

INTELLECTUAL CHANGE IN THE SENIUM

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Until recent years it has been generally assumed that intellectual development and decline ran a relatively smooth and predictable course. Childhood was a period of rapid, uniform intellectual growth followed by a period of slower development during adolescence and perhaps early adulthood. After reaching its peak in the late teens or early twenties a gradual decline appeared. This intellectual decline was thought of as continuing at a constant rate throughout adult life until death intervened.

The factual basis for this conceptualization was derived mainly from the many cross-sectional studies of intellectual development using measures obtained from standardized tests of intelligence. The great majority of these studies were performed on children, considerably fewer on adults, and practically none on very old or senescent individuals. Such cross-sectional studies, while useful for normative purposes, only reveal performance differences between age groups. Nevertheless, they have been commonly interpreted so as to imply that these data are actually valid indicators of age changes. The fact remains, however, that age change can only be measured by longitudinal studies, i.e., the repeated testing of the same individuals at successive intervals during the life span. Fortunately some longitudinal data are available. Most important of these are the Bayley and Oden (1955) follow-up study of gifted children now in the mid-years of life, and Owens' (1953, 1962) retesting of college students thirty and forty years after their original examination. Neither of these studies report early decline in performance on tests of intelligence; indeed, they indicate continued growth rather than decline in the years of middle adulthood. These findings suggest that the drop in scores commonly reported on cross-sectional studies may reflect the environmental advantages of younger cohorts rather than the intellectual decline of their seniors.

In view of these results, the question remains at what point does the developmental curve turn downward, if indeed it does. It is not quite so absurd as it seems to question whether or not intellectual deterioration occurs in the normal course of life. Bear in mind that the two studies mentioned above do not support the hypothesis of intellectual decline in mid-adult life. Furthermore, a preliminary analysis of Moosehaven data, which will be reported on in more detail later, showed no significant change in performance in a group of men, aged 65 to 92, after a mean inter-test interval of 2.2 years (Arms, 1953). Indeed, the 26 younger subjects in this study, aged 65 to 75, showed a slight, but statistically insignificant improvement in performance on the second test. Surely these results would not allow one to predict confidently that deterioration would be found over a longer time interval.

Decline in Performance

The above analysis was a part of a study which was started at Moosehaven, a fraternal home for the aged, in 1949 and has been carried on there ever since. In fact the latest round of testing was conducted this summer on survivors of this study group (Arms, 1953; Kleemeier, Justiss, Jones and Rich, 1961; Obrist, Henry, and Justiss, 1961; Obrist, Busse, Eisdorfer, and Kleemeier, 1962). In all approximately 200 male residents of this institution were tested one or more times on the Wechsler-Bellevue Intelligence Scale for Adults. All were over age 65 and ranged up into the 90's.

In the 12 year period for which data are now available, 70 subjects were tested two or more times, with intervals of several years between tests. Thirteen of these were each tested four times. Since these latter subjects had received the most tests, their results were chosen for preliminary analysis, and are presented in Figure 1.

