TABLE 1

| Name | Diagnosis | | Drug therapy | Outcome | Total protein (grams per cent.) | Electrophoretic analysis of serum: Areas A | $a_1 + a_2$ | $\beta$ | $\gamma$ | $\frac{\gamma}{\text{Total}}$ per cent. | $\frac{\gamma}{A}$ | T.P. (gm/per cent.) $\times \frac{\gamma}{\text{Total}}$ | $\frac{a}{A}$ |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| | | *Class* | | | | | | | | | | | |
| F.Q. | Acute R.F. R.H.D.–M.S.–M.I. | I–E | None* | Rec. | ... | 113 | 17 | 28 | 38 | 19.4 | 0.34 | ... | .15 |
| F.Q. | R.H.D.–M.S.–M.I. | I–A | None | Rec. | 7.50 | 123 | 17 | 26 | 31 | 15.7 | 0.25 | 1.17 | .14 |
| J.C. | Acute R.F. R.H.D.–M.I. | I–E | None* | Rec. | 7.62 | 76 | 30 | 28 | 53 | 28.4 | 0.70 | 2.16 | .39 |
| J.C. | R.H.D.–M.I. | I–A | None | Rec. | 7.37 | 113 | 14 | 20 | 33 | 18.3 | 0.29 | 1.35 | .12 |
| G.S. | Acute R.F. R.H.D.–M.I. | II–E | None* | Rec. | 8.50 | 119 | 18 | 28 | 54 | 24.8 | 0.45 | 2.12 | .15 |
| G.S. | R.H.D.–M.I. | II–B | None | Rec. | 7.25 | 109 | 10 | 21 | 42 | 23.0 | 0.39 | 1.67 | .09 |
| R.S. | Acute R.F. R.H.D.–M.S.–M.I. | II–E | None* | ? | ... | 74 | 20 | 33 | 52 | 29.0 | 0.70 | ... | .27 |
| R.S. | Acute R.F. R.H.D.–M.S.–M.I. | II–E | Salicylates | ? | 7.25 | 76 | 31.5† | | 17.5 | 14.0 | 0.23 | 1.02 | .. |
| A.L. | Acute R.F. R.H.D.–M.I. | I–E | None | Rec. | 7.75 | 75 | 24 | 25 | 51 | 29.3 | 0.68 | 2.27 | .32 |
| J.M. | Acute R.F. R.H.D.–M.I. | II–E | Small doses salicylates | Rec. | 8.12 | 98 | 29 | 30 | 45 | 22.2 | 0.46 | 1.80 | .30 |
| S.C. | Acute R.F. R.H.D.–M.S.–M.I. | IV–E | Massive doses of salicylates and treatment for congestive failure | Died 2 days after sample was taken | 7.25 | 61 | 41 | 24 | 44 | 25.9 | 0.72 | 1.88 | .67 |
| V.V. | Acute R.F. (Sed rate) R.H.D.–M.S.–M.I. | II–E | None | Rec. | 7.00 | 118 | 13 | 24 | 33 | 17.6 | 0.28 | 1.23 | .11 |
| D.R. | Control | | .... | .... | 7.75 | 131 | 17 | 38 | 26 | 12.0 | 0.20 | 0.93 | .13 |
| D.R. | Control | | .... | .... | 7.50 | 120 | 46† | | 23 | 12.2 | 0.19 | 0.92 | .. |
| K.J.T. | Control | | .... | .... | 7.37 | 78 | 7 | 28 | 14 | 11.0 | 0.18 | 0.81 | .09 |
| B.D. | Control | | .... | .... | 7.37 | 68 | 41† | | 25 | 18.6 | 0.37 | 1.37 | .. |

* Salicylate therapy began after taking of specimens.
† $a_1 + a_2 + \beta$.

from rheumatic fever analyzed in the same electrophoretic cell reveal ratios similar to those reported for normals in the literature in three, and the serum in one (line 16, table 1), was found to be abnormal. The alpha globulin-albumin ratios reported as being increased in cases of febrile disease[7] are less consistently elevated in the specimens analyzed in this study.

It is appreciated that the reported results are only suggestive and much further work along these lines remains to be done. The reported changes may be just another non-specific manifestation of rheumatic fever. However, this method of study offers a quantitative approach not heretofore available in the study of the mechanism of this obscure disease, since it is possible to study in quantitative fashion a constituent of the blood which is found in increased amounts in rheumatic fever.

### Summary and Conclusions

Electrophoretic analysis of specimens of the blood serum of a small number of cases of rheumatic fever during the "active" and "inactive" stages of the disease show consistent increases above "normal" in the gamma globulin fraction of the serum.

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## URONIC ACIDS IN ANIMAL BODIES

It is known generally that quantitative methods for estimating glucuronic acid in biological materials are not specific but may include other uronic acids. In spite of this, much of the literature on the subject ignores the possibility that galacturonic acid and its salts may also be present in animal tissues and fluids. There is perhaps justification, in the case of the carnivore, for considering the uronic acid as glucuronic. This assumption is not justified for the herbivore where it is known that the diet is rich in polygalacturonic compounds in the form of pectic substances. (Pectin "consists chiefly of partially

methoxylated polygalacturonic acids," as mentioned in the National Formulary VII, p. 316.)

This point is confirmed and made more clear by observations made in this laboratory in connection with studies on the transfusion treatment of shock with pectin sols[1, 2, 3] (autoclaved and clarified for control of molecular size and sterility). Our previously published method[4] has made it possible to obtain the data on the uronic acid contents of livers, as given in table 1.

TABLE 1

| Source of samples | Mg uronic acid per gram of liver: Individual specimens | Mg uronic acid per gram of liver: Average | Remarks |
|---|---|---|---|
| Suckling rabbits (composite sample from 4 animals) | ... | 1.5 | Note increase in uronic acid content when diet changes to include plant material known to contain galacturonic acid. |
| Adult rabbits (on commercial rabbit ration) | 17.3<br>38.5<br>28.3<br>19.0<br>27.2 | | |
| | | 26.1 | (Herbivorous) |
| Human, normal adults | 5.6<br>7.3<br>4.4 | | |
| | | 5.8 | (Omnivorous) |

The uronic acid in infant rabbit livers is no doubt glucuronic, but the tremendous increase following the use of a diet containing grains, alfalfa and other plant material must certainly result from galacturonic rather than glucuronic.

The logical conclusions from this would be that in the omnivorous human the uronic acids in tissues and fluids would also include galacturonic. Thus, intravenously injected pectin (a polygalacturonide) would not be a source of substances as foreign to the human body as would some of the other plasma substitutes which have been given consideration. This is in harmony with reports that a considerable proportion of injected acacia remains in the liver,[5] a situation not found with pectin,[1] a substance of easily hydrolyzable galacturonide nature. Spleen enlargement and alteration may take place in some cases immediately after pectin injection,[6, 7] but there is later a return to normal.[8]

The significance of the distribution of the individual uronic acids in tissues of animals having different basic feeding habits offers an interesting field of study. A consideration of this general subject would lead to a better understanding of human metabolism.

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8 Work now in progress in our laboratory.

## PRODