CARDIOVASCULAR DISEASE AND INTELLIGENCE

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It has long been suspected that age-related physiological pathology may account for some of the decrements in cognitive performance observed in elderly subjects. Several investigators have reported intellectual and psychomotor deficits in aged subjects with cardiovascular disease (hereafter abbreviated CVD); however, our previous attempt to study the effects of CVD upon cognitive functioning in a cross-sequential design failed to yield an observable relationship between CVD and change in performance on tests of intellectual ability over a 14 year period. The subjects in that study participated on three occasions--1956, 1963, 1970--and thus probably were a highly select group of individuals (cf Riegel and Riegel, 1972; Schaie, Labouvie, and Barrett, 1973). None of the males in the two oldest cohorts (year of birth prior to 1900) evidenced CVD symptomatology until after 1970, and it is therefore possible that CVD-related attrition from the longitudinal sample might be suppressing a CVD effect in that sample. The purpose of the present investigation is to explore the frequency of CVD diagnoses in subjects who dropped out of the sample, and to determine the relationship between CVD and cognitive abilities in an expanded sample, -- i.e. both those subjects who participated in all three test occasions , and those who dropped out of the sample after the second test occasion.

METHOD

Since the incidence of diagnosable CVD is age-related, it was decided that only those subjects over 42 years of age at the time of first testing would be included in the present investigation. Medical health histories were available for 85 such men and women who had participated at all three times of measurement,

and for 70 men and women who had dropped out of the study after the first two times of measurement. A detailed description of the methods for translating medical health records into quantifiable coded variables has been described previously. The incidence of CVD was established by scanning the medical records for CVD diagnosis classifications from the ICDA coding system. Classification was then made for the time period in which the CVD symptom first appeared--on or before 1963, on or before 1970, and after 1970.

Measurement Variables

The present study reports the results on 10 dependent variables. Five of these are subtests of the PMA-- Verbal Meaning (V), Space (S), Reasoning (R), Number (N), and Word Fluency (W); two, Intellectual Ability (IQ) and Educational Aptitude (EQ), are derived from the PMA by the equations IQ = V + S + 2R + 2N + W, and EQ = 2V + R; and the remainder, Motor-Cognitive Rigidity (MCR), Personality Perceptual Rigidity (PPR), and Psychomotor Speed (PS), are factor scales from Schaie's Test of Behavioral Rigidity.

RESULTS

Relationship between CVD and Experimental Mortality

In order to test the hypothesis that subjects from the attrited samples would evidence a disproportionate frequency of CVD, contingency tables were compiled separately by sex for the factors of CVD and Participation (See Table 1). These tables confirmed a relationship between Participation and the presence of CVD across the duration of the longitudinal study for males. For example, by 1963 nearly 43% of the male dropouts exhibited CVD, compared to only 17.5% of those males retested. By 1973 fully 80% of the male dropouts had signs of CVD, while only 50% of those retested evidenced signs of CVD. In contrast, differences were not significant for women, although the trend was in the expected direction with a slightly higher percentage of female dropouts having CVD symptoms.

Analyses of Variance of Dependent Measures

The most appropriate design to analyze these data would be an expansion of a cross-sequential design to include CVD as a blocking factor; however, cell sizes did not permit such differentiation. Consequently, three conceptually different sets of analyses were undertaken in which subjects were collapsed over one of the blocking factors and the effects of the others were tested. Repeated measures analyses of variance designs for each dependent measure were:

CVD x Participation x Sex x Time (collapsed over cohort)

CVD x Cohort x Sex x Time (collapsed over participation)

Cohort x Participation x Sex x Time (collapsed over CVD)

Keep in mind that in these designs, cohort is a factor confounding age changes and cohort differences, as in a cross-sectional design, while Time of measurement confounds age changes between times of testing with environmental effects over the same time period. Findings for the main effects involving Sex, Cohort, Time of Measurement, and Participation are generally consistent with similar effects reported previously (cf Schaie and Strother, 1968). Only results specifically pertaining to effects of CVD will be reported here.

