ORIGINAL ARTICLE

Postural Instability in Idiopathic Parkinson's Disease: Determination of VEP, BAER, and SSEP Cutoff Values for an Early Screening of Fall

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Received: 03 April 2025; *Accepted:* 25 April 2025

Abstract

Backgrounds and aims: Idiopathic Parkinson's disease (IPD) patients had progressively increased slowness, rest tremors, rigidity, and postural instability (PI). Postural stability depends on sensory inputs from visual, auditory, and somatosensory modalities. We tried to find important cutoff values of visual evoked potential (VEP), brainstem auditory evoked response (BAER), and short-latency somatosensory evoked potentials (SSEP) for determining postural stability in IPD patients.

Methodology: About 50 IPD patients were recruited in a cross-sectional observational study. A pull test was used to determine postural stability. Patients were subgrouped into tremor dominant (TD variant) (n = 37) and PI and gait disorder (PIGD) (n = 13). We generated receiver operating characteristic (ROC) curves to classify patients into posturally stable and unstable and measured VEP, BAER, and SSEP cutoff values. The area under the curve (AUC) >0.8 was taken as significant.

Results: Significant VEP N75, P100, and N145 cutoff values were noted bilaterally in IPD and its subgroups (TD and PIGD). Except for wave I, the latency of all other BAER waves showed significant cutoff values bilaterally in IPD and subgroups (TD and PIGD). Most BAER cutoff values in the IPD and TD subgroups reached 100% specificity. No significant SSEP values were noted.

Discussion: Many significant VEP and BAER parameters with good sensitivity and specificity would guide clinicians in predicting PI and falls in IPD. The TD had lower BAER latency cutoff values than the PIGD. The postural stability of the TD subgroup was more dependent on the vestibular sensory input than that of the PIGD subgroup. Less vestibular compensatory support in PIGD led to a more severe phenotype than in TD.

Conclusion: We found many evoked potential significant cutoff values determining postural stability in IPD and its subgroups (TD and PIGD). Lesser vestibular compensatory support in PIGD led to a more severe phenotype than in TD.

Journal of The Association of Physicians of India (2025): 10.59556/japi.73.1166

Introduction

Idiopathic Parkinson's disease (IPD) is a progressive neurodegenerative disease of the brain. Patients have progressively increased slowness, rest tremors, rigidity, and postural instability (PI), apart from many other manifestations.[1] As the disease progresses, patients become more prone to recurrent falls, which lead to recurrent hospital admission, increased morbidity, and mortality. PI is a serious feature of IPD. Many factors are involved in PI, including loss of postural reflexes, medication side effects, freezing, festination, orthostatic hypotension, fear of falls, age-related reduced peripheral sensation, and leg muscle weakness.[2] If we can diagnose and treat PI at the earliest, we can give them a healthy, long life.

At present, the diagnosis of PI is subjective. Various scales are validated for the diagnosis of PI and the progression of Parkinson's disease.[3,4] A "pull test" is a simple test where the patient is quickly pulled forward or backward by the shoulder. If the patient takes more than two steps to recover balance or no postural response occurs, it is considered "positive" for PI.[1] Hoehn and Yahr's scale was published in 1967 by Hoehn and Yahr.[5] It had stages I to V as PI worsened and Parkinson's disease progressed. The Movement Disorder Society—Unified Parkinson's Disease Rating Scale (MDS-UPDRS) was a modification of the original UPDRS score, which showed the motor and nonmotor symptom severity of IPD.[6]

Postural stability also depends on many factors, including the motor and sensory systems. Many articles discussed the role of visual evoked potential (VEP),[7] brainstem auditory evoked response (BAER),[7,8] and short-latency somatosensory evoked potentials (SSEP)[7,9–12] on postural stability. The studies showed that patients with Parkinson's disease had significant abnormalities in the evoked potentials compared to healthy subjects. Studies also pointed out a correlation between evoked potential changes and the severity of the disease.[12] As the role of various sensory inputs was revealed to be important in the postural stability of Parkinson's disease, we tried to find out important cutoff values of VEP, BAER, and SSEP determining the postural stability in Parkinson's disease patients.

Methodology

Study Subjects

This is a cross-sectional, observational study conducted in the inpatient and outpatient clinics of the Department of Neurology, Teaching Hospital, from September 2017 to August 2020. We enrolled 50 IPD patients of both genders and various age-groups. IPD patients were subgrouped into postural instability and gait disorder (PIGD) variant and tremor dominant (TD) variant according to their clinical phenotype. Detailed neurological examinations were performed. Informed consent was obtained before recruiting them. The Institutional Ethics Committee approved the study (NMC/958).

The inclusion criteria of this study are as follows:

- All IPD cases were diagnosed in adherence with the UK Parkinson's Disease Society Brain Bank Clinical Diagnostic Criteria.[13]

The exclusion criteria of this study are as follows:

- Cases of secondary Parkinsonism.

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How to cite this article: Mukherjee J, Roy MK, Chaudhuri J, *et al.* Postural Instability in Idiopathic Parkinson's Disease: Determination of VEP, BAER, and SSEP Cutoff Values for an Early Screening of Fall. J Assoc Physicians India 2025;73(9):28–36.

- Patients with previously known ophthalmological disorders (uncorrected refractive errors, glaucoma, retinopathies, etc.) and hearing difficulties after thorough evaluation by specialist doctors were excluded.
- Patients having abnormalities in pain, touch, and joint position sense.
- Patients having motor weakness in the lower limbs.
- Patients with orthostatic hypotension and vertigo.

Study Design

This is a cross-sectional, observational study. The pull test was used to detect PI. IPD patients and the subgroups (PIGD and TD) were divided into patients with PI and those without. Then, we analyzed the evoked potential values of the two groups.

Parameters for Evaluation

Evoked potentials were recorded using Nihon Kohden NeuroPack II Plus.