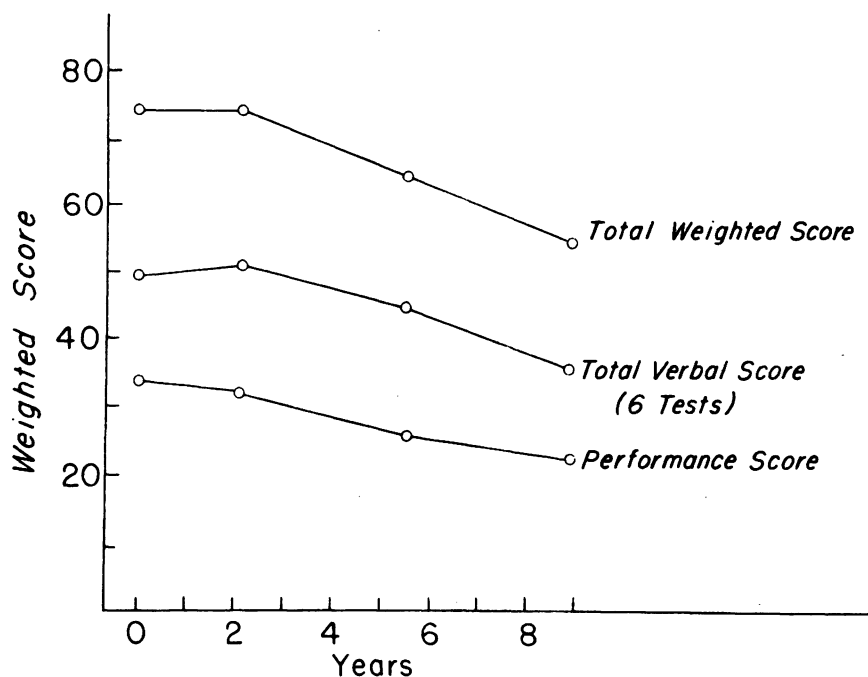


Fig. 1. Mean performance of 13 aged men on the Wechsler-Bellevue Intelligence Scale, Form I, on four successive tests.

This figure shows a decline in performance appearing by the third test; although, as would have been expected from the earlier analysis (Arms, 1953), no such trend would have been anticipated from the first two tests alone. Analyses of variance of these results show that the score differences on all three measures shown are significant at the .001 level.

These curves pose a problem. Why should there be a change in the rate of decline at the end of the second test? This is particularly curious, because of the wide age range of subjects represented in the curves. Does it mean that all subjects, regardless of age, experience a change at the end of the second test; or are some, but not all, subjects influencing the rate of change?

Individual curves

An answer is suggested in Figure 2, in which are shown the curves of nine subjects who were included in Figure 1 and who were living on January 1, 1961. Here we see that while there is a general drop in test score, considerable individual variation exists. Furthermore, the shape of the curve seems to be independent of the age of the subject.

Compare these curves with those shown in Figure 3, which represent the performance of four subjects who died shortly after the final test of the series. Each of these is characterized by a marked drop in performance and is distinctly different from those shown in Figure 2.

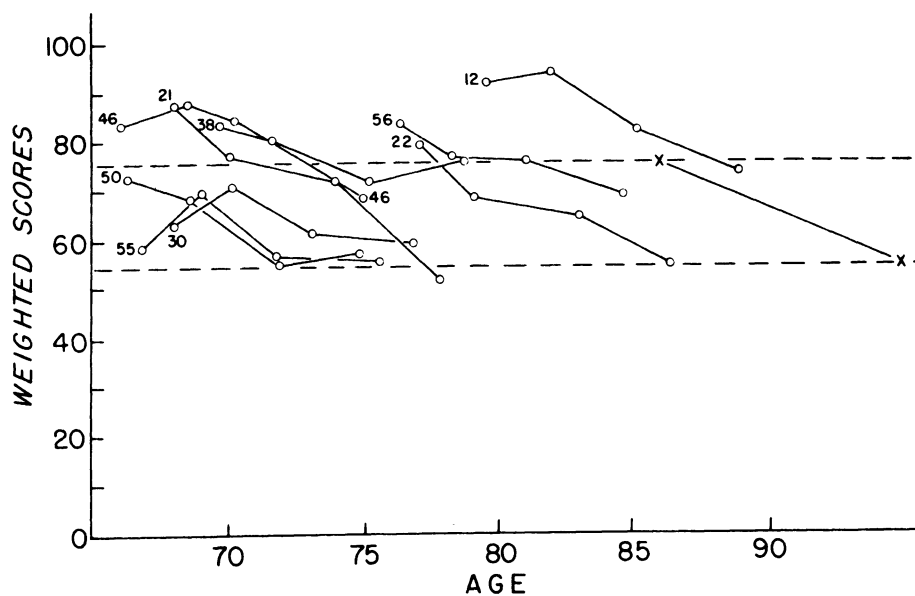


Fig. 2. Successive total weighted Wechsler-Bellevue scores, by age, of nine aged male subjects who were living on January 1, 1961. Upper dashed line represents mean first test score of all 13 subjects who were given four tests; lower line represents mean score of last test. The diagonal connecting these two represents the average rate of decline over the mean interval (8.99 years) between the first and last test.

