

Making Predictions About Recovery: Is There HOAP?

Bruce E. Porch

Veterans Administration Medical Center, Albuquerque, New Mexico

Sheelagh Callaghan

University of New Mexico, Albuquerque, New Mexico

Once damaged, the brain, designed for survival, changes itself neurochemically and neurophysiologically in an attempt to readjust to its various breakdowns in processing, and tries to reprogram its circuits in an effort to once again reach operational levels. What factors retard or facilitate this recovery process are poorly understood. Some possible variables have recently been summarized by Darley (1972), Eisensen (1981), and Porch, Collins, Wertz and Friden (1980) to include age, education, site and extent of damage, type and amount of treatment, etc., most of which were suggested long ago.

Prior to 1960, the problem of predicting recovery from aphasia was not adequately studied. In part, this omission was due to the general view that all patients improve somewhat as "spontaneous recovery" occurs, but that there was considerable variation in this process and therefore prediction was difficult if not impossible to attempt. Another reason that recovery was poorly understood was the lack of reliable methods for measuring and describing changes over time. During the 1960's, improved psychometric evaluation techniques stimulated several studies dealing with recovery.

Schuell (1965a, 1965b) studied aphasic patients once they had become neurophysiologically stable, and reported that these patients often maintained the level of functioning that had been established after spontaneous recovery. Although Schuell recognized the importance of providing a prognosis to the patient's family early post onset, she did not address this problem, because she was aware of the numerous variables affecting recovery during the acute period.

In addition to the family's questions, the patient's employer is concerned about how soon the patient will be able to return to work (Aten, 1979). Earlier predictions also become necessary to serve as a foundation for rehabilitative planning and decision making (Porch *et al.*, 1980). Similar problems arise when medical-legal decisions have to be based upon the patient's present level of functioning, rather than the patient's potential capacity at some time in the future (Rada, Porch and Kellner, 1975).

During the late 1960's Porch (1970), feeling that the patient, his family, and the professionals associated with him needed earlier predictions to serve as a foundation for rehabilitative planning, formulated some hypotheses about recovery. His view was that the damaged brain will show its potential for improvement through its highest communicative performances. He felt that the highest subtest scores, regardless of modality, indicate a target level which could serve as a conservative prediction of the patient's level of functioning at some future date. This method of predicting the PICA Overall score from the high scores was called the High-Overall

Prediction or HOAP method, and was the first attempt to use statistical procedures for prediction. Porch and Porec (1977) report specific applications of the predictive information provided by the PICA to areas of testamentary capacity, levels of competency, quantification of degree of impairment, as well as differentiation of aphasia from nonaphasic states.

Porch (1978), in experimenting with other indices which might be useful in predicting change over time, hypothesized that a patient responding to PICA subtests which contained relatively homogeneous items should show his potential on a given task through his highest item scores in much the same way he shows his overall potential through his modality or subtest scores in the HOAP method. Porch also conjectured that the more variability there was within a subtest's scores, the more probable it was that positive change in that specific ability would eventually occur. He examined a variety of indices of intrasubtest variability and eventually determined that the differences between the highest item score and each of the other item scores (Peak Mean Difference) was the most practical expression of variability within subtests, and that such an index might be an indicant of potential subtest change.

Recently, other researchers (Wertz, Deal and Deal, 1980; Aten and Lyon, 1978) have studied some of these predictive methods and have raised questions as to their usefulness in predicting recovery. However, these studies had some methodological and interpretive limitations which have led to some uncertainties in the interpretation of their results and have left the clinician confused about whether predictions should be attempted.

Most clinical aphasiologists who use the PICA have relied on these predictive methods, in lieu of something better, even though we would like to have more precision in our predictions. Therefore, we asked ourselves whether we should abandon PICA predictions altogether, modify them, or retain them as they presently stand. The studies designed to answer our queries are presented in this paper. First, our purpose is to discuss previous articles that deal with HOAP prediction and Peak Mean Difference. Then, recent analyses of patient data over time are presented which have further examined the predictive validity of these methods. Finally, we shall attempt to reconcile differences, if they exist.

