crease and chloroform poisoning with a less marked increase in the residual reduction, the changes being accompanied by a rise in the non-protein nitrogen, to which they are undoubtedly due in large measure. After the injection of one gram of glucose, while there was the usual marked temporary hyperglycemia, there was no change in the unfermented residuum. Subsequent to the administration of xylose the reducing power of the blood after fermentation returned to its previous value in four hours in the normal rabbit, two hours in phlorhizin diabetes, four hours in chloroform poisoning, but was scarcely back to the control level in eight hours in tartrate nephritis.

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Evidence for the supernormal phase and a recovery curve of conduction in the human heart.

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Recent work by Ashman[1] and by Ashman and Woolley[2] has demonstrated that the compressed or injured cardiac muscle of the turtle may, after excitation, recover its conductivity along a curve which passes through a supernormal phase. Thus an impulse traversing the compressed muscle will travel most rapidly during the supernormal period. If the degree of compression is sufficient only impulses arriving at the compressed region during the supernormal phase are transmitted; earlier or later ones are blocked. These experimental observations add much weight to the argument for a supernormal phase in the recovery of conductivity in the human hearts discussed below.

Lewis and Master[3] reported two cases of block in the human heart which they interpreted as evidencing a supernormal phase. In their first case typical complete heart block was interrupted at

[1] Ashman, Richard, *Am. J. Physiol.*, 1925, lxxiv, 140.

[2] Ashman, R., and Woolley, E., PROC. SOC. EXP. BIOL. AND MED., 1925, xxiii, 159.

[3] Lewis, T., and Master, A. M., *Heart*, 1924-25, xi, 371.

times by a ventricular systole in response to an auricular impulse and only when the latter arose during late systole or early diastole. Auricular impulses arising later in diastole were blocked.

We have obtained electrocardiograms of two clinical cases of heart block which show phenomena attributable to a supernormal phase. Our first case is similar to Lewis and Master's excepting that, (a) a series of auricular impulses are transmitted if the auricles are rapid; (b) ventricular standstill supervenes when, as a result of auricular slowing, conduction fails, and (c) the period of ventricular quiescence is interrupted at irregular intervals by idioventricular contractions which do or do not permit conduction depending upon their time relations with the auricular systoles. As in Lewis and Master's case, conduction time increases slightly as the interval between the R-wave and the subsequent P-wave widens.

In our second case most of the P-waves which fall between about 0.06 sec. before the R and 0.36 sec. after the R-wave are transmitted. Earlier and later impulses are blocked. There is some overlapping of blocked and transmitted impulses at either end of this range.

The conduction times (P-Q) vary from 0.60 to 1.01 second. Their average duration exceeds the average length of the auricular cycles. Unlike Lewis and Master's first case and our first case, the conduction times become progressively shorter with longer ventricular rest until block suddenly supervenes. These facts may be explained on the assumption that only a very short stretch of the conducting tissue recovers through a supernormal phase, and that the greatly prolonged conduction times are due to time occupied by the impulse in passing other damaged tissue.

When corrections are made for variations in the auricular rate and for the duration of the rest period on the ventricular side of the block,[4] a curve of recovery of conductivity is obtained of a formed similar to those determined in animal experiments.[5, 6] The curve demonstrates that in the depressed conducting tissue of this heart conductivity had not fully recovered at the end of more than a second.

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[4] Herrmann, G. R., and Ashman, R., *Am. Heart J.*, 1926, i, 269.

[5] Ashman, Richard, *Am. J. Physiol.*, 1925, lxxiv, 121.

[6] Lewis, T., and Master, A. M., *Heart*, 1925, xii, 209.