Visual Evoked Potentials

The recording was made for the checkerboard-patterned reversal VEP (CBPR VEP). VEP was recorded from each eye separately with surface electrodes, with the reference electrode placed on Fz, the active electrode on Oz, and other electrodes on O1 and O2 as per the International 10–20 system. The analysis time was 500 ms, and 256 sweeps were averaged. N75 latency, P100 latency, and P100 amplitude were recorded. P100 latency is the interval between the stimulus and the peak of the major positive component.

Brainstem Auditory Evoked Potential

Auditory evoked responses were obtained by brief acoustic click stimuli delivering monophasic square pulses of 100 ms duration to headphones with a monoaural stimulus intensity of 60–65 dB HL. As many patients were experiencing subclinical hearing loss in this sample, we resorted to gradually increasing the decibel if a BAER waveform was not obtained. The contralateral ear was masked with continuous white noise at 30–40 dB below the BAER stimulus. Recording electrodes were placed at the vertex (location Cz of the International 10–20 system) and the mastoids (Mi and Mc). The amplitude and latency of waves I to V were recorded.

Somatosensory Evoked Potential

The anode was placed just proximal to the palmar crease, the cathode was placed between the tendons of the palmaris longus muscle, 3 cm proximal to the anode, and the median nerve was stimulated. The details of the SSEP measurements were already discussed in our other article on the evoked potentials.[12]

Statistical Analysis

We performed standard statistical methods using IBM SPSS software version 26. The Kolmogorov–Smirnov test was done for the normality of data distribution. All VEP, BAER, and SSEP parameters were classified according to postural stability. Receiver operating characteristic (ROC) curves showed significant cutoff values to determine the PI in IPD and subgroups. The area under the curve (AUC) >0.8 was taken as a good result to classify them into patients with or without PI.

Results

Demography and Descriptive Parameters

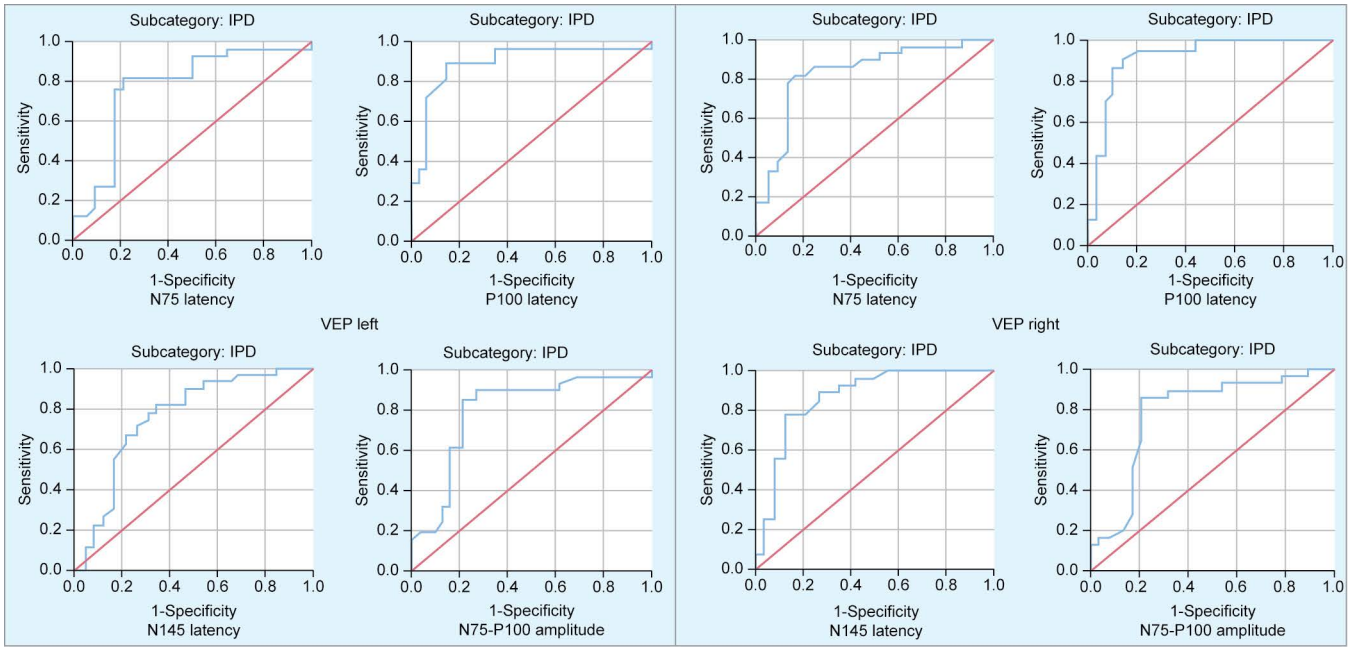
Idiopathic Parkinson's disease patients had an age at onset of 44–78 years (mean 57.4 years), a duration of illness of 1–6.5 years (mean 3 years), and an age at presentation of 47–80 years (60.4 years), which was mostly similar in its TD variety. Compared to the TD group, the PIGD subtype had a later age at onset (56.9 vs 58.9 years) and age at presentation (59.8 vs 61.9 years). IPD patients had a male preponderance (male = 58%). The PIGD subgroup had a female preponderance (male = 46.2%) in contrast to the TD group (male = 62.2%).

VEP, BAER, and SSEP Parameters (Figs 1,2 and 3)

The minimum, maximum, mean, and standard deviation values of the VEP, BAER, and SSEP parameters of the IPD patients and their subgroups are mentioned in Table 1.