In the CVD x Participation design, main effects significant at or beyond the 1% level of confidence for CVD were obtained on all PMA subtests and on the MCR factor from the TBR. A robust CVD x Time interaction was obtained for PS. Significant interactions for CVD x Participation were not observed on any dependent measure (see Table 2, Figure 1).

The results from the CVD x Cohort analysis (Table 3, Figure 2) suggested that controlling for age differences reduces the amount of variance accounted by CVD, nevertheless, reliable effects for CVD were observed for Number and Intellectual Ability at the 1% level of confidence, and 5% level trends were found for Verbal Meaning, Reasoning, Educational Aptitude, and MCR. Again, a robust CVD x Time

interaction was obtained for Psychomotor Speed. The mean differences representing these effects were in the expected direction, with CVD subjects performing at a lower level than subjects without CVD, and evidencing greater time related decline in Psychomotor Speed.

The results of these ANOVA's indicated that the inclusion of data from subjects who did not participate in the third testing revealed CVD effects which had been obscured by the attrition of significant numbers of subjects with CVD from the longitudinal sample. One might speculate that, had medical data been available for subjects who dropped out after the first time of testing, the effects would have been greater.

The most conservative estimates of the CVD effects in these data are obtained in the CVD x Cohort analysis, wherein age differences in CVD diagnosis are controlled by the use of Cohort as a factor in the analysis. The robust main effects for CVD on Number and Intellectual Ability in this analysis, as well as the CVD x Time interaction for Psychomotor Speed, are in agreement with other studies which have isolated CVD-related differences in cognitive ability (e.g., Spieth, 1965; Wilkie and Eisdorfer, 1971) and suggest effects for CVD which are independent of chronological age.

Nevertheless, the most impressive effects are the main effects involving age differences. In both the Cohort x Participation and CVD x Cohort designs, main effects for Cohort/Age were observed for all dependent measures at or beyond the 1% level of confidence, with the sole exception of PPR in the CVD x Cohort analysis, where the level of confidence was 5%.

DISCUSSION

Caution is required in the interpretation of these results, since their pattern does not necessarily confirm a causal relationship between CVD and a decline in cognitive ability. The strongest indication for a time-bound, CVD-mediated decrement is the CVD x Time interaction for Psychomotor Speed. The remaining

effects, while not incompatible with the decremental model, do not necessarily support it. No differential decline was seen in subjects with or without CVD pathology, and further, subjects in the younger cohort with CVD show small time-related increments in performance between measurement points on Verbal Meaning and Number.

We cannot therefore conclude that CVD produces cognitive deficits, since these results leave open the possibility that some unidentified factor confounded with CVD may actually be the locus of the observed effects. One possible confound is socioeconomic status (SES) differences between disease groups; however, an analysis of these data using two demographic variables, occupational status and yearly family income as covariates showed that both of these variables reduce the accountable variance for CVD on most subtests, but do not eliminate the effects of CVD for Number and IQ.

Other factors which may be correlated with CVD might include depressive affect (Botwinick and Storandt, 1974), behavioral typologies (e.g., Type A-Type B distinctions, cf. Rosenman and Friedman, 1971), and performance deficits, perhaps due to differential responses to periods of stress and overarousal, as suggested by Eisdorfer and Wilkie (1976). Unpublished data from our project suggests that CVD in "disengaged" elderly women appears to be more frequent than in elderly women as a whole. In these subjects, it may be equally likely that poor performance is due to the cumulative effects of impoverished environmental circumstances as it is to CVD pathology. Certainly, further investigation of other possible explanations is in order before the effects observed for CVD in this study are interpreted as evidence for a physiologically mediated decline in intelligence.