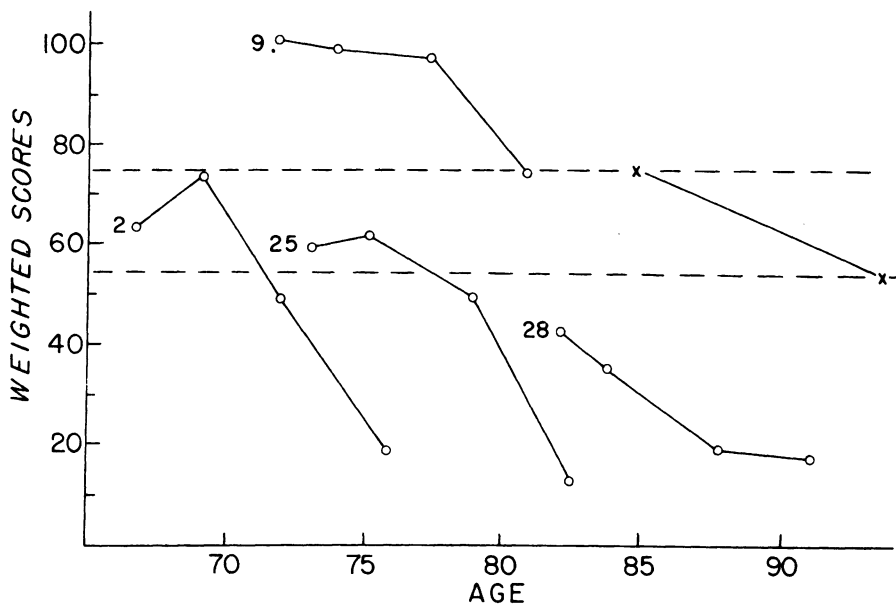


Fig. 3. Successive total weighted Wechsler-Bellevue scores, by age, of four male subjects who died before January 1, 1961. See legend Fig. 2 for details.

Terminal Drop

These curves suggest that factors related to the death of the individual cause a decline in intellectual performance, and that the onset of this decline may be detected in some instances several years prior to the death of the person.

In order to test this hypothesis, an analysis was made of data obtained from the 70 subjects who had been given at least two tests. Of these, 37 were living on January 1, 1961, and 33 had died prior to this date. Fortunately these two groups were of equal age at the time of their last test (deceased, 79.88 years; living, 79.56). Furthermore, there appeared to be no difference between the two groups in initial level of performance, thus any differential rate of decline between the groups could reasonably be attributed to factors related to the death of the one group.

Since the periods of time between successive tests were different for each subject, the mean annual rate of drop in total weighted score, in performance score and in verbal score were used to compare the two groups. Of the three only the performance score showed a significant difference (.001 level), and this was in the expected direction. Thus the mean annual drop in performance score was 1.99 points for the deceased group as opposed to .98 for the survivors. Since some of the survivors were also approaching death, and indeed some died within a few months of the arbitrary date chosen for the analysis, the reported differences between the two groups are conservative estimates of the possible influence of the "death factor" upon performance.

Institutionalization cannot be invoked as a cause of the obtained score difference, because the deceased group experienced its drop in performance over a shorter period of time than did the surviving group (4.58 years vs. 5.69 years; p less than .05).

It therefore appears that we have here strong evidence for the existence of a factor, which might be called terminal drop or decline, which adversely affects intellectual performance and is related to impending death of the aged person.

Other Evidence in Support of the Terminal Drop Hypothesis

Evidence in support of this hypothesis may be deduced from other studies, although they were not designed with this in mind. Most pertinent are the findings of Jarvik, Kallmann, Falek and Klaber (1957) in their study of changing intellectual functions of senescent twins. These authors compare curves of intellectual

"change" during the senium derived from their own longitudinal study with comparable cross-sectional data. While both sets of curves show a progressive decline through the seventh and eighth decades, the rate of decline in the cross-sectional material is appreciably greater. Since the longitudinal data were based upon two tests given to each subject with an inter-test interval of about eight years, it is obvious that at the time of the initial test subjects in this group had a minimum life expectancy of at least eight years. It is unlikely, therefore, that the initial test scores used in the calculation of the longitudinal curves were influenced by terminal drop. There is no such assurance for the subjects who provided the cross-sectional data. Indeed we would anticipate that the more aged these subjects were the greater the probability that some of them would already be closely approaching death and thus more likely to experience terminal decline in test performance. This could explain the increasingly poorer performance of the older age groups of cross-sectional subjects when compared to their age peers in the longitudinal group.