HIGH-OVERALL PREDICTION

HOAP predictions were first described by Porch (1973) a decade ago and were recently studied by Wertz, Deal and Deal (1980). This method uses the nine high percentiles of a large random sample of left hemisphere lesion patients to predict an eventual outcome level for a given patient. The prediction may be made either through the use of the High-Overall Tables in the PICA manual (Porch, 1973, pg. 113) or by using HOAP slopes--lines drawn on a recovery curve graph connecting the Overall (OA) percentile of each level at one month post onset with its corresponding Nine High percentile at six months post onset. For instance, the 50th percentile has a Nine High score of 13.13 which in the overall column is equal to the 82nd percentile. Therefore, a slope would be drawn from the 50th percentile at one month to the 82nd percentile at six months post onset. By using this strategy, a series of slopes may be constructed which enables one to estimate the six MPO target from points other than at just the one MPO, as in the High-Overall method. The two methods are identical when predictions are made from 1 MPO to 6 MPO. Wertz, Deal and Deal (1980) seemed unaware of this,

since they tested the two methods separately to compare them and found the two predictive results to be "essentially similar."

What is HOAP prediction? As with any prediction, this method should be thought of as a kind of actuarial approach in which one tries to make a best guess about some future outcome, recognizing that all subjects will not fall exactly on the predicted point, but rather, subjects will form a normal distribution around that point. In terms of HOAP prediction, we would expect to see a normal distribution with the mean at the predicted point and the standard deviation about ten.

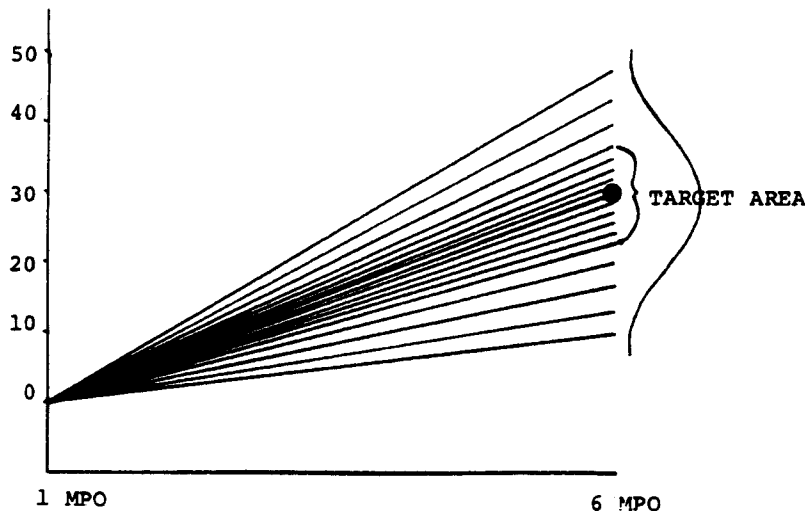


Figure 1. Theoretical Distribution of Scores Around the Target Point.

Figure two shows an actual distribution for 82 patients and how it fulfills these expectations. Two thirds of the time we would expect patients to fall within ten percentile points of the target, a finding that was verified by Wertz *et al.* (1980) last year, although they found this "frightening" and a sign that HOAP predictions were not practical.

It is not surprising that some patients either do not reach their target, or exceed their predicted target. The surprising thing is that HOAP predictions, using only one score, predict as well as they do. What we now need to do is to develop correction factors to improve the accuracy of predictions on more patients and thereby make the normal curve around the target more kurtotic. We must be cautious about simply condemning these approaches and abandoning them. Without them we are relegated to an earlier state of the art of talking about "good, fair or poor" prognoses with no numbers or reference points to offer our patients, or to those interested in their recovery. As long as we keep in mind that these are statistical best guesses and we continue to be conservative in their use, the advantages of having these interim methods far outweighs not having any predictions at all. Certainly we need to improve these methods and in this paper we hope to suggest some starting points.