The question remains as to the explanation of the lack of CVD effects found in the initial examination of our 14 year longitudinal data. Certainly the attrition of disproportionate numbers of individuals with CVD from the sample plays a

crucial role. This attrition, however, does not offer a sufficient explanation since subjects with CVD were present in the 14 year sample. If CVD affects cognitive behavior discontinuously, as suggested by Birren (1964), then subjects in the 14 year sample may not have had sufficient pathology to produce cognitive deficits. There were, however, several subjects in the sample with severe levels of CVD pathology, and if we assume that the CVD effects in the 7 year sample are not artifactual, it seems more likely that the relative stability in performance in these subjects is best explained by a decrement with compensation model (cf. Schaie, 1973).

We find little support in these data for the proposition that age differences in performance should be considered secondary to cardiovascular pathology. The robust effects for cohort membership in the CVD x Cohort analyses reported above would contraindicate any assertion that CVD by itself can account for age differences in intelligence. Similarly, Cohort x Time interactions and Time of Measurement main effects found in earlier analyses of these data (Schaie and Strother, 1968) were unaffected by the use of CVD as a blocking factor. Furthermore, hierarchical ANOVA's with these data, entering CVD before Cohort, still yielded large age differences in cognitive ability. The robust effects for age and cohort related factors in these analyses indicate that a substantial amount of previously observed age differences may be independent of cardiovascular disease. Thus, although CVD can account for differences in adult cognitive behavior, other socio-psychological antecedent variables may also be useful in explaining the source of these age-related effects (Baltes and Labouvie, 1973).

TABLE 1
CONTINGENCY TABLES: CVD BY PARTICIPATION
(1956, 1957-1963)

		Males		Females	
		Dropouts	Survivors	Dropouts	Survivors
1956					
No CVD	N[a]	30	37	31	39
	ROW[b]	44.8%	55.2%	44.3%	55.7%
	COL[c]	85.7%	92.5%	88.6%	86.7%
	TOT[d]	40.0%	49.3%	38.7%	48.7%
CVD	N	5	3	4	6
	ROW	62.5%	37.5%	40.0%	60.0%
	COL	14.3%	7.5%	11.4%	13.3%
	TOT	6.7%	4.0%	5.0%	7.5%
	X^2 = .33 (1 d.f.); N. S.			X^2 = .01 (1 d.f.); N. S.	
1957-1963					
No CVD	N	20	33	19	31
	ROW	37.7%	62.3%	38.0%	62.0%
	COL	57.1%	82.5%	54.3%	68.9%
	TOT	26.7%	44.0%	23.7%	38.7%
CVD	N	15	7	16	14
	ROW	68.2%	31.8%	53.3%	46.7%
	COL	42.9%	17.5%	45.7%	31.1%
	TOT	20.0%	9.3%	20.0%	17.5%
	X^2 = 4.63 (1 d.f.); p .05			X^2 = 1.22 (1 d.f.); N. S.	

a - the absolute number or frequency of individuals in a cell.

b - the relative percentage of individuals across a row in a given cell.

c - the relative percentage of individuals down a column in a given cell.

d - the percentage of the total number of individuals in a table within the cell.

TABLE 1 (CONT'D)
CONTINGENCY TABLES: CVD BY PARTICIPATION
(1964-1970; 1971-1973)

		Males		Females	
		Dropouts	Survivors	Dropouts	Survivors
1964-1970					
No CVD	N	11	26	14	22
	ROW	29.7%	70.3%	38.9%	61.1%
	COL	31.4%	65.0%	40.0%	48.9%
	TOT	14.7%	34.7%	17.5%	27.5%
CVD	N	24	14	21	23
	ROW	63.2%	36.8%	47.7%	52.3%
	COL	68.6%	35.0%	60.0%	51.1%
	TOT	32.0%	18.7%	26.2%	28.7%
		$X^2 = 7.13$ (1 d.f.); $p<.01$		$X^2 = .32$ (1 d.f.); N .S.	
1971-1973					
No CVD	N	7	20	7	17
	ROW	25.9%	74.1%	29.2%	70.8%
	COL	20.0%	50.0%	20.0%	37.8%
	TOT	9.3%	26.7%	8.7%	21.2%
CVD	N	28	20	28	28
	ROW	58.3%	41.7%	50.0%	50.0%
	COL	80.0%	50.0%	80.0%	62.2%
	TOT	37.3%	26.7	35.0%	35.0%
		$X^2 = 6.05$ (1 d.f.); $p<.05$		$X^2 = 2.18$ (1 d.f.); N. S.	