Additional evidence from the senescent twin study parallels our own findings. Original scores of subjects who survived through the taking of the second test were compared with the scores of those who died before the retest was administered. Although the results were statistically insignificant, the obtained mean score of the survivors was higher than that of the deceased group. It will be recalled that on a similar comparison we were able to demonstrate on the Moosehaven sample a greater mean annual rate of decline on the Wechsler-Bellevue total performance test score for the deceased group. We could not, however, show a difference in initial test performance between the two groups.

Again Jarvik and her associates noted that in 10 of twelve comparisons there was an increase in the mean intra-pair test score differences on the 1955 tests over those obtained on the original (1947) tests obtained eight years prior to this time. Although again not significant statistically, the findings suggest that twins may become less alike in intellectual performance as they age in the senium. This is the kind of change which would be anticipated, if one of the twins in a pair began to experience terminal decline in anticipation of death, while the other remained in relatively good health.

Terminal Drop and EEG

The fact that decline in intelligence test score is related to the death of the person suggests the presence of an

organic factor related in some way to the health of the individual. An investigation by Obrist, Henry and Justiss (1961), based on the Moosehaven sample, sheds some light on this question. Their longitudinal study of 114 aged males shows a progressive slowing of the occipital alpha rhythm with increasing age. Furthermore, 28 cases who died shortly after their last EEG experienced a significant mean change of 0.6 cps. over a five year period, in contrast to a drop of only 0.3 cps. experienced over a seven year period by 28 surviving subjects. Thus the terminal drop found in intellectual performance seems to have its counterpart in EEG.

That intelligence test performance and EEG are indeed related was also demonstrated by Obrist and his associates (Obrist, Busse, Eisdorfer and Kleemeier, 1962). These investigators found a small but statistically significant correlation between intelligence test scores and alpha frequency on a sample of 115 Moosehaven subjects whose mean age was 77.0 years. In addition subjects whose tracings were characterized by diffuse slow activity exhibited significantly poorer intelligence test performance than those subjects whose tracings were free of this abnormality.

Although age could not be excluded as a source of at least a part of the common variance of EEG and intelligence, it was felt that health was a more significant contributor. This contention was supported by a failure to find any relationship between EEG and intelligence in aged persons living independently in the community. The fact that 26% of the Moosehaven group could be considered as convalescent because of some acute or chronic illness suggested that health rather than age per se might be responsible for the EEG-intelligence relationship, particularly since only a negligible number of the independently living older sample could be considered convalescent. Additional support for this point of view was provided by the finding that subjects showing some clinical evidence of arteriosclerosis exhibited a tendency both toward lowered intelligence test performance and mean alpha frequency when compared to a matching group without arteriosclerosis. Obrist and his associates suggest that "...variations in cardiovascular pathology more than any other factor, are responsible for the results obtained here, as well as those reported in the literature."

Conclusions

The significance of these studies can be summarized as follows. Great individual differences in intelligence test performance and in the amount of decremental change in this performance are found as

age increases in the senium. These differences may be accounted for to a substantial degree by the presence of terminal drop or decline in performance associated with the death of the subject, but are not directly related to age in the senium. The presence of this death factor tends to associate intellectual decline to adverse change in health or organic integrity of the person rather than to so-called "normal" age change. It suggests further that any decline in performance not accounted for by terminal drop cannot simply be attributed to passage of time or to age alone, but must be explained in more explicit terms.

This reasoning leads to the defensible position that there is no evidence for age change in intelligence in the senium, and that changes which are found are better related to the physical state or health of the organism. Supporting this reasoning is the demonstrated fact of terminal drop both in intellectual performance and in EEG as well as the positive relationship found between EEG and intelligence test scores. The further linkage of both of these indices of decline to the presence of cardiovascular disease gives us all the more reason to believe that it is incorrect to refer to observed decrement in intelligence in the later years of life as the product of normal age change.

On the positive side these results permit the speculation that, in the absence of cardiovascular and other pathological conditions, post-maturational decline in intelligence does not occur. An additional point suggested by the data is that when intellectual decline does occur it is likely to follow an individual pattern, marked by relatively sharp decline, rather than a gradual wasting of ability as would be inferred from age curves of intellectual change based upon cross-sectional data.

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