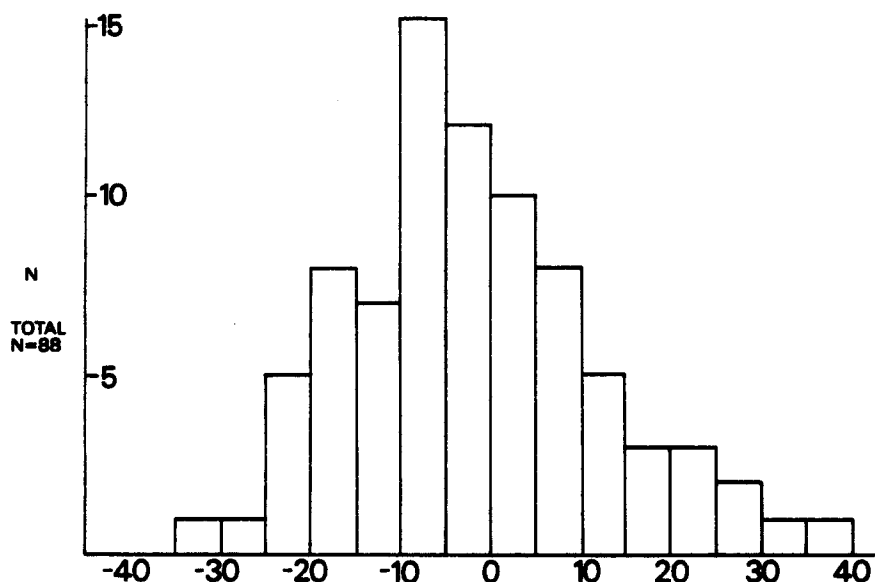


Figure 2. Actual Distribution of Scores Around a Target Point for 88 Left Hemisphere Patients.

The present study retrospectively examined the long term recovery patterns of fifty patients. Twenty-five had PICA test results compatible with left hemisphere lesions, and twenty-five had signs which Porch (1967) associated with bilateral or diffuse brain effects. The left and BL groups were separated into treated and untreated subgroups according to how much treatment each had received. Treated patients received at least 100 hours of individual treatment in the first six MPO and 200 hours of treatment by twelve MPO. Second, we subdivided the BL group according to how many BL tests each patient had during the recovery period. Since the number of tests varied from patient to patient, only the test results at one, three, six, nine, and twelve MPO were analyzed.

The test results of primary importance for prediction were at the one and six months post onset of recovery. As mentioned previously, the one month overall PICA score provided a six month target score. The six month overall score was then compared to this target score to result in a Target Difference score. Analysis of these difference scores was performed for each of the subgroups, with the results presented in Table 1. All numbers in Table 1 are PICA percentiles indicating the difference between the HOAP predicted target and the OA percentile at six and twelve MPO. Negative numbers indicate that patients were below the target and positive numbers show these patients to be above their predicted target. All of these percentiles are based on the 1973 norms, not the 1981 ones. The results were as follows:

1. Left hemisphere patients approximate the HOAP predictions more closely than do BL patients.
2. The more BL tests a patient has, the more he misses his HOAP target.

3. Treated patients approximate their targets better than do untreated patients.
4. Recovery continues after the usual six month plateauing occurs, especially in the left group and in BL patients who had only one BL test.

Table 1. A comparison of target difference scores (T_D) for treated and untreated brain damaged patients. The bilateral (BL) patients are divided according to how many BL PICA results were found among tests administered at 1, 3, 6, 9, and 12 MPO.

LESION	TREATMENT	6 MPO T_D	12 MPO T_D
LEFT	TREAT	1.77	7.00
	UNTREAT	9.46	5.14
	COMB.	5.62	1.33
BL 1	TREAT	3.60	2.20
BL 2		6.67	11.00
BL 3		9.67	5.00
BL T		6.09	5.44
BL 1	UNTREAT	13.40	1.00
BL 2		9.25	8.00
BL 3		17.70	12.33
BL U		13.08	4.50

What are the implications of these findings as far as prediction is concerned? If a patient with left hemisphere damage is treated, we may want to add a five or ten percent correction to our prediction. If the patient has BL tests at any time, we may want to correct our prediction downward five or ten percent, depending upon how many BL tests were seen. With specified left hemisphere patients, the current formulae still seem to be the best guess on an actuarial basis.

INTRASUBTEST VARIABILITY AND RECOVERY

Another variable which may be related to recovery is intrasubtest variability, described by Porch in 1978. He suggested that on a task which has high internal consistency, a patient should achieve relatively homogeneous scores, since equally difficult stimuli should tend to interact with the patient's system in similar ways. Further, variability within a homogeneous subtest indicates processing problems of various kinds, and the highest scores within the subtest indicate a potential functioning level for that patient on that task.

