be due to a difference in the rates of elution of Cr^{51} from the cells.

Since the labeling of erythrocytes with Cr^{51} involves attachment of the metal to hemoglobin (*4*), and since the hemoglobins of normal adult red cells (mainly hemoglobin A, *5*) differ markedly from the predominating hemoglobin (hemoglobin F) in the red cells of the newborn (*6*), it was decided to determine whether a difference exists in the ability of the respective hemoglobin to retain the Cr^{51} label.

Erythrocytes, obtained from the blood of normal adults and from the umbilical cord of normal full-term infants, were washed with saline to remove plasma proteins, then resuspended in suitable volumes of saline to bring the hemoglobin concentrations to the same level (about 12 g percent). Equal volumes (5 to 15 ml) of the two types of washed cell suspensions were incubated with equal amounts of radioactive Cr^{51} (280 to 980 μc as $Na_2Cr^{51}O_4$) for about 2 hours at 37°C. The labeled cells were repeatedly centrifuged and washed, with one or two prolonged periods (3 to 24 hours) of contact between cells and saline, till the supernatants exhibited a very low degree of radioactivity, as measured in a well-type scintillation counter. After removal, by this means, of essentially all unbound Cr^{51} from the cells, the latter were hemolyzed by the addition of 1 vol of water and 0.4 vol of toluene. After centrifugation to remove stroma, the concentration and radioactivity of the clear hemoglobin solutions were determined. Suitable aliquots containing approximately equal amounts of the Cr^{51} label were subsequently dialyzed over periods of time ranging from 65 to 213 hours against 12 or more changes of saline, each of 100 ml. The radioactivity of the dispersion medium was finally measured, and appropriate correction was made for decay of the isotope.

Under strictly comparable experimental conditions, an unequivocal difference was evident in the rate at which Cr^{51} was eluted from the two types of hemoglobin solutions. In a typical experiment (Fig. 1), the percentage of the isotope eluted from cord blood hemoglobins after 87 hours of dialysis was 2.3 times that eluted from adult hemoglobin, whereas the amount of Cr^{51} bound per unit concentration of hemoglobin was approximately the same. In these experiments, the ratio of percentage of Cr^{51} eluted from cord hemoglobins to percentage of Cr^{51} eluted from adult hemoglobins ranged from 2.0 to 2.9. Variations in the absolute percentages of chromium eluted (ranging from 17 to 27 percent of the total for cord and from 5.9 to 13.5 percent for adult) did not affect the net results. Most of the elution, moreover, occurred during the first 24 to 37 hours of dialysis, confirming the work of Gray and Sterling (*4*).

Whether or not the hemoglobins show the same differentiation in the intact red cell is still uncertain, but the results reported here lend indirect support to Mollison's suggestion that much of the difference in survival rates of red cells of the adult and of the newborn, as determined with Cr^{51}, is only apparent. It is probable that molecular differences in these hemoglobins are responsible. This aspect and its implications for thalassemia (Hb F) and other hemoglobin diseases are receiving further attention (*7*).

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7. We wish to express our thanks to the National Cancer Institute of Canada for funds to purchase Cr^{51} (Abbott sodium radio-chromate) and to H. Blondal and G. E. Delory for help and advice.

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Reversal of Virus-Caused Stunting in Plants by Gibberellic Acid

Reports on the use of gibberellic acid in overcoming genetic (*1*) and physiologic (*2*) dwarfism suggested testing whether it could induce growth of plants stunted by certain virus diseases. Three leafhopper-borne viruses that cause severe stunting in susceptible plants were chosen for the experiment. Corn stunt virus of the Mesa Central strain (*3*) was inoculated into seedlings of hybrid sweet corn, *Zea mays,* of the variety Country Gentleman, by means of viruliferous *Dalbulus maidis*. The Eastern strain of aster yellows virus was introduced into seedlings of China asters, *Callistephus chinensis,* by the leafhopper *Macrosteles fascifrons*. Wound tumor virus was transferred to seedlings of crimson clover, *Trifolium incarnatum,* by *Agalliopsis novella*. Six weeks after inoculation, severely stunted plants were grouped according to size and sprayed with a freshly prepared water solution of gibberellic acid at 100 ppm (*4*). A control group of similar sized plants was sprayed with distilled water. Both groups were maintained on the same greenhouse bench.

Gibberellic acid was reapplied twice, at weekly intervals. The striking effect of the treatment is shown in Figs. 1–3. The photographs were taken 1 week after the last application. The treated plants had resumed growth, while diseased controls had remained stunted. The internodes of yellowed asters and stunted corn elongated to twice the original length, while the clover petioles increased 3 times in length and assumed an erect position during the 4-week period. In corn, the reversal of stunting became visible 48 hours after the first application, while in asters and clover it was noticed only after 5 days.

It is apparent that, under the conditions of the test, gibberellic acid influenced significantly the growth of virus-stunted plants. Although stunting could be overcome to a considerable degree by three applications of gibberellic acid, diseased plants retained other signs of virus infection. Leafhopper vectors were

Fig. 1. China asters infected with aster yellows: (left) untreated control; (right) treated with gibberellic acid.

Fig. 2. Hybrid sweet corn infected with corn stunt: (left) untreated control; (right) treated.

Fig. 3. Crimson clover with wound tumor: (left) untreated control; (right) treated. [Photos for Figs. 1–3 by J. A. Carlile]

able to recover the respective viruses from plants treated with gibberellic acid as readily as from untreated controls. Further studies will be directed to explain the mechanism of action of gibberellic acid in the reversal of virus-caused stunting.