TABLE 2

CVD X PARTICIPATION X SEX X TIME
ANOVA
(CVD on basis of presence by 1963)

F RATIOS

Effect	V	S	R	N	W	IQ	EQ	MCR	PPR	PS
C	12.20***	6.90**	9.14**	17.64***	11.11***	19.27***	12.70***	9.76**	1.73	4.69*
P	10.96***	5.19*	18.13***	3.74	5.47*	13.03***	13.06***	11.19***	7.48**	8.16**
S	<1	7.11**	<1	<1	1.17	<1	<1	1.50	<1	1.67
CP	2.90	<1	<1	1.57	2.32	1.54	2.38	4.12*	<1	4.03
CS	4.16*	<1	1.12	<1	<1	<1	3.52	2.10	<1	<1
PS	<1	1.49	<1	<1	8.44**	2.31	<1	<1	3.76	1.95
CPS	1.45	<1	<1	<1	2.31	<1	1.09	1.18	<1	<1
$MS_{E(B)}$	143.97	107.40	120.06	143.50	159.14	142.73	141.41	101.80	133.78	134.65
T	<1	5.42*	13.17***	1.84	70.75***	16.46***	1.55	2.34	19.68***	122.42***
TC	<1	3.26	<1	1.39	2.46	4.50*	<1	1.28	<1	7.45**
TP	3.25	<1	1.91	3.19	<1	<1	1.92	<1	<1	3.67
TS	2.19	2.94	<1	<1	<1	<1	1.84	<1	<1	3.90
TCP	2.28	<1	1.18	<1	1.01	<1	1.52	<1	<1	<1
TCS	<1	<1	<1	<1	1.02	<1	<1	2.15	<1	1.07
TPS	<1	<1	1.62	3.58	<1	<1	<1	1.51	<1	<1
TCPS	<1	<1	<1	<1	1.22	<1	<1	1.05	<1	<1
$MS_{E(W)}$	24.39	23.70	11.21	18.42	23.77	13.76	19.53	38.61	23.02	18.10

* $p < .05$
** $p < .01$
*** $p < .001$

TABLE 3

CVD X COHORT X SEX X TIME
ANOVA
(CVD on basis of presence by 1963)
F RATIOS

Effect	V	S	R	N	W	IQ	EQ	MCR	PPR	PS
CVD	4.12*	2.23	4.21*	7.52**	3.17	7.40**	4.68*	4.58*	1.11	<1
CHRT	22.74***	9.99**	25.27***	13.75***	9.97**	28.31***	25.56***	24.50***	5.33*	26.50***
S	<1	5.29*	<1	<1	1.43	<1	<1	<1	<1	5.24*
CC	<1	<1	<1	1.16	<1	<1	<1	<1	2.41	<1
CvS	<1	<1	<1	<1	<1	<1	<1	<1	<1	<1
CtS	<1	<1	<1	<1	1.56	<1	<1	3.01	<1	<1
CCS	<1	<1	<1	<1	<1	<1	<1	1.03	<1	<1
$MS_{E(B)}$	133.60	101.76	108.62	134.73	160.61	127.99	128.90	90.92	131.82	120.16
T	<1	4.65*	10.23**	<1	50.01***	11.03***	<1	3.55	16.63***	97.78***
TCv	<1	1.95	<1	<1	1.13	1.82	<1	<1	<1	6.73**
TCt	8.01**	1.05	<1	3.99*	<1	5.19*	7.26**	3.40	3.45	1.46
TS	<1	2.21	<1	<1	<1	<1	<1	<1	<1	3.48
TCC	1.74	<1	1.51	2.34	<1	<1	1.21	2.93	1.01	<1
TCvS	<1	<1	<1	<1	<1	<1	<1	4.61*	<1	<1
TCtS	<1	<1	<1	<1	<1	<1	<1	<1	<1	1.78
TCCS	<1	<1	<1	<1	<1	<1	<1	<1	<1	<1
$MS_{E(W)}$	23.74	23.42	11.09	18.46	23.85	13.30	18.92	38.15	21.89	18.34