VEP

Significant N75, P100, and N145 cutoff values on the right side were 69.95 ms (AUC = 0.833), 111.65 ms (AUC = 0.91), and 152.1 ms (AUC = 0.858); those on the left side, P100, were 111.6 ms (AUC = 0.88) in IPD patients. Above those values, the patients became posturally unstable. In the TD patients, significant N75, P100, and N145 cutoff values on the right side



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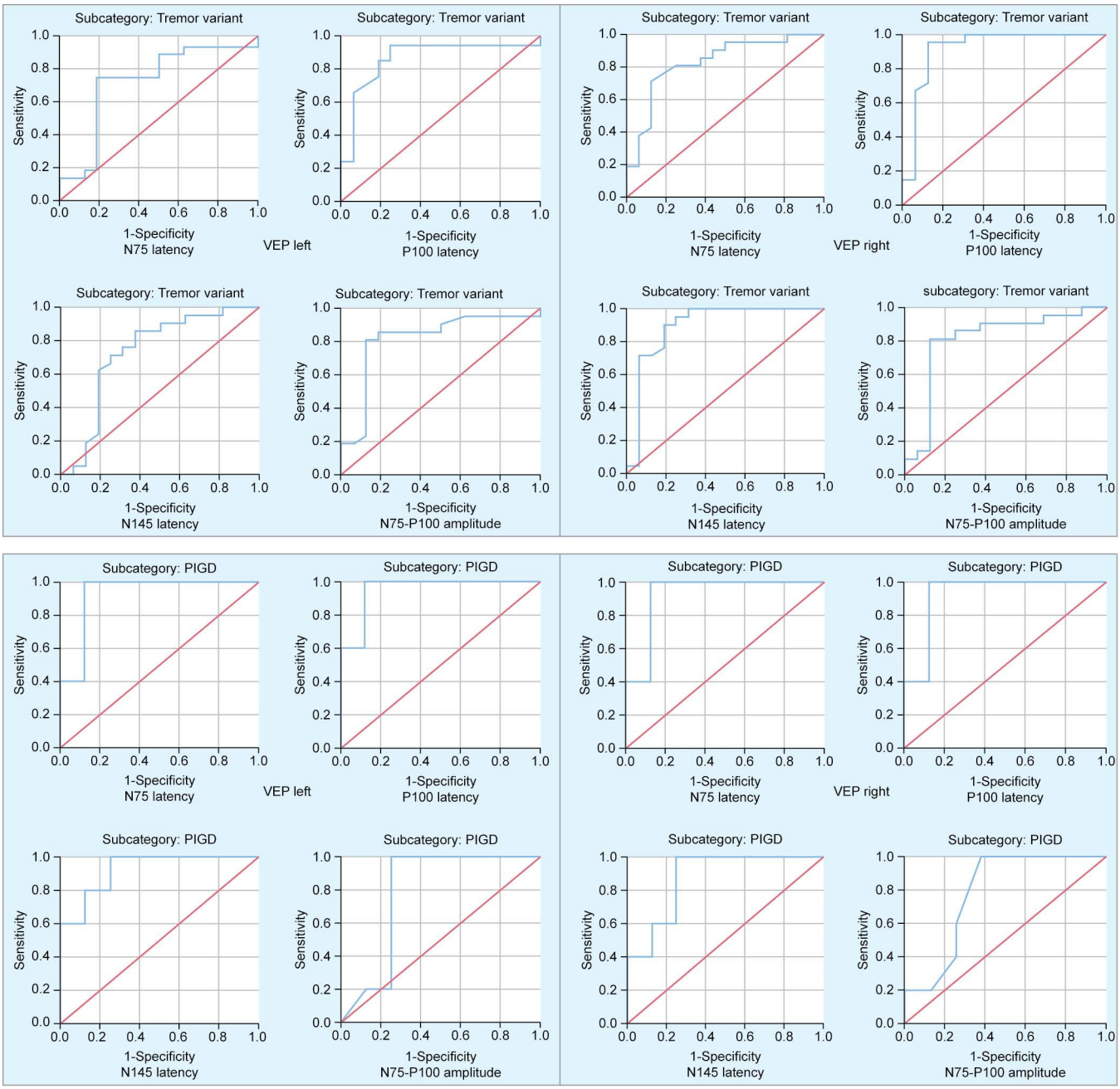


Fig. 1: ROC curves showing VEP in IPD and its subgroups—TD and PIGD variant

were 69.95 ms (AUC = 0.829), 113.7 ms (AUC = 0.918), and 153.25 ms (AUC = 0.897); those on the left side, P100, were 111.85 ms (AUC = 0.872). Additionally, the VEP amplitude cutoff was 4.04 µV (AUC = 0.81) on the right side and 4.12 µV (AUC = 0.817) on the left side, below which the TD patients became posturally unstable. PIGD patients' right side significant N75, P100, and N145 cutoff values were 69.55 ms (AUC = 0.925), 108 ms (AUC = 0.925), and 140.4 ms (AUC = 0.875); those on the left side were 73.05 ms (AUC = 0.925), 107 ms (AUC = 0.95), and 141.45 ms (AUC = 0.925). Patients' postural stability was hampered above the cutoff values (Table 2).