Although there are several possible indices of intrasubtest variability, the easiest to compute and the most discussed method is the Peak Mean Difference (PMD) score which is the cumulative difference between the highest item score on a subtest and each of the other nine item scores. It is calculated by subtracting the subtest mean from the peak or highest score; hence the label "Peak Mean Difference" score. On the surface, it would seem that the PMD might be a good predictor. However the question of what it predicts, and how it does so remains to be answered.

Aten and Lyon (1978) tried to use PMD to predict changes in scores and met with disappointing results, which they attributed to problems in the design of the PICA itself. On close inspection, however, the poor prediction they got can be explained more easily on other grounds. First, Aten and Lyon used pooled data of 24 patients, not realizing that PMD scores in some patients differ in the way they change over time. Patients with lesions in the central middle cerebral area of the brain have processing and motoric problems which act to depress both overall scores and variability scores early. However, as the motoric problems resolve or are compensated for, and more processing is attempted, both overall and variability scores increase, thus creating an "F" shaped pattern on the recovery curve graph. On the other hand, posterior lesion patients tend to start recovery with processing problems but no motoric deficits. Initially, their scores are depressed but they exhibit high variability scores which over time descend as the overall scores increase. This produces a "C" type pattern of recovery. A third group, which remains to be fully understood, has little change in either the OA or PMD over time, maintaining a "FLAT" pattern as presented in Figure 5. Obviously, if PMD data containing all three of these patterns is analyzed, then the results will be confounded, and a washout of possible significant effects occurs.

A second observation about PMD is that test changes are expected "at the fulcrum of the curve," on tasks which have fairly high subtest means and relatively low intrasubtest variability. Subtests with high PMD scores are too far down the patients curve to change at the time of the test, but will change later as the curve moves positively on the task continuum (Porch, 1981). In the Aten and Lyon study, subjects were at the 42nd percentile OA which puts their group response curve at a point in the test field where the tasks on the fulcrum of the curve are visual and auditory ones, both of which are within the gestural modality. Therefore, we would expect a priori that if PMDs were correlated with subtest and modality changes in an attempt to predict outcome, only those tasks just referred to should have significant correlations. As Aten and Lyon stressed in discussing their results, the only significant correlations they got were on... visual subtests... auditory subtests... and the gestural modality. This is exactly the findings we expect according to PICA theory.

In the Aten and Lyon study their sample mean OA percentile at 2 months post onset was 42, which when plugged into a HOAP slope prediction estimates a target percentile of 67. Their sample at 12 MPO had an OA percentile of 67.5.

Returning to study of subtypes of PMD patterns over time, we retrospectively analyzed recovery of 87 patients who had PICA test results over at least a six month period of time. Table 2 summarizes the descriptive test data on this sample which is divided into "C," "F," and "FLAT" PMD patterns and BL patients. All four groups have large ranges in all parameters. The "F" group has the lowest first test but exceeds the predicted OA mean of

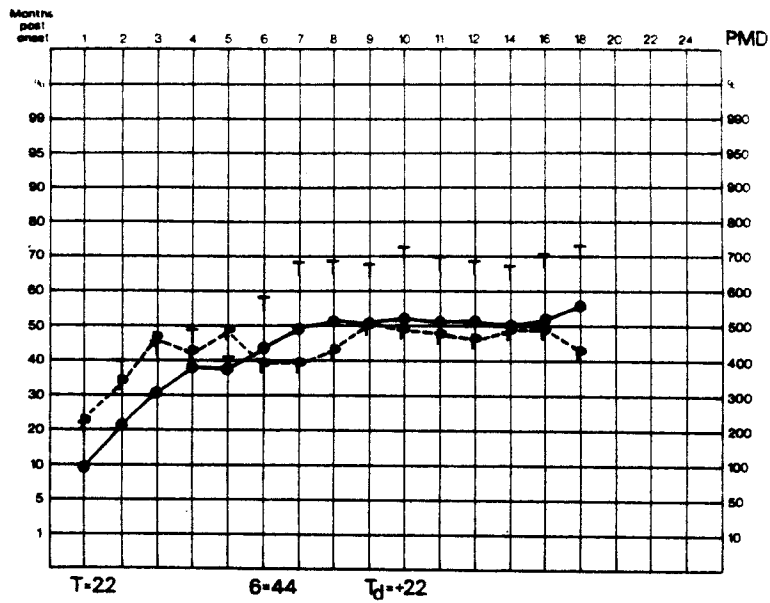


Figure 3. Rising Peak Mean Difference Producing an "F" shaped pattern.