*p < .05
**p < .01
***p < .001

FIGURE 1A
CVD X PARTICIPATION X SEX X TIME

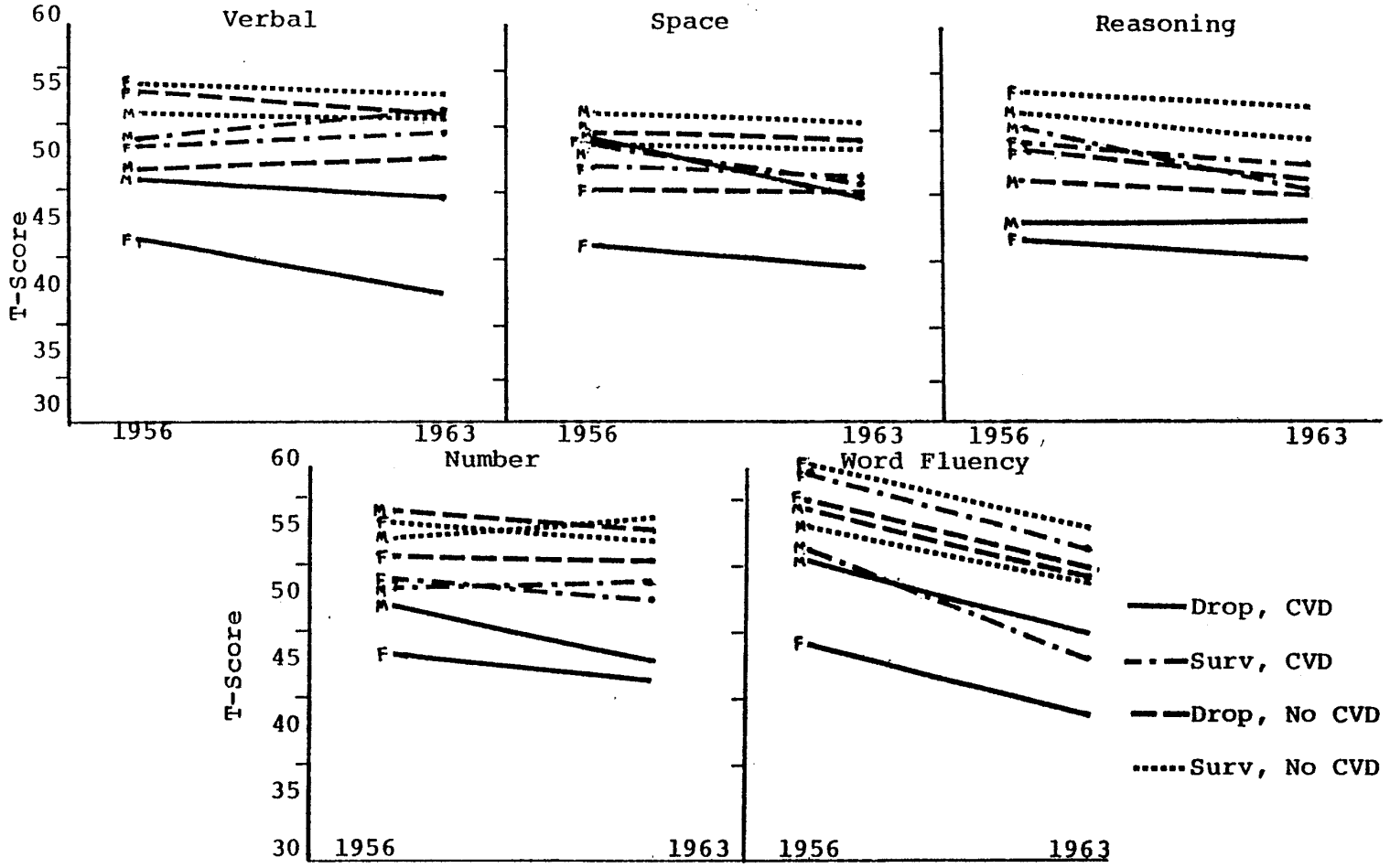


