"maladjusted" to "adjusted" profiles for the criterion group, the Nebraska sample, and the Brigham Young sample were 36:64, 31:69, and 50:50, respectively. Perhaps if greater care had been exercised in selecting comparable cross-validation samples the size of the shrinkage might have been smaller.

The results obtained in this study have a threefold significance. (i) The decision rules should aid a counseling center in detecting emotional maladjustment in an entering freshman class. (ii) The use of the computer in an entirely new area of intelligent problem-solving has been demonstrated. (iii) This study could pave the way for rigorous investigation of clinical decision-making, which is more subtle than personality test interpretation.

BENJAMIN KLEINMUNTZ
Department of Psychology,
Carnegie Institute of Technology,
Pittsburgh, Pennsylvania

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2. Grateful acknowledgment for assistance in computational and computer programming is made to R. Dale Shipp, graduate mathematics student at Carnegie Institute of Technology.
3. The revised set of MMPI rules will be furnished to interested readers upon request.
4. This research was supported in part by the National Institute of Mental Health, grant No. M-5701.

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Antibodies to Genetic Types of Gamma Globulin after Multiple Transfusions

Abstract. *Seventeen of 24 sera from children who had received multiple transfusions contained agglutinating antibodies against a Gm factor absent in the individual's serum gamma globulin. Each of these agglutinators was highly specific for a single Gm factor, and all proved useful as reagents for genetic typing. The accumulated evidence indicates that they resulted from genetically foreign gamma globulin introduced by transfusion.*

Agglutinating antibodies, used for delineation of the various genetic types of human globulin, occur rarely in normal sera and occasionally in sera from patients with rheumatoid arthritis (*1*). Through the use of such antibodies a number of genetic factors in human globulin have been delineated. These factors occur as alleles at two genetic loci (the Gm and Inv loci) (*2*). The implications for the administration of foreign genetic types of gamma globulin as transfusions or as pooled gamma globulin have not been ascertained. As an approach to this question, a search for antibodies against Gm and Inv factors has been carried out in children who have received multiple transfusions for chronic anemia. Blumberg *et al.* (*3*) found precipitating antibodies against beta lipoproteins in a small number of individuals in a similar group. In the study reported here, 17 of 24 children were found to have agglutinating antibodies against one of the Gm factors.

Twenty of the 24 children had thalassemia major, three had congenital nonspherocytic hemolytic anemia, and one had elliptocytosis. All had received at least three transfusions and some had received more than 40; the last transfusion had been given approximately 1 month prior to testing. With techniques previously reported from this laboratory (*4*), Gm-specific antibodies were detected by their ability to agglutinate group O, Rh_o erythrocytes coated with appropriate anti-D sera. The specificity of the agglutinators was determined by inhibition of agglutination with normal sera of known Gm type, and Gm typing of all the sera was carried out with inhibition techniques in which test reagents of known specificity were used. Gm typing of sera containing anti-Gm agglutinators was accomplished by a variety of procedures in which the effects of these agglutinators in the typing system were nullified. Inhibition by 2-mercaptoethanol proved particularly useful.

The data for the patients studied are summarized in Table 1. Antibodies against factors Gm(a), Gm(b), or Gm(x) were found in sera from 15 of the 20 children with thalassemia and in sera from two of the three children with congenital hemolytic anemia. In each case agglutination was inhibited only by individual sera of appropriate Gm type; all such sera proved useful as highly specific typing reagents. No patient had serum containing antibodies against more than one Gm factor, and in each instance the patient's serum lacked the factor for which the agglutinator was specific. Anti-Gm(x) agglutinators were found only in those individuals whose sera contained the Gm(a) factor. The one patient whose serum lacked the Gm(b) factor had a low but definite anti-Gm(b) titer. Anti-Inv agglutinators were carefully sought but were not found, although at least seven of the sera lacked the Inv(a) factor. Precipitating antibodies to specific types of gamma globulin have not been demonstrated in any of these sera.

Table 1. Anti-Gm agglutinators in the 24 individual patients grouped according to Gm phenotype.

Agglutinator	
Type	Titer
a + b + x +	
	0
	0
	0
a + b + x −	
Anti(x)	1:80
Anti(x)	1:40
Anti(x)	1:8
	0
	0
	0
a − b + x −	
Anti(a)	1:1280
Anti(a)	1:320
Anti(a)	1:320
Anti(a)	1:160
Anti(a)	1:160
Anti(a)	1:80
Anti(a)	1:80
Anti(a)	1:80
Anti(a)	1:40
Anti(a)	1:40
Anti(a)	1:16
Anti(a)	1:16
Anti(a)	1:8
	0
a + b − x −	
Anti(b)	1:10

Sera from 27 pediatric patients who had not received transfusions were screened for anti-Gm agglutinators. No such agglutinators were found in this control group. Serum from 24 of these patients lacked at least one of the Gm factors.

The studies reported here revealed that 71 percent of the sera from a group of children who had received multiple transfusions contained anti-Gm agglutinators, and the facts which have been presented strongly support the conclusion that these antibodies resulted from the introduction of foreign gamma globulin through multiple transfusions. This incidence of specific anti-Gm agglutinators contrasts markedly with the low incidence in normal individuals previously reported (*1*). The possibility that normal individuals with sera containing such agglutinators may have received foreign gamma globulin should be carefully reconsidered. The clinical significance, if any, of the presence of these anti-Gm antibodies in patients that have received multiple transfusions remains to be explored (*5*).

