

# Detection of Parkinson Disease Using Machine Learning Algorithm

Dr D J Samatha Naidu<sup>1</sup>, A.Raja Mohan Reddy<sup>2</sup>  
<sup>\*1</sup>Principal, APGCCS, Rajampet  
<sup>2</sup>MCA Department, APGCCS, Rajampet

---

## Abstract

Parkinson's disease (PD) is considered a malison for mankind for several decades. Its detection with the help of an automated system is a subject undergoing intense study. This entails a need for incorporating a machine learning model for the early detection of PD. For discovering a full proof model, the cardinal prerequisite is to study the existing computational intelligent techniques in the field of research used for PD detection. Many existing models focus on singular modality or have a cursory analysis of multiple modalities. This encouraged us to provide a comparative literature study of four main modalities signifying major symptoms used for early detection of PD, namely, tremor at rest, bradykinesia, rigidity, and, voice impairment. State- of-the-art machine learning implementations namely Logistic Regression (LR), Support Vector Machine (SVM), Decision Tree (DT), K-nearest neighbors (KNN), Stochastic Gradient Descent (SGD) and Gaussian Naive Bayes (GNB) are executed in these modalities with their respective datasets. Furthermore, ensemble approaches such as Random Forest Classifier (RF), Adaptive Boosting (AB) and Hard Voting (HV) are implemented.

---

Date of Submission: 21-08-2021

Date of acceptance: 05-09-2021

---

## I. INTRODUCTION

Parkinson's disease (PD) is a long-termed, neurological disorder that causes a person to lose control over several body functions including speech. It is the second most common neurodegenerative disease after Alzheimer's disease [1]. Dr. James Parkinson was the first to describe this condition called 'paralysis agitation' or 'the shaking palsy' [2]. In the 21st century, PD is a ubiquitous issue. In 2015, PD affected 6.2 million people and resulted in about 1,17,400 deaths globally. This accounts for various researches [3] [4] [5] to be undertaken to study and eventually cure the disease. The loss of nerve cells in the part of the brain called the substantia nigra causes PD. These nerve cells or neurons create an organic chemical named dopamine which acts as a neurotransmitter between the parts of the brain and central nervous system that helps to control and co-ordinate body movements. Although this disease can be diagnosed at an early stage [6], its long term treatment is not yet discovered. The clinical diagnosis of the patient by the doctor was focused on his/her sense and experience, based on his/her knowledge and studying previous cases of PD from large databases in the hospitals. But with the advent of strong tools like Artificial Intelligence and Machine Learning, this took a subtle turn [7], various state-of-the-art machine learning tools and techniques analyzed the high dimensions of data in the datasets which made the work of prediction simple.

## II. EXISTING WORK

In the existing work, the condition is usually diagnosed by a neurologist based on a review of your symptoms and a physical exam. A DaTscan to visualize your brain's dopamine system may help confirm diagnosis. Blood tests and other imaging tests, such as an MRI scan, don't diagnose Parkinson's.

### Limitations of Existing Work:

- Detection of Parkinson's disease is difficult.
- Results are not accurate.
- It takes more time to give results.
- High time resolution.
- High variation in results.
- Low efficiency.

### III. PROPOSED WORK

In proposed work, we use implement machine learning algorithms to detect Parkinson’s disease such as Logistic Regression (LR), Support Vector Machine (SVM), Decision Tree (DT), K-nearest neighbors (KNN), Stochastic Gradient Descent (SGD) and Gaussian Naive Bayes (GNB) are executed in these modalities with their respective datasets. Furthermore, ensemble approaches such as Random Forest Classifier (RF), Adaptive Boosting (AB) and Hard Voting (HV) are implemented.

#### Features of proposed work:

- Low time resolution.
- High efficiency.
- Low variation in results.
- Complexity is low.
- Detection of Parkinson’s disease is easy.
- Prediction results are accurate.