FIGURE 1B
CVD X PARTICIPATION X SEX X TIME

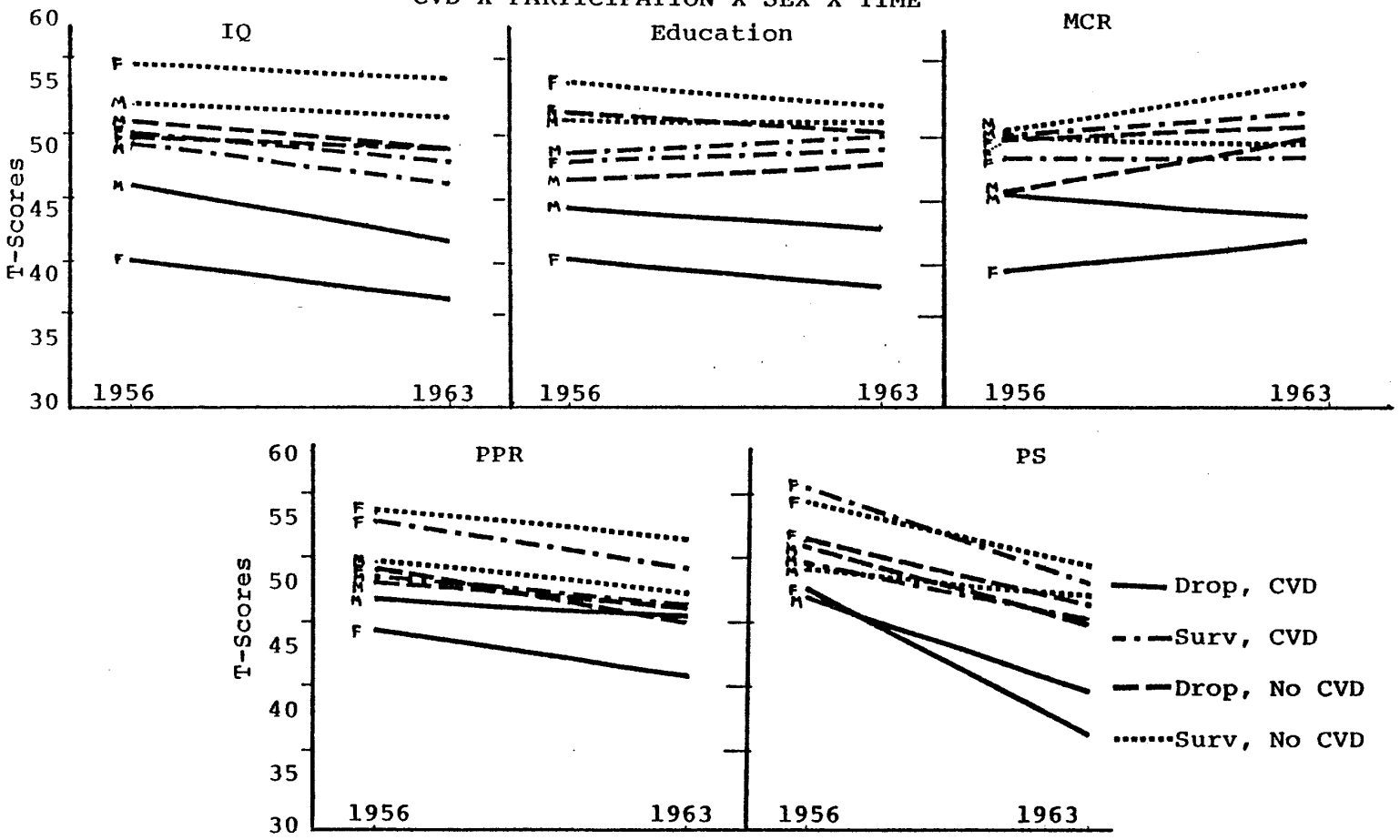