BAER

Except for BAER wave I, the latency of all other waves showed significant cutoff values associated with the postural stability of the IPD patients, and both TD and PIGD subgroups were impaired. In IPD patients, the significant cutoff latencies of wave II, III, IV, V, I–III interval, III–V interval, and I–V interval on the right side were 2.685 ms (AUC = 0.952), 3.815 ms (AUC = 0.914), 4.825 ms (AUC = 0.884), 5.825 ms (AUC = 0.916), 2.31 ms (AUC = 0.938), 2.015 ms (AUC = 0.868), and 4.35 ms (AUC = 0.941). On the left side, the significant cutoff latencies of wave II, III, IV, V, I–III interval, and I–V interval were 2.735 ms (AUC = 0.955), 3.825 ms (AUC = 0.921), 4.83 ms (AUC = 0.863), 5.84

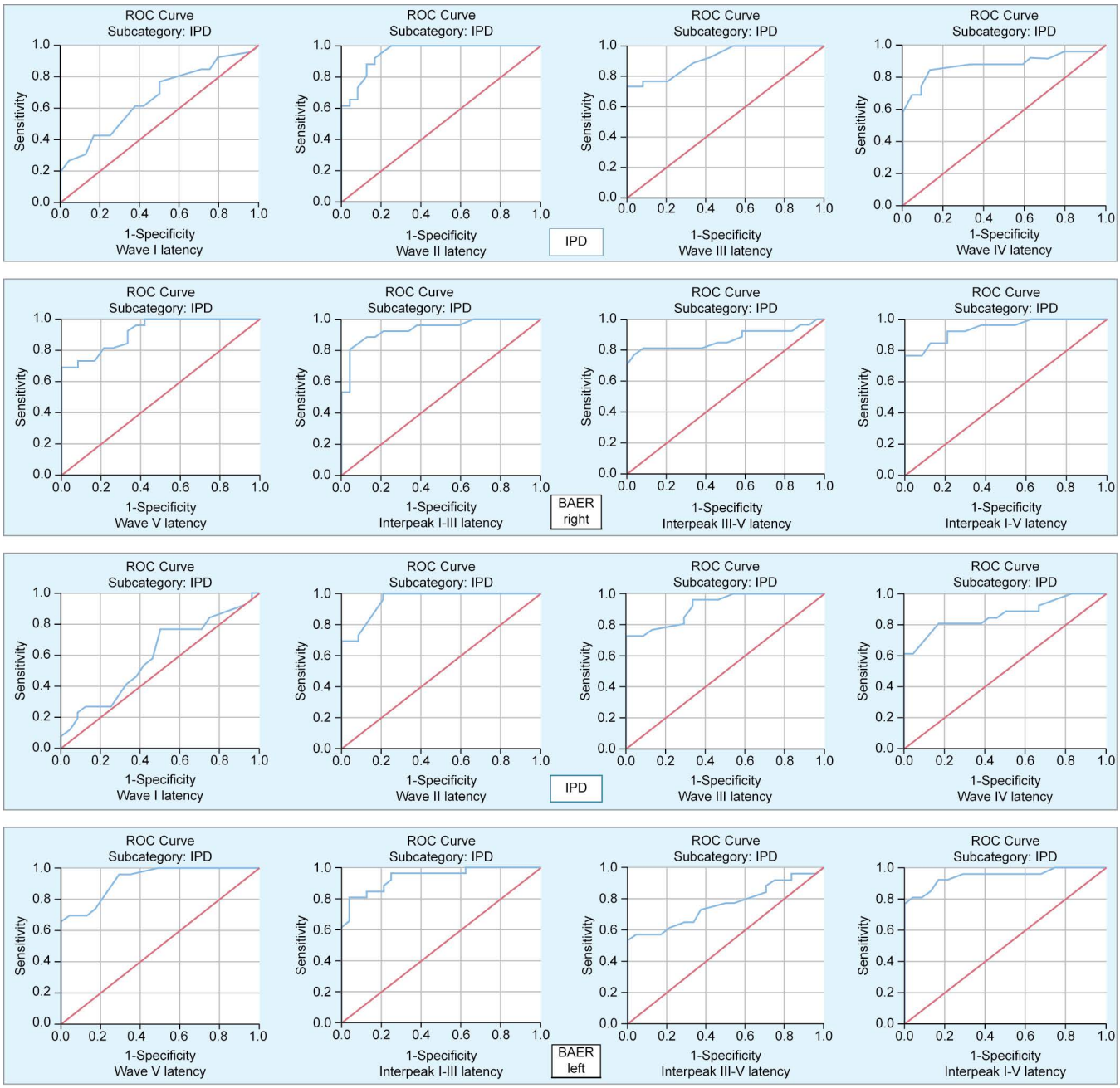


Fig. 2: ROC curves showing BAER in IPD

ms (AUC = 0.925), 2.275 ms (AUC = 0.938), and 4.36 ms (AUC = 0.947), respectively. Patients' postural stability was hampered above the cutoff values (Table 2).

The TD and PIGD subgroups followed a similar pattern, but their significant cutoff values were different. The TD variant had a lower BAER latency cutoff than the PIGD variant for PI. On the right side, the significant cutoff latencies for TD and PIGD were wave II (2.685 vs 2.83 ms), III (3.825 vs 3.945 ms), IV (4.825 vs 4.895 ms), V (5.825 vs 5.975 ms), I–III interval (2.31 vs 2.37 ms), III–V interval (2.02 vs 2.055 ms), and I–V interval (4.315 vs 4.35 ms). On the left side, the significant cutoff latencies for TD and PIGD were wave II (2.735 vs 2.81 ms), III (3.83 vs 3.935 ms), IV (4.83 vs 4.86 ms), V (5.84 vs 5.985 ms), I–III interval (2.275 vs 2.365 ms), and I–V interval (4.345 vs 4.405 ms). The latency of wave I did not show any significant cutoff values determining the postural stability in any patient group. The amplitude ratio of wave V/I had a significant cutoff value of 1.645 (AUC = 0.813) on the left side in PIGD patients. Most of the BAER cutoff values in the IPD and TD subgroups reached 100% specificity (Table 2).