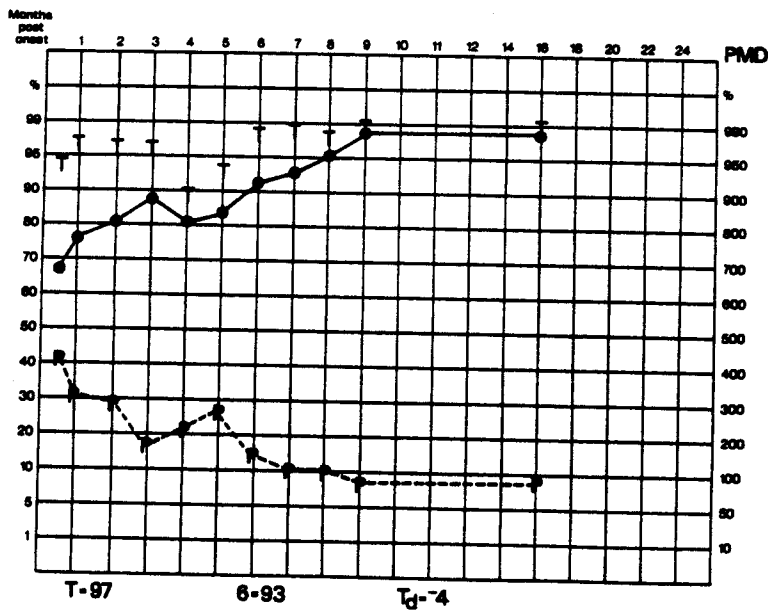


Figure 4. Falling Peak Mean Difference producing a "C" shaped pattern.

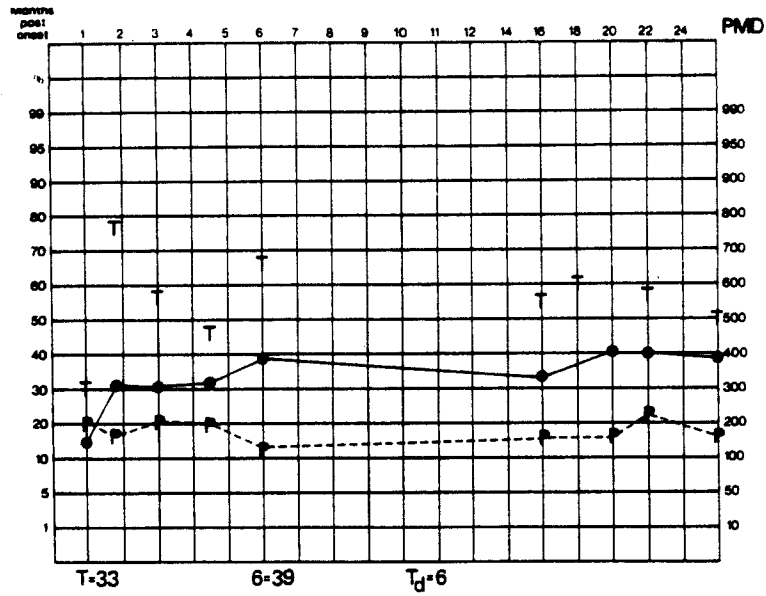


Figure 5. Flat Peak Mean Difference producing a "FLAT" shaped pattern.

