it may be assumed that either the radiocalcium was removed from a homogeneous pool or that its deposition and removal occurred in the same area. Induced excess excretion of Ca^{45} and of other metals was also found after the administration of Ca-Na-EDTA. These experiments will be reported in detail elsewhere (*19, 20*).

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Agglutinin Linkage and Antibody Globulins

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Linkage of anti-A and anti-B isoagglutinins in some O sera was demonstrated by Landsteiner and Witt (*1*). Similar linkage can be demonstrated in some cases between "nonspecific cold agglutinins" which occur normally in most sera and cold agglutinins acting specifically at higher temperatures. A typical example out of nine such sera is given below.

An A_2B serum containing the "extra" agglutinin alpha$_1$ (anti-A_1) was repeatedly absorbed in the cold with O cells. Considerable loss of the anti-A_1 resulted.

Titer before absorption:	256
Titer after absorption:	16

Thirty sera containing isoagglutinins were similarly absorbed in the cold with O cells. There was no loss of isoagglutinin titer in any of these sera.

Linkage may therefore exist between isoagglutinins and isoagglutinins, and between cold agglutinins and cold agglutinins, but not between isoagglutinins and cold agglutinins. *It appears that linkage can only take place between antibodies in the same globulin fraction.*

This would be supported by the biochemical findings of Cohn (*2*) who showed that isoagglutinins do not belong to the γ-globulin fraction, to which cold agglutinins are believed to belong (*3*). The absorption experiments of Crawford and Mollison (*4*) also indicate that isoagglutinins and cold agglutinins belong to different globulin fractions. By absorption of anti-globulin sera with sensitized red cells from cases of hemolytic anemia, or with cells that have been exposed to incomplete anti-Rh, or to normal cold antibodies, they were able to prepare sera which would no longer agglutinate the type of cells used for absorption but could agglutinate one or more of the other types.

The work of Crawford and Mollison also suggests that the auto-antibodies of hemolytic anemias differ from the normal cold auto-antibodies. This would be in keeping with the different mode of origin of the two types, which are thought to arise from auto-immunization and heterogenetic bacterial stimuli, respectively. It would be difficult to confirm this by agglutinin-linkage studies, because the various red cells used for absorption would act in a similar manner upon both these types of antibodies.

Wiener (*5*) has emphasized that there is a slender line of demarcation between isoagglutinins and cold agglutinins. This is undoubtedly so, but there does seem to be a difference in their behavior when absorbed in the cold with O cells.

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Motor Nerve Filament Block Produced by Botulinum Toxin[1]

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Burgen, Dickens, and Zatman (*1*) have shown that during the neuromuscular paralysis produced by botulinum toxin (type A) excitation of a motor nerve releases no acetylcholine (ACh) from its muscular terminals. This finding could be explained by assuming either that the toxin blocks motor nerve terminals just proximal to the site of ACh-release, or that it interferes with the process of release itself. Experiments were carried out to decide between the two alternatives, using the cat's gracilis muscle *in situ* (*2*) and the guinea pig's excised serratus muscle

[1] This work will be reported in full at a later date. The project was supported by a grant from the Defence Research Board of Canada.