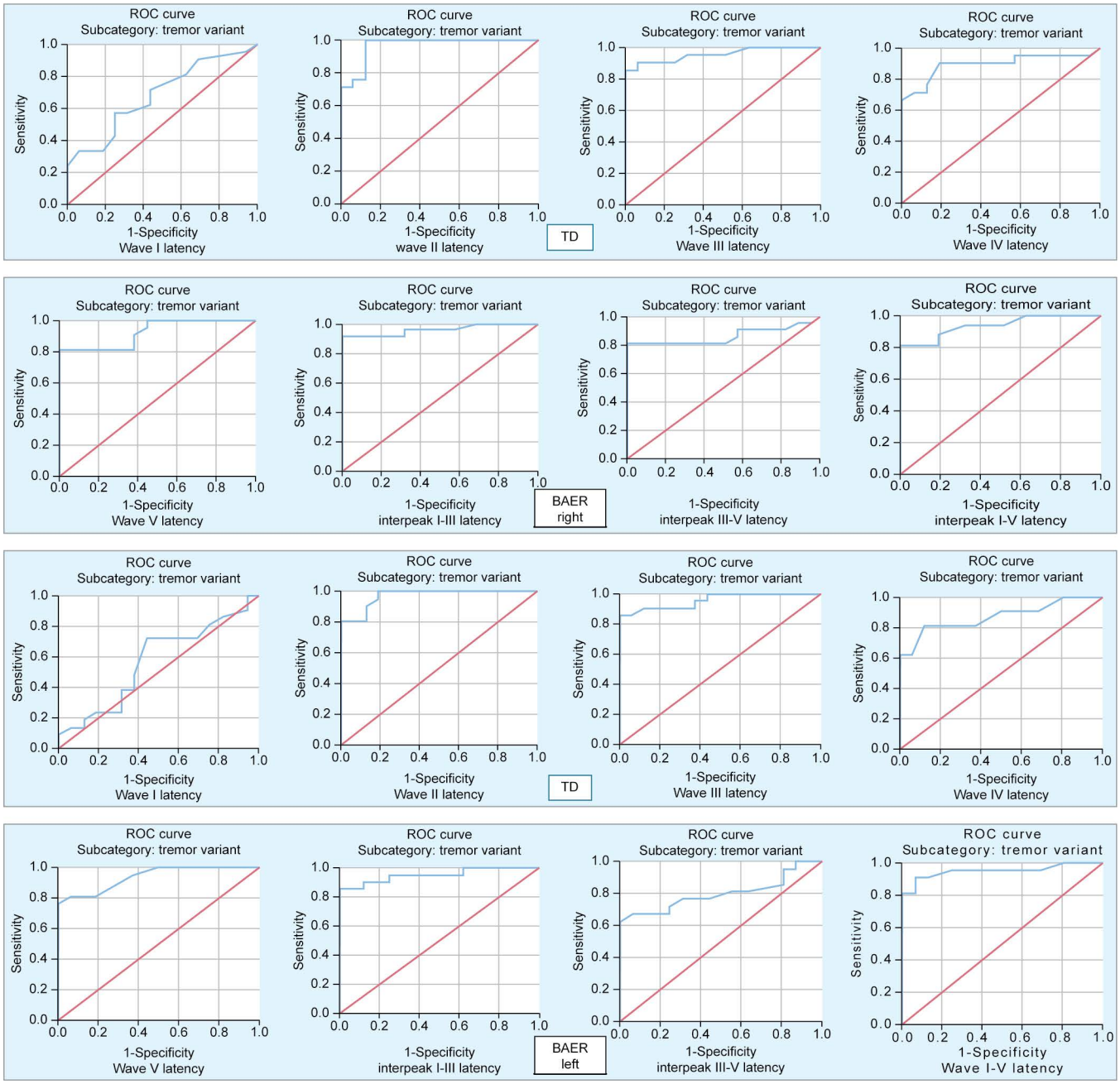
SSEP

Since the upper limb SSEP values had not achieved AUC >0.8 in ROC curves, no significant cutoff value was found determining the postural stability in any patient (Table 2).

Discussion

Dopaminergic and nondopaminergic neural circuits were involved in gross human postural control and associated with various sensory inputs (visual, vestibular, and somatosensory). These inputs were integrated into the premotor cortex, supplementary motor cortex, cerebellum, and basal ganglia.[14,15] The motor signal is refined in the cerebellum and basal ganglia and then transmitted to the primary motor cortex, the pedunculopontine nucleus in the brainstem, and downward to the corticospinal tract.[14,15] The basal ganglia also serve for somatosensory integration, automatic postural responses, and muscle tone maintenance.[16] Dysfunction in the basal ganglia and its connections leads to PI in IPD. We measured the cutoff values of VEP, BAER, and SSEP to find the threshold level of these parameters (visual, vestibular, and somatosensory inputs) in PI in IPD and its subgroups.

Petrova et al. found that abnormal VEP and BAER results in IPD, which denoted the specific dysfunctions of the brainstem and brain hemispheres, were associated with motor and nonmotor symptoms of IPD.[8] Bohnen et al. highlighted that imbalance in IPD depended on the inability to utilize vestibular information efficiently to maintain an upright stance.[17] This was independent of their visual and somatosensory processing and dopaminergic losses in the nigrostriatal area.[17] Another study showed that neurovestibular dysfunction measured with multimodality evoked potentials predicted falls in IPD over a 1-year follow-up.[18] We classified IPD patients according to their postural stability. We found many significant VEP and BAER parameters showing good sensitivity and specificity in classifying the IPD and its subgroups (TD and PIGD) according to their postural stability status. The values would guide clinicians to determine and predict PI and falls in those patients.