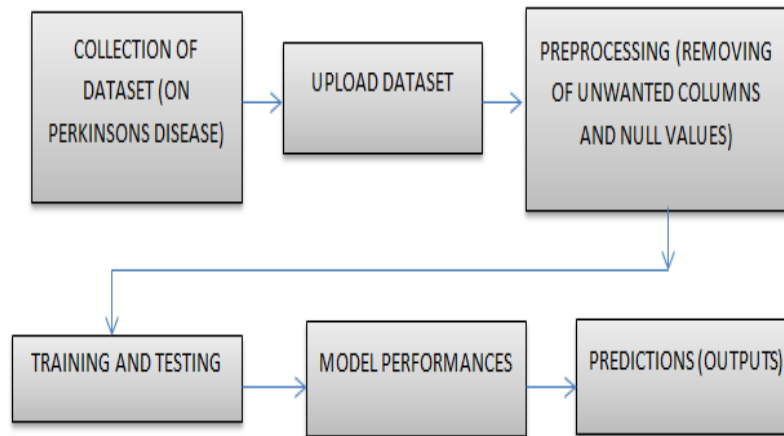
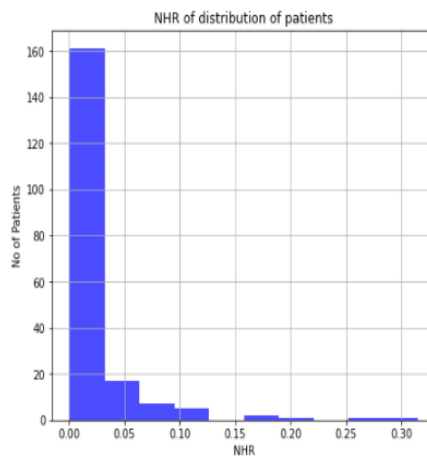


Figure1: Block Diagram of Upload Dataset

here the collection of patient’s data set is collected and uploaded in the dataset and further the data is sent to processing of data where the noisy values and missing values are removed and the processed data is sent to training and testing then the obtained data is performed in the model where we get the final prediction.

### IV. RESULTS



PD patients scan reports

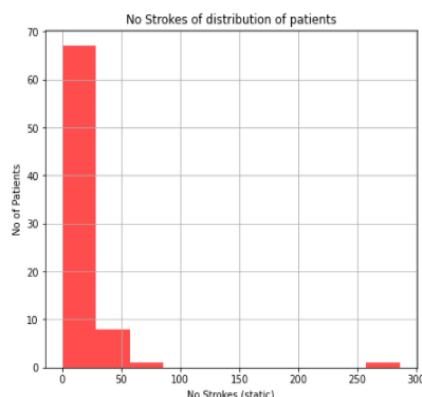
The cases were those recently reported in detail<sup>2</sup> and started with uncultured groups of 131 consecutive recipients whose first kidneys were given by blood relatives and of 58 consecutive recipients of nonrelated kidneys (35 volunteers and 23 cadavers). The related cases were compiled 2½–7½ years ago and the nonrelated

cases from 1½–7½ years ago. The immunosuppression for 112 of the patients was azathioprine and prednisone; the last 77 were also given heterologous ALG.

Of the 189 cases, all were included if the raw lymphocyte antigen typing data were available for both donor and recipient. There was sufficient information with transplantations from 56 siblings, 49 parents, 8 more distant relatives (aunts, uncles, and cousins which were included for the various statistical analyses with the siblings) and 38 nonrelatives (total 151). Correlations of match with survival were made in all 151 typed cases. For the related recipients, matches were correlated with steroid dosage and homograft function at 1 and 2 years in the event of survival for these periods; the same applied in nonrelated cases except that survival periods for sampling were 1 and 1½ years.

Correlations of the matches expressed by the NHR with histopathology were done only if the homografts sampled had been in residence for at least 2½ months, thereby excluding 8 of the 151 cases because of death before this time. Twenty-one specimens included in the analysis were obtained at autopsy or at homograft nephrectomy from 2½ to 27 months after transplantation. Most of the tissues studied (113 total) were biopsies taken after 15–33 months. Nine of the homografts have never become available under any of the foregoing circumstances. The tissues were examined with light microscopy, and in most instances by electron microscopy and immunofluorescence (IF) as described elsewhere.<sup>2</sup> Insofar as the method of tissue collection permitted, the presence or absence of the 13 features listed were determined and graded in severity from 0 to 4.