JAMES C. ALLEN
HENRY G. KUNKEL
Rockefeller Institute, New York

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Aftereffect of Seen Motion with a Stabilized Retinal Image

Abstract. *Prolonged inspection of uniformly moving contours affects differentially the luminance threshold for the detection of test contours as a function of the direction of motion of the test contours. This finding supports a new explanation of the well-known aftereffect.*

If one views a train of contours moving steadily across the visual field and then turns to a nonmoving scene, the stationary scene appears to be in motion. The direction of the movement aftereffect is opposite to the movement of the contours that was initially seen. This widely known and easily observed phenomenon has never been satisfactorily explained (*1*).

Recently, Hubel and Wiesel (*2*) have reported neural cells in the visual cortex of the cat which respond to stimuli moving in a single direction within the visual field. Such a finding offers a new basis of explanation of the movement aftereffect. It may be that extended viewing of stimuli moving in one direction can produce differential adaptation at the cortex, affecting only cells sensitive to movement in that direction. Such an explanation has been proposed by Sutherland (*3*): "the direction in which something is seen to move might depend upon the ratios of firing in cells sensitive to movement in different directions, and after prolonged movement in one direction a stationary image would produce less firing in the cells which had just been stimulated than normally, hence apparent movement in the opposite direction would be seen to occur." If the perception of motion depends upon the action of these direction-specific cells, it follows that prolonged viewing of a stimulus moving in one direction will elevate the threshold for the subsequent detection of stimulus patterns moving in that direction. We made a study (*4*) to find whether a threshold elevation exists and, if it does, whether it is related to movement aftereffect. According to the theory, magnitude of the movement aftereffect and magnitude of threshold elevation should be covariant.

To obtain maximal differential adaption to motion it is necessary to present a subject with a stimulus having truly unidirectional motion. This is ordinarily impossible, since involuntary movements of the eye, present even during "steady" fixation, superimpose a random spectrum of eye motions upon the motion of the physical stimulus (*5*). We eliminated the effect of involuntary eye movements and rendered our stimulus unidirectional by presenting the targets as stabilized retinal images. This technique optically "locks" a stimulus onto one retinal area (*6*).

The subject focused upon a black point on a luminous circular field subtending a visual angle of 4°30′ (luminance 1.19 millilambert). The stabilized target, a rectangle with sides subtending visual angles of 2°14′ and 1°35′, respectively, was centered within this field. It comprised bright vertical stripes, 6 minutes of arc wide, separated by areas of background field 32 minarcs wide. These stripes could be made to move either to the right or to the left within the rectangle. The stimulus was presented in a 15-second repeating cycle with three phases. (i) Inspection: for 5.0 second the subject viewed the moving stripes (stripe luminance, 1.48 mlam). (ii) Interval: for 2.8 seconds the subject viewed the circular background field; no stripes were presented. (iii) Test: for 7.2 seconds the subject "tracked" his threshold for stripe detection. "Tracking" is a psychophysical procedure in which the subject diminishes the intensity of the stimulus when he detects the target and increases the intensity when he does not detect it (*7*). During the test phase the subject kept the intensity of the stimulus just above or just below his luminance threshold for stripe detection. Key presses on either of two keys moved a photometric wedge interposed in the optical path of the stimulus so as to modulate intensity either up or down. Recording equipment provided a record of the intensity of the stimulus as a function of time. Catch trials, in which no stripe target was presented, were interspersed during the test phase.

Luminance thresholds for the detection of moving stripes were measured under two conditions. In the "reverse" (*R*) condition, the direction of motion

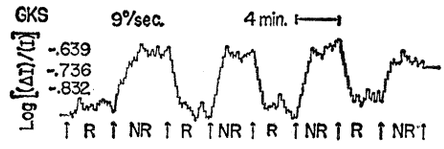

Fig. 1. A continuous record of luminance threshold values during one session. Changes of condition, between *R* and *NR*, are shown at the bottom of the record.

during the inspection phase was opposite to that during the test phase; in the "nonreverse" (*NR*) condition, the direction of motion during the inspection and test phases was the same. According to the prediction, the luminance threshold would be higher in the *NR* than in the *R* condition. Threshold elevation was determined in three subjects for various velocities of the stripes. The velocity ranged from 10′50″ visual angle per second, where motion is just perceptible, to 15° per second, where the observer reports a blur in which the individual stripes are not discernible. A typical session required 32 minutes of observations. Blocks of tests under conditions *R* and *NR* were presented alternately in a balanced design. The same velocities were used for the inspection and the test phases.

For velocities between 4° and 9° visual angle per second, the luminance

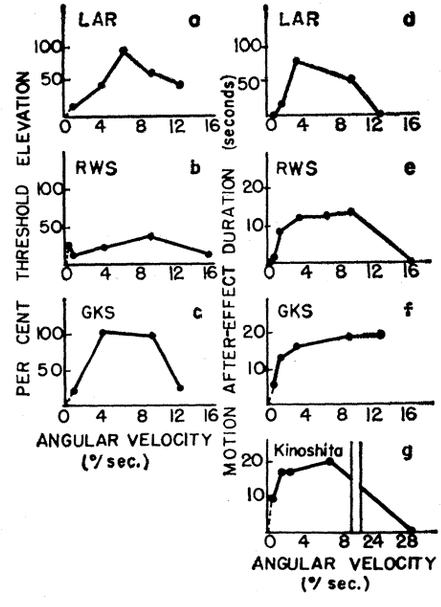

Fig. 2. (*a–c*) Threshold elevation, 100 $[(\overline{NR}-\overline{R})/\overline{R}]$, as a function of stimulus velocity. Each point is based on the mean of 170 to 300 measurements. (*d–f*) Duration of motion aftereffect as a function of stimulus velocity for the three subjects of *a–c*. (*g*) Comparable data of Kinoshita on the duration of motion aftereffect, for one subject.