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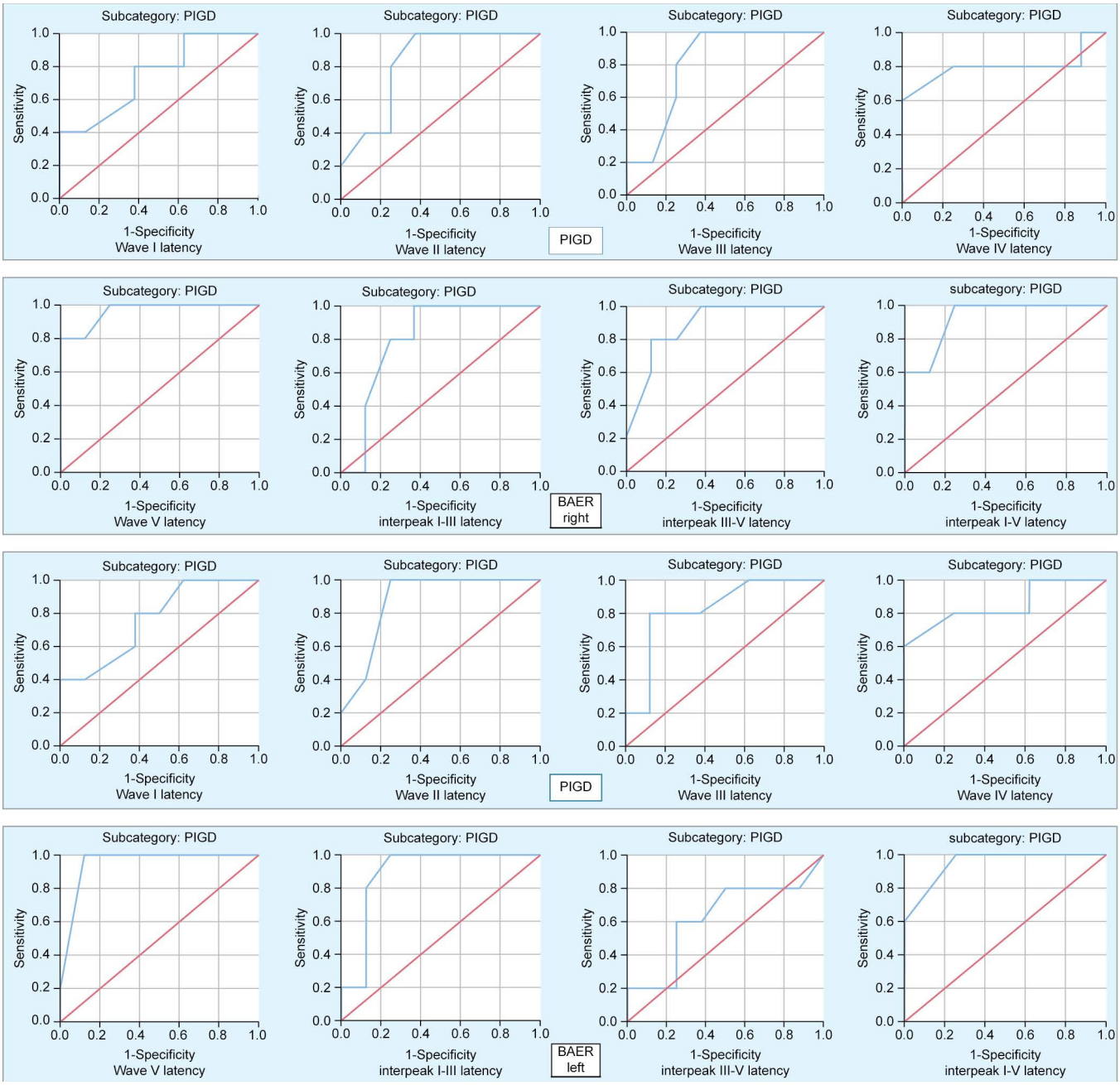

Fig. 3: ROC curves showing BAER in IPD subgroups—TD and PIGD variant

We noted that the TD variant had lower BAER latency cutoff values than the PIGD variant. We could hypothesize that the postural stability of the TD subgroup was more dependent on the vestibular sensory input than the PIGD subgroup. TD patients usually remain posturally stable for a longer time than PIGD patients. Previous studies.[19–21] showed that PIGD had aggravated motor and nonmotor symptoms compared to the TD. Lesser vestibular compensatory support might cause this severity in PIGD.

Tachibana et al. found no significant differences in N13, N20, and central conduction time (CCT) among IPD and healthy subjects. We found no significant AUC in SSEP values in IPD and its subgroups. However, before noting the SSEP parameters as insignificant, we need to study them with a larger sample size.

Though many studies have been done with VEP, BAER, and SSEP in IPD, our study first tried to find the cutoff values that determine the postural stability of the patients. In this respect, it is a novel idea. The PI cutoff values of VEP, BAER, and SSEP will help physicians across the globe identify patients prone to fall and injury. It will also help in understanding the progression of the disease.

Conclusion

Visual, vestibular, and somatosensory inputs were necessary to maintain good postural stability. We found many significant evoked

Table 1: Descriptive details of demographical parameters, VEP, BAER, and SSEP in IPD and its subgroups (TD and PIGD variant)