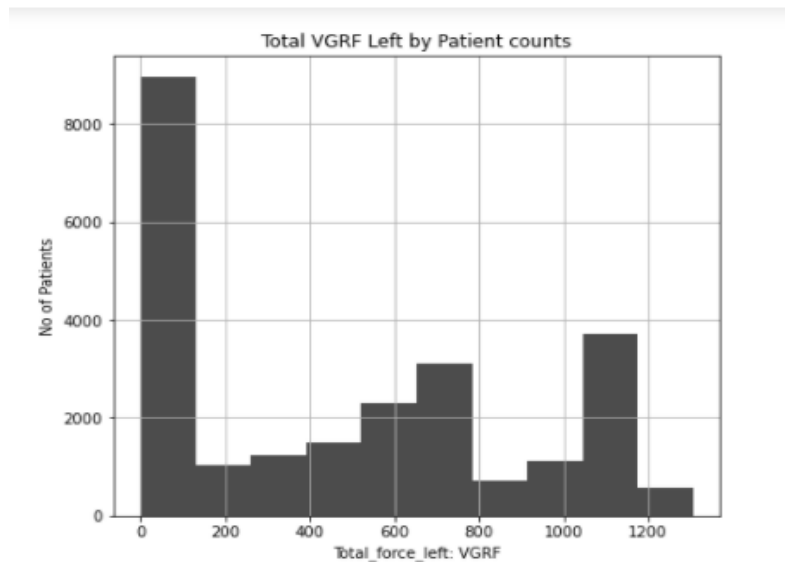
The NHR calculation<sup>1</sup> was based upon the hypotheses that the major histocompatibility factors are on two loci of a single (HL-A) chromosome, that each locus governs the expression of two histocompatibility antigens, and that the measurement of either more or less than two antigens at one or the other locus is by definition probably a methodologic artifact. In the formula,  $NHR = \frac{1}{4}$  (donor-recipient antigen identities/antigen incompatibilities), adjustments were made by the designation of “potential relations” if a full complement of alleles could not be defined in either the donor or recipient, particularly the former. The result was to depreciate the NHR value in situations with transplantation of *no antigen to antigen* by considering this as a “potential” rather than as an identity and by therefore preventing it from contributing to a high NHR score. A second adjustment was to consider the following three families of cross reacting antigens to be operationally identical\*: HL-A 1 (HL-A 3 if present as a third antigen); HL-A 5 (Te 6, Te 55, Te 58); and HL-A 7 (Te 51, Te 60). This latter adjustment tended to improve the NHR scores by changing a number of incompatibilities to compatibilities; in no instance was the NHR worsened. In some cases in which typing was carried out several years ago with a multispecific antiserum called “Old 3,” a positive reaction could have been due to HL-A 9 or HL-A 10 on the first sublocus or HL-A 5, HL-A 12, or Te 60 on the second sublocus. The interpretation of the “Old 3” reactions was on a highly individual basis. All NHR scores were computed by Rapaport<sup>2</sup> without knowledge of the outcome in the cases under scrutiny. To those monitoring these calculations, it was obvious that an element of judgment and intelligence was introduced which rendered the determinations much more than a technical exercise, and which would make difficult a duplication of the scores by the simple insertion of data into a strict mathematical formula.



### NO PD patients

Perioperative stroke occurred in 20 of 530 patients (3.8%) undergoing TEVAR. The cohort was 55% male and a mean age of  $75.2 \pm 8.9$  years (range, 57–90 years). Among patients with perioperative strokes, the indication for surgery was degenerative aneurysm in 14 (mean diameter, 6.8 cm), acute type B dissection in four, penetrating atherosclerotic aneurysm in one, and aortic transection in one. Cases were performed urgently or as an emergency in 60%. The proximal landing zone was zone 2 in 11 or zone 3 in nine. All strokes were embolic. The vascular distribution of stroke involved the anterior cerebral (AC) circulation in eight (zone 2, n = 5) and the posterior cerebral (PC) circulation in 12 (zone 2, n = 6). Laterality of cerebral infarction included five

right-sided, eight left-sided, and seven bilateral strokes. Nine strokes were diagnosed <24 hours after operation. There was no difference in baseline demographics, aortic pathology, acuity, zone coverage, preoperative left subclavian artery revascularization, number of stents, or estimated blood loss between stroke groups based on vascular distribution. Independent risk factors for any perioperative stroke were chronic renal insufficiency (odds ratios [OR], 4.65; 95% confidence interval [CI], 1.22-17.7;  $P = .02$ ) and history of prior stroke (OR, 4.92; 95% CI, 1.69-14.4;  $P = .004$ ); the risk factor for AC stroke was prior stroke (OR, 7.67; 95% CI, 1.25-46.9;  $P = .03$ ) and the risk factors for PC stroke were age (OR, 1.11; 95% CI, 1.00-1.23;  $P = .04$ ), prior stroke (OR, 7.53; 95% CI, 1.78-31.8;  $P = .006$ ), zone 2 coverage (OR, 6.11; 95% CI, 1.15-32.3;  $P = .03$ ), and penetrating atherosclerotic ulcer (OR, 32.7; 95% CI, 1.33-807.2;  $P = .03$ ). Overall in-hospital mortality was 20% ( $n = 4$ ), with those sustaining PC strokes observed to trend toward increased mortality (33% vs 0%;  $P = .12$ ). Patients with AC strokes were more likely than those with PC strokes to achieve complete recovery of neurologic deficits before discharge (75% vs 17%;  $P = .02$ ).



**Total VGRF Left by Patient counts**

The data was recorded by Yogev et al. [30] and was downloaded for analysis from Physionet website [31, 32]. Time series of the VGRF signal recorded from 29 PD patients (Hoehn and Yahr score =  $2.3 \pm 0.40$ , UPDRS score =  $39.31 \pm 12.38$  mean age  $71.1 \pm 8.05$  years) and 18 age-matched controls (mean age  $71.6 \pm 6.6$  years) during normal level ground walking were used [31].