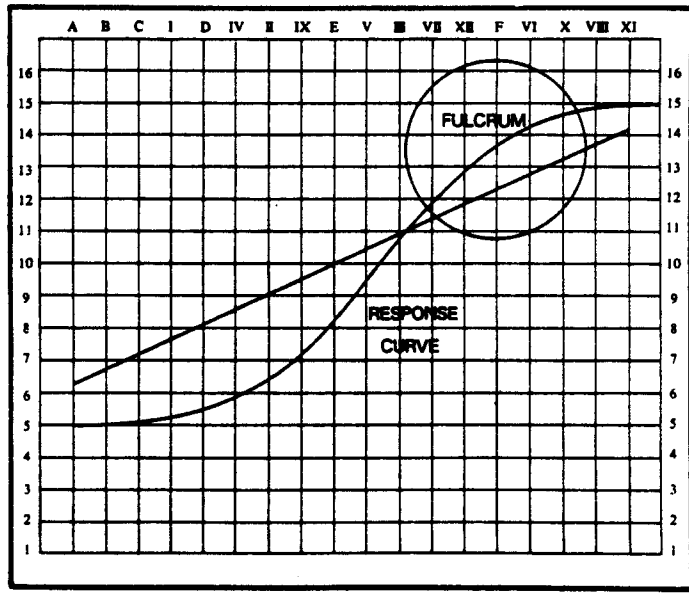


Figure 6. The fulcrum of the response curve where subtest changes first occur.

42nd percentile by 4 percentile points. The "C" group missed the 80th percentile by 5 points. The "FLAT" group would normally be predicted to reach the 83rd percentile, but only reached the 63rd percentile. The BL group, as could be expected after corrections are applied to the prediction, missed the 57 percentile target by 12 points.

Table 3 summarizes group data on Target Difference (T_D) or how far each group missed the six month target. Overall Difference (OA_D) between the lowest and the highest OA percentile each patient had at any point in time, the highest PMD the patients had at any time (PMD_{hi}), and the PMD_D which is the difference between the patients highest and lowest PMD at any time. Note that only the "F" group exceeds the six month targets. This group also had the largest OA differences. Although the "F" group had the lowest PMD at one month post onset, they eventually achieved the highest PMD and the most PMD change.

The relationships among these variables were examined for the total sample studied and the results of correlational analyses are presented in Table 4. A summary of these correlations follows.

1. The more the overall changes the more you miss the target.
2. The higher the PMD is at any time, the more you miss the target.
3. The more the PMD changes, the more you miss the target.
4. The higher the patient's PMD_{hi} , the more OA change occurs.
5. The higher the PMD, the more OA change occurs.
6. The more PMD changes, the more the OA changes.
7. The higher the PMD_1 , the higher the PMD_{hi} .
8. The higher the PMD_1 , the greater the PMD change.

Table 5 shows the correlations for the "C" group only. This group starts with a high PMD which falls to about the 200 level. The main results for this subgroup are these:

1. The more the OA changes, the more you miss the target.
2. The more the PMD changes, the more you miss the target.
3. The higher the PMD_1 , the more the PMD changes.
4. The higher the patient's PMD_{hi} , the more the OA changes.

Table 6 offers the correlations of the "F" group that has a rising PMD during recovery. This is the group which in earlier tables showed the best recovery. These correlations suggest the following:

1. High variability is related to the greatest OA change.
2. The higher the PMD, the greater the PMD change.

The "FLAT" group whose PMD stays fairly constant during recovery yielded the data in Table 7. The expected finding, that the highest PMD tends to be related to the PMD_1 , proved true.

Finally, as would be expected in the BL group, Table 8 shows that the more the intrasubtest variability (PMD_{hi}), the greater the OA change. Also, the more the PMD changes, the more the OA changes. Lastly, the higher the maximum variability (PMD_{hi}), the more change there is in variability.

Table 2. Descriptive data on the four groups studied showing PICA Overall Percentiles at 1 and 6 MPO and Total Peak Mean Difference at 1 MPO.

		"C"	"F"	"FLAT"	"BL"
OA ₁	\bar{X}	48.6	19.9	51.6	29.4
	S	17.4	13.4	23.7	26.9
	r	13-76	3-48	14-92	2-88
OA ₆	\bar{X}	74.9	46.3	63.2	44.6
	S	15.7	16.5	21.5	30.1
	r	20-98	23-77	35-98	4-89
PMD ₁	\bar{X}	410.8	297.9	298.6	334.8
	S	99.0	119.9	79.7	93.4
	r	195-631	138-561	145-430	175-528
N		38	15	16	18

Table 3. Comparison of Target Difference (T_D), Overall Difference (OA_D), Highest Peak Mean Difference (PMD_{hi}), and Maximum Peak Mean Difference Change Over Time (PMD_D).