Subcategory	*IPD = 50*				*TD = 37*				*PIGD = 13*			
Parameters	*Min*	*Max*	*Mean*	*Std. deviation*	*Min*	*Max*	*Mean*	*Std. deviation*	*Min*	*Max*	*Mean*	*Std. deviation*
Age at onset (year)	44	78	57.418	6.8107	44	78	56.903	7.2428	51	66	58.885	5.3741
Duration of illness (year)	1	6.5	2.962	1.3993	1	6.5	2.935	1.5555	2	5	3.038	0.853
Age at presentation (year)	47	80	60.38	6.884	47	80	59.84	7.167	53	70	61.92	5.993
N75 (ms) R_VEP	45.6	106.9	72.394	14.4143	45.6	106.9	72.638	15.2467	55.6	90.0	71.700	12.2598
P100 (ms) R_VEP	76.3	141.3	106.268	15.8845	76.3	141.3	106.286	16.6640	87.4	125.2	106.215	14.0439
N145 (ms) R_VEP	102.1	173.1	142.396	17.0263	113.1	173.1	144.268	17.3760	102.1	156.4	137.069	15.3760
N75-P100 amp (μv) R_VEP	1.20	6.78	4.1912	1.48062	1.20	6.78	4.0470	1.56032	2.74	6.70	4.6015	1.18379
N75 (ms) L_VEP	46.8	123.8	73.220	15.5129	46.8	123.8	72.930	16.1678	56.8	99.4	74.046	14.0520
P100 (ms) L_VEP	80.0	136.3	106.268	14.5480	80.0	136.3	106.638	15.1047	84.6	122.8	105.215	13.3468
N145 (ms) L_VEP	15.4	176.1	141.046	23.7688	15.4	176.1	142.192	25.7120	98.7	156.7	137.785	17.5499
N75-P100 amp (μv) L_VEP	1.47	7.20	4.2512	1.55713	1.47	7.20	4.1451	1.62457	1.96	6.20	4.5531	1.35997
I (ms) R_BAER	1.48	1.69	1.5564	0.05240	1.48	1.69	1.5489	0.05343	1.51	1.63	1.5777	0.04456
II R_BAER	2.38	3.01	2.7676	0.15026	2.38	3.01	2.7443	0.16220	2.65	2.98	2.8338	0.08272
III R_BAER	3.44	4.07	3.8454	0.15251	3.44	4.07	3.8146	0.16191	3.76	4.04	3.9331	0.07123
IV R_BAER	4.46	5.10	4.8452	0.14949	4.46	5.10	4.8292	0.15997	4.58	4.99	4.8908	0.10696
V R_BAER	5.38	6.12	5.8532	0.25086	5.38	6.12	5.8068	0.26607	5.56	6.12	5.9854	0.13782
I–III R_BAER	1.90	2.57	2.2892	0.16332	1.90	2.57	2.2659	0.17713	2.21	2.52	2.3554	0.09153
III–V R_BAER	1.60	2.25	2.0078	0.14248	1.60	2.19	1.9922	0.15325	1.80	2.25	2.0523	0.09765
I–V R_BAER	3.79	4.63	4.2968	0.26405	3.79	4.63	4.2578	0.28579	4.02	4.60	4.4077	0.14715
V/I (amp μv) R_BAER	1.57	1.76	1.6260	0.04703	1.57	1.76	1.6295	0.05071	1.57	1.68	1.6162	0.03429
I (ms) L_BAER	1.45	1.69	1.5666	0.06150	1.45	1.69	1.5635	0.06533	1.48	1.65	1.5754	0.05027
II L_BAER	2.44	3.10	2.7614	0.15020	2.44	3.10	2.7386	0.16264	2.65	2.96	2.8262	0.08140
III L_BAER	3.50	4.13	3.8410	0.15015	3.50	4.13	3.8151	0.15613	3.59	3.99	3.9146	0.10485
IV L_BAER	4.47	5.06	4.8476	0.13617	4.47	5.06	4.8354	0.14709	4.68	4.99	4.8823	0.09506
V L_BAER	5.37	6.14	5.8602	0.23982	5.37	6.14	5.8141	0.25132	5.56	6.14	5.9915	0.14177
I–III L_BAER	1.93	2.64	2.2818	0.16903	1.93	2.64	2.2616	0.17885	2.01	2.48	2.3392	0.12599
III–V L_BAER	1.67	2.38	2.0336	0.14924	1.67	2.23	1.9989	0.14102	1.97	2.38	2.1323	0.13046
I–V L_BAER	3.81	4.66	4.3010	0.25734	3.81	4.65	4.2605	0.27374	3.98	4.66	4.4162	0.16184
V/I (amp μv) 0020 L_BAER	1.57	1.72	1.6412	0.03761	1.57	1.72	1.6454	0.03739	1.57	1.70	1.6292	0.03707
N9 R_SSEP	8.59	9.27	8.9460	0.17826	8.59	9.27	8.9330	0.19719	8.79	9.13	8.9831	0.10531
N13 R_SSEP	11.95	14.24	13.1460	0.60388	11.95	14.24	13.1143	0.56434	11.96	14.19	13.2362	0.72233
N20 R_SSEP	17.63	20.28	19.0510	0.66140	17.63	20.28	19.0157	0.63577	17.75	20.19	19.1515	0.74756
N13-N20 R_SSEP	5.67	6.13	5.9050	0.12888	5.67	6.13	5.9014	0.14162	5.79	6.00	5.9154	0.08657
N9 L_SSEP	8.60	9.23	8.9484	0.16365	8.60	9.23	8.9376	0.18250	8.85	9.11	8.9792	0.08967
N13 L_SSEP	11.93	14.20	13.1432	0.60227	11.93	14.20	13.1141	0.55847	11.93	14.17	13.2262	0.73171
N20 L_SSEP	17.63	20.30	19.0352	0.66368	17.63	20.30	18.9986	0.63048	17.70	20.27	19.1392	0.76827
N13–N20 L_SSEP	5.65	6.10	5.8920	0.12558	5.65	6.10	5.8846	0.13266	5.75	6.10	5.9131	0.10467

L, left; PIGD, postural instability gait disorder variant of IPD; R, right; TD, tremor dominant variant of IPD

Table 2: Cutoff values with sensitivity, specificity, and AUC of VEP, BAER, and SSEP values in IPD and its subgroups (TD and PIGD variant)