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Weber's Law and the Difference Threshold for the Velocity of a Seen Object

Although a fairly extensive literature exists concerning the visual perception of stimulus movement (*1*), only one experiment has been reported (*2*) which deals directly with the measurement of difference thresholds for the velocity of a seen object. According to that report: "An approximate correspondence with Weber's Law was found, the divergence from it appearing, in general, as an increase of the threshold at both ends of the range of initial velocities. The Mean Threshold (0.5 probability of perception, corrected for guessing) was, in favourable conditions, about 12 per cent of the initial velocity. Whether the stimulus was an increase or a decrease of velocity made no marked difference."

It is found, however, that when Hick's data are plotted (Fig. 1, broken curve), the resulting function may be interpreted as passing through an optimal $\Delta V/V$ value, rather than being a generally straight line of zero slope, which approximate correspondence with Weber's law would require. Because of this interesting alternative interpretation, a partial replication of Hick's experiment was undertaken.

Hick obtained thresholds for instantaneous increments and decrements in velocity for a pip horizontally deflected across the face of a cathode-ray tube. The total excursion of the pip seems to have been about 3.5 in., the velocity increment (or decrement) being introduced at the mid-point. For the replication, the total excursion of the pip (in inches) and the initial velocities of the pip (in inches per second) closely matched Hick's values (*2*). However, since it proved convenient to use a scope hood giving a viewing distance of 10 in., the viewing distance was approximately one-half of the 21-in. distance used by Hick. The chief consequences of this difference in viewing distance were the yield of a range of initial velocities higher than those of Hick when velocity is measured in terms of visual angle per second and the doubling, approximately, of the total angular excursion of the pip. Since Hick found no marked difference between incremental and decremental thresholds, it was decided to restrict this replication to incremental velocities only.

The remaining curves of Fig. 1 compare the incremental data obtained in the present experiment with those of Hick. In the new data, each point is based on 300 values—30 judgments for each of ten subjects; similar information is not reported in the earlier work. The thresholds in the present study were determined by average z-score computations (*3*); other methods were tried and yielded similar over-all functions.

In general, the findings of the two experiments agree fairly well, showing that as initial velocity increases, $\Delta V/V$ at first decreases and then increases. Of interest is the fact that when the upper limit of initial velocities is extended, as in the present experiment, it becomes evident that if Weber's law holds at all, it does so for a very small portion of the usable range only. It may, indeed, be safe to conclude that the Weber fraction passes through a minimum in the 1-to-3-degrees-per-second region of the range of initial velocities.

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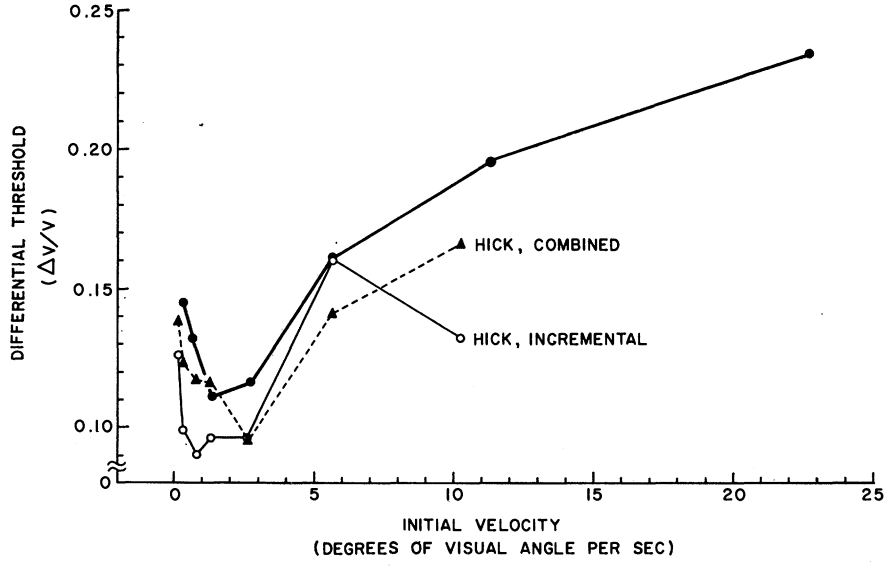

Fig. 1. Difference thresholds for the velocity of a seen object as a function of initial velocity. The uppermost curve is for data gathered in the study here reported; the other curves were plotted from tables presented by Hick (*2*).

Evidence for an Accessory Pathway of Galactose Metabolism in Mammalian Liver

Recent studies on the disease galactosemia have revealed it to be the result of the congenital deficiency of a specific enzyme important in the conversion of ingested galactose to glucose derivatives (*1*). Normally, ingested galactose is first converted by means of adenosine triphosphate and galactokinase to α-galactose-1-phosphate (Gal-1-P), which can then be transformed to α-glucose-1-phosphate (G-1-P) through a series of reactions involving uridine diphosphate glucose (UDPG) and uridine diphosphate galactose (UDPGal). Thus (*2*):

$$\text{Gal*-1-P} + \text{UDPG} \underset{\text{P-Gal transferase}}{\rightleftharpoons} \text{UDPGal*} + \text{G-1-P} \quad (1)$$

$$\text{UDPGal*} \underset{\text{UDPGal-4-epimerase}}{\rightleftharpoons} \text{UDPG*} \quad (2)$$

$$\text{UDPG*} + \text{PP} \underset{\text{UDPG pyrophosphorylase}}{\rightleftharpoons} \text{UTP} + \text{G*-1-P} \quad (3)$$

The asterisk traces the galactose moiety through its conversion to glucose-1-phosphate. In galactosemia, the enzyme catalyzing reaction 1 (P-Gal transferase) is