		"C"	"F"	"FLAT"	"BL"
T _D		-1.2	3.8	-10.8	-5.3
		14.9	14.8	9.2	9.6
		-25-38	-19-26	-31-7	-29-11
OA _D		32.8	34.7	14.9	21.2
		16.5	14.1	9.9	14.5
		8-79	18-57	3-36	1-50
PMD _{hi}		443.9	494.6	349.7	431.2
		94.5	83.9	106.6	102.9
		313-631	290-602	170-514	252-628
PMD _D		223.5	256.5	117.9	183.2
		102.6	124.1	91.4	106.4
		46-469	64-440	6-315	37-407

Table 4. Correlations between Target Difference Score, Overall Difference, Peak Mean Difference at 1 MPO, Highest Peak Mean Difference and Maximum Peak Mean Difference Change Over Time for all subjects.

"PEARSON r" All Subjects					
	T _D	OA _D	PMD ₁	PMD _{hi}	PMD _D
T _D	1.00	.70 ^{xxx}	-.11	.35 ^{xxx}	.57 ^{xxx}
OA _D		1.00	.23 ^{xx}	.59 ^{xxx}	.56 ^{xxx}
PMD ₁			1.00	.45 ^{xxx}	.08
PMD _{hi}				1.00	.64 ^{xxx}
PMD _D					1.00

xxx p < .001 xx p < .01 x p < .05

Table 5. Correlations between Target Difference, Overall Difference, Peak Mean Difference at 1 MPO, Highest Peak Mean Difference and Maximum Peak Mean Difference Change Over Time for the "C" Group.

"SPEARMAN r"					
"C"	T _D	OA _D	PMD ₁	PMD _{hi}	PMD _D
T _D	1.00	.79 ^{xxx}	.09	.44 ^{xx}	.61 ^{xxx}
OA _D		1.00	.14	.47 ^{xxx}	.47 ^{xx}
PMD ₁			1.00	.76 ^{xxx}	.19
PMD _{hi}				1.00	.46 ^{xx}
PMD _D					1.00

xxx p < .001 xx p < .01

Table 6. Correlations between Target Difference, Overall Difference, Peak Mean Difference at 1 MPO, Highest Peak Mean Difference and Maximum Peak Mean Difference Change Over Time for the "F" Group.

"SPEARMAN r"					
"F"	T _D	OA _D	PMD ₁	PMD _{hi}	PMD _D
T _D	1.00	.51 ^x	-.55 ^x	.21	.41
OA _D		1.00	.04	.65 ^{xx}	.53 ^x
PMD ₁			1.00	-.11	-.39
PMD _{hi}				1.00	.80 ^{xxx}
PMD _D					1.00

xxx_p < .001

xx_p < .01

x_p < .05

Table 7. Correlations between Target Difference, Overall Difference, Peak Mean Difference at 1 MPO, Highest Peak Mean Difference and Maximum Peak Mean Difference Change Over Time for the "FLAT" Group.

"SPEARMAN r"					
"FLAT"	T _D	OA _D	PMD ₁	PMD _{hi}	PMD _D
T _D	1.00	.49 ^x	-.53 ^x	-.29	.33
OA _D		1.00	.09	.35	.27
PMD ₁			1.00	.86 ^{xxx}	-.12
PMD _{hi}				1.00	.21
PMD _D					1.00

xxx_p < .001

x_p < .05

Table 8. Correlations between Target Difference, Overall Difference, Peak Mean Difference at 1 MPO, Highest Peak Mean Difference and Maximum Peak Mean Difference Change Over Time for the "BL Group.

"SPEARMAN r"					
"BL"	T_D	OA_D	PMD_1	PMD_{hi}	PMD_D
T_D	1.00	.36	-.16	.20	.49 ^x
OA_D		1.00	.34	.77 ^{xxx}	.55 ^{xx}
PMD_1			1.00	.26	.03
PMD_{hi}				1.00	.66 ^{xxx}
PMD_D					1.00

xxx_p < .001

xx_p < .01

x_p < .05

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