Subcategory	*IPD = 50*					*TD = 37*					*PIGD = 13*				
Parameters	*AUC*	*p-value*	*cutoff value*	*SN*	*SP*	*AUC*	*p-value*	*cutoff value*	*SN*	*SP*	*AUC*	*p-value*	*cutoff value*	*SN*	*SP*
N75 (ms) R_VEP	0.833	<0.001	69.95	76.9	87.5	0.829	0.001	69.95	71.4	87.5	0.925	0.013	69.55	100	87.5
P100 (ms) R_VEP	0.91	<0.001	111.65	88.5	83.3	0.918	<0.001	113.7	95.2	87.5	0.925	0.013	108	100	87.5
N145 (ms) R_VEP	0.858	<0.001	152.1	92.3	62.5	0.897	<0.001	153.25	90.5	81.2	0.875	0.028	140.4	100	75
N75-P100 amp (μv) R_VEP	0.776	0.001	4.04	84.6	79.2	0.81	0.001	4.04	81	88.5	0.788	0.092	5.25	60	75
N75 (ms) L_VEP	0.773	0.001	70	76.9	79.2	0.738	0.014	70	76.2	81.2	0.925	0.013	73.05	100	87.5
P100 (ms) L_VEP	0.88	<0.001	111.6	88.5	83.3	0.872	<0.001	111.85	85.7	81.2	0.95	0.008	107	100	87.5
N145 (ms) L_VEP	0.755	0.002	148.75	80.8	66.7	0.735	0.015	149.05	76.2	68.7	0.925	0.013	141.45	100	75
N75-P100 amp (μv) L_VEP	0.787	0.001	4.19	84.6	79.2	0.817	0.001	4.12	81	87.5	0.788	0.092	5.21	80	75
I (ms) R_BAER	0.668	0.041	1.555	61.5	62.5	0.683	0.059	1.525	71.4	56.2	0.75	0.143	1.575	80	62.5
II R_BAER	0.952	<0.001	2.685	61.5	100	0.967	<0.001	2.685	71.4	100	0.825	0.057	2.83	80	75
III R_BAER	0.914	<0.001	3.815	73.1	100	0.957	<0.001	3.825	85.7	100	0.813	0.067	3.945	80	75
IV R_BAER	0.884	<0.001	4.825	57.7	100	0.897	<0.001	4.825	66.7	100	0.8	0.079	4.895	80	75
V R_BAER	0.916	<0.001	5.825	69.2	100	0.924	<0.001	5.825	81	100	0.963	0.007	5.975	80	100
I–III R_BAER	0.938	<0.001	2.31	80.8	95.8	0.955	<0.001	2.31	90.5	100	0.8	0.079	2.37	100	62.5
III–V R_BAER	0.868	<0.001	2.015	69.2	100	0.862	<0.001	2.02	81	100	0.888	0.023	2.055	80	75
I–V R_BAER	0.941	<0.001	4.35	76.9	100	0.943	<0.001	4.315	81	100	0.925	0.013	4.35	60	100
V/I (amp μv) R_BAER	0.568	0.409	1.635	42.3	62.5	0.571	0.462	1.625	47.6	56.2	0.625	0.464	1.635	60	75
I (ms) L_BAER	0.596	0.244	1.575	46.2	62.5	0.571	0.462	1.56	47.6	62.5	0.763	0.124	1.575	80	62.5
II L_BAER	0.955	<0.001	2.735	69.2	100	0.972	<0.001	2.735	81	100	0.875	0.028	2.81	100	75
III L_BAER	0.921	<0.001	3.825	73.1	100	0.957	<0.001	3.83	85.7	100	0.825	0.057	3.935	80	87.5
IV L_BAER	0.863	<0.001	4.83	61.5	100	0.869	<0.001	4.83	61.9	100	0.85	0.04	4.86	80	75
V L_BAER	0.925	<0.001	5.84	65.4	100	0.938	<0.001	5.84	76.2	100	0.95	0.008	5.985	100	87.5
I–III L_BAER	0.938	<0.001	2.275	80.8	95.8	0.952	<0.001	2.275	85.7	100	0.888	0.023	2.365	80	87.5
III–V L_BAER	0.765	0.001	1.995	0.538	100	0.796	0.002	1.995	61.9	100	0.625	0.464	2.08	60	62.5
I–V L_BAER	0.947	<0.001	4.36	76.9	100	0.949	<0.001	4.345	81	100	0.95	0.008	4.405	80	87.5
V/I (amp μv) L_BAER	0.699	0.016	1.645	69.2	79.2	0.656	0.108	1.645	66.7	68.7	0.813	0.067	1.645	80	100
N9 R_SSEP	0.571	0.388	8.995	65.4	41.7	0.598	0.312	8.995	66.7	43.7	0.463	0.826	8.995	60	50
N13 R_SSEP	0.63	0.116	13.005	65.4	66.7	0.676	0.07	13.05	71.4	68.7	0.45	0.77	13.005	40	62.5
N20 R_SSEP	0.642	0.086	18.985	65.4	66.7	0.704	0.036	18.935	71.4	68.7	0.4	0.558	18.335	20	87.5
N13–N20 R_SSEP	0.569	0.404	5.825	38.5	75	0.622	0.209	5.825	42.9	81.2	0.288	0.213	5.83	20	62.5
N9 L_SSEP	0.553	0.522	8.875	34.6	79.2	0.598	0.312	8.885	42.9	75	0.35	0.38	8.925	20	62.5
N13 L_SSEP	0.624	0.132	13.045	65.4	62.5	0.671	0.078	13.045	71.4	68.7	0.425	0.661	13.08	40	50
N20 L_SSEP	0.647	0.076	18.705	50	87.5	0.711	0.03	18.705	57.1	81.2	0.375	0.464	18.325	20	87.5
N13–N20 L_SSEP	0.583	0.317	5.805	42.3	83.3	0.64	0.15	5.805	47.6	87.5	0.275	0.188	5.805	20	75

L, left; PIGD, postural instability gait disorder variant of IPD; R, right; SN, sensitivity; SP, specificity; TD, tremor dominant variant of IPD

potential cutoff values determining postural stability in IPD and its subgroups (TD and PIGD). Lesser vestibular compensatory support in PIGD led to a more severe phenotype than TD.

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