All the subjects were taking antiparkinsonian medications and their prescriptions were unaltered at the time of experiment. Written consent was collected from all participants, and the study was approved by the Tel-Aviv Sourasky Medical Center Human Studies Committee. As per experimental protocol, the subjects walked at their natural pace on level ground for two minutes, and data was acquired with a sampling frequency of 100 Hz. The system used to collect gait data consisted of eight sensors beneath each foot and a recording unit. A small and light (19 x 14 x 4.5 cm; 1.5 kg) recording unit was carried at the waist. A memory card contained in the unit stored the measurement data during the test, which was later transferred to a computer for analysis. To accurately describe the sensor location inside the insole, it was assumed that the subject was in a comfortable standing position with both legs parallel to each other. Then, coordinates of the sensor location can be displayed as shown in Fig 1. It was assumed that (0, 0) is the origin and lies just between the legs and the person was facing towards the positive side of the Y-axis.

**V. CONCLUSION**

Artificial Intelligence and medical sciences have developed a relationship that helps to cure pervasive disease like PD. Various symptoms like Bradykinesia, Tremor at rest, Rigidity and Voice Impairment can be detected for early detection of PD. There is no definite medical procedure/diagnosis to cure parkinsonism of a person, which even applies to bioinformatics. But strong tools like Machine Learning have abridged the process of detecting PD by making it economically viable and effective. Based on the researches discussed in this paper, machine learning can assist doctors in detecting PD.

Simple like electronic devices, like a mobile phone for voice recording, using software like Tappy for detecting slowness in movement, and many more can be utilized for detection. The detection of bradykinesia and tremor leads to the concrete results for the early detection of this disease. Moreover, noticed the accuracy of

detection could be increased in two ways, by implementing ensemble approaches like bagging, boosting, voting, and by increasing the size of the dataset.

#### REFERENCES

- [1]. D. B. Calne "Is idiopathic parkinsonism the consequence of an event or a process" *Neurology*, Vol. 44, no. 15, pp. 5–5, 1994.
- [2]. J. Parkinson "An Essay on Shaking Palsy. London: Whittingham and Rowland Printing 1817".
- [3]. Surathi, P., Jhunjhunwala, K., Yadav, R., Pal, P. K. "Research in Parkinson's disease in India: A review : *Annals of Indian Academy of Neurology*" 19(1), 9–20. doi:10.4103/0972-2327.167713, 2016.
- [4]. Muthane UB, Chickabasaviah YT, Henderson J, Kingsbury AE, Kilford L, Shankar SK, et al. "Melanized nigral neuronal numbers in Nigerian and British individuals" *Mov Disord.* 2006;21:123941.
- [5]. Baldereschi M, Di Carlo A, Rocca WA, Vanni P, Maggi S, Perissinotto E, et al. "Parkinson's disease and parkinsonism in a longitudinal study: Two-fold higher incidence in men. ILSA Working Group. Italian Longitudinal Study on Aging." *Neurology.* 2000;55:135863.
- [6]. Steven T. DeKosky, Kenneth Marek "Looking Backward to Move Forward: Early Detection of Neurodegenerative Disorders" *The American Association for the Advancement of Science* Vol. 302, Issue 5646, pp. 830-834 DOI: 10.1126/science.1090349, 2003.
- [7]. Farhad Soleimani Gharehchopogh, Peyman Mohammadi and Parvin Hakimi "Application of Decision Tree Algorithm for Data Mining in Healthcare Operations: A Case Study." *International Journal of Computer Applications*, Vol: 52, No:6, PP:21-26, 2012.
- [8]. A. Schrag, C. D. Good, K. Miszkielec, H. R. Morris, C. J. Mathias, A. J. Lees, and N. P. Quinn "Differentiation of atypical parkinsonian syndromes with routine MRI." *Neurology*, Vol. 54, pp. 697–702, 2000.
- [9]. S. L. Wu, R. M. Liscic, S. Kim, S. Sorbi, and Y. H. Yang "Nonmotor symptoms of Parkinson's disease." *Parkinson's Dis.*, 2017. DOI:10.1155/2017/4382518, 2017.
- [10]. T. Yousaf, H. Wilson, and M. Politis "Imaging the nonmotor symptoms in Parkinson's disease." *Int. Rev. Neurobiol.*, Vol. 133, pp. 179–257, 2017. [12] C. G. Goetz, et al. "Movement disorder society-sponsored revision of the unified Parkinson's disease rating scale (MDSUPDRS): Scale presentation and clinimetric testing results." *Mov. Disord.*, Vol. 23, no. 15, pp. 2129–70, 2008.