FIGURE 2A

CVD X COHORT X SEX X TIME

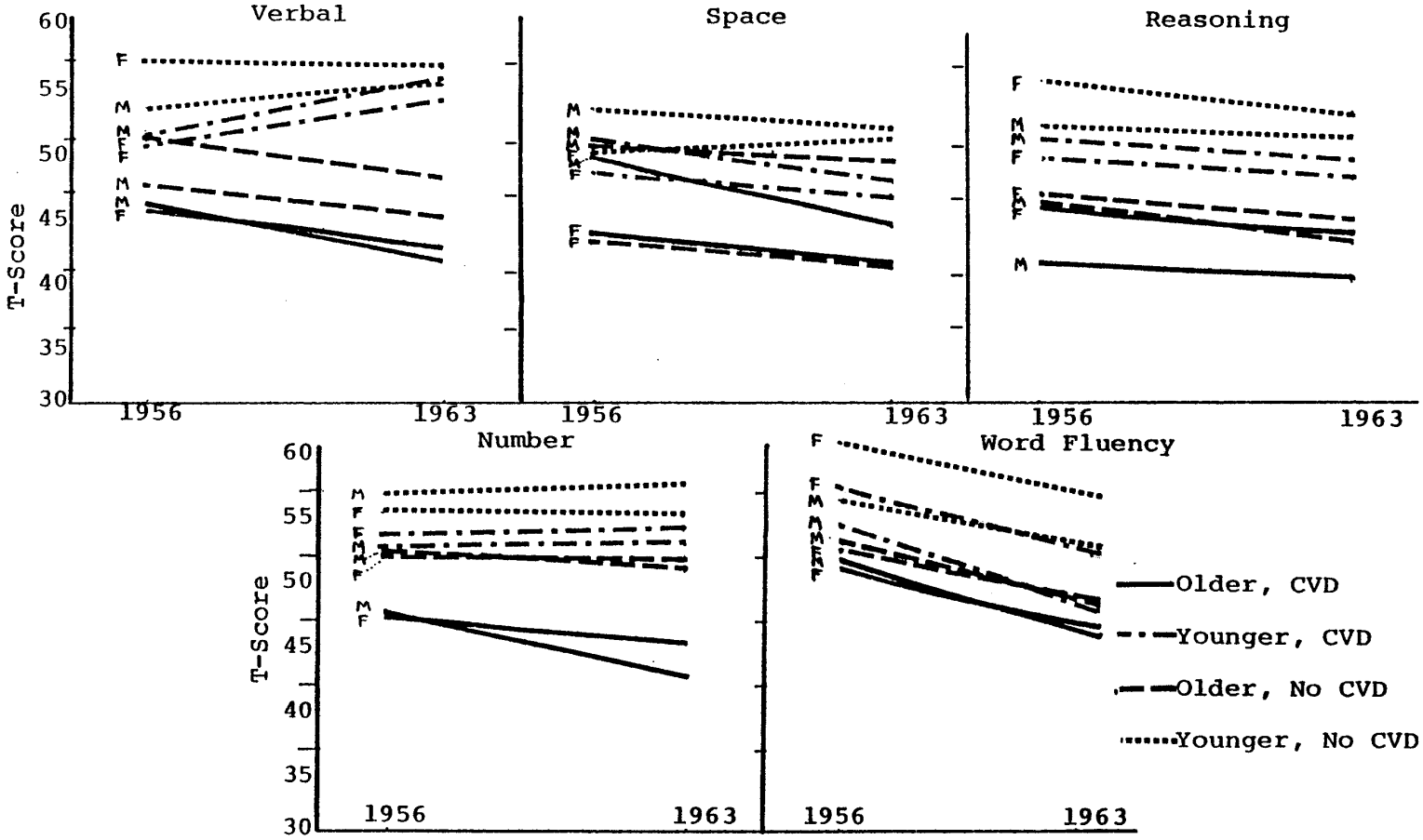
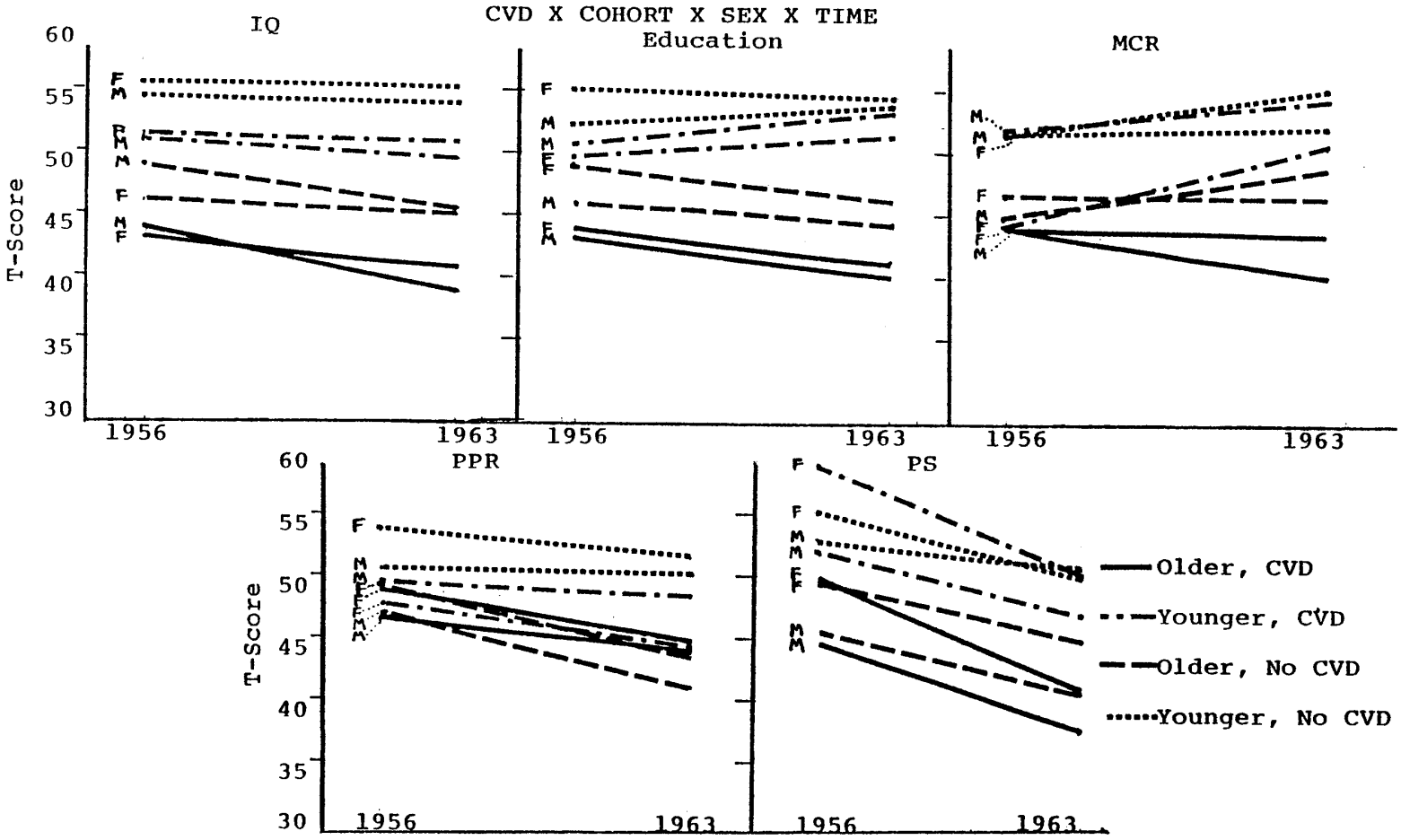


FIGURE 2B

CVD X COHORT X SEX X TIME



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