dependent variable, and the rest of others are considered as the independent variables. The determinants of the BP are as follows.

The mean BP is inversely separately correlated with the RBC ($P<0.01$), PC ($P=0.10$), PCC ($P<0.01$), BGR ($P<0.01$), BU ($P<0.01$), PCV ($P=0.02$), WBCC ($P<0.01$), HTN ($P=0.02$), PED ($P<0.01$), ANE ($P<0.01$). So, for the chronic kidney patients, the mean BP is higher if their RBC or PC or PCC or BGR or BU or PCV or WBCC is low. It also indicates that the mean BP is higher for the kidney patients who have no HTN or PED or ANE than the patients having HTN or PED or ANE. Also the mean BP is directly separately correlated with the SC ($P<0.01$), DM ($P=0.01$), CAD ($P<0.01$) and APPET ($P<0.01$). Thus, the mean BP is higher for the kidney patients who have high SC value. Mean BP is higher for the kidney patients having DM or CAD than the patients without these. For the kidney patients with poor APPET have high mean BP than the patients with good APPET.

Based on the joint Gamma and Log-normal models [16], the above results have been presented. This report only contains the different blood pressure mean models determinants. Variance models determinants along with their derivations will be presented in the full paper. The above described different blood pressure mean models determinants are related with the cardiac patients who underwent DSE, chronic kidney and shock patients. This report presents many determinants for different blood pressures. The determinants are different for different types of blood pressures. For different types of patients, the determinants for a specific blood pressure are different. This report also presents the association among the different types of blood pressures. Only the BP is stated in the chronic kidney patients. It is not clearly defined. In case of shock data, it is seen that SBP and DBP are inversely related. They should be directly related in case of cardiac patients. But for shock patients, it looks different relation. It may be due to shock. This report gives a clear idea about the different blood pressure determinants and their associations for different types of patients to the medical practitioners. Medical researcher is advised to derive the BP determinants separately for different types of patients and for different types of BP. These BP determinants depend on the types of patients and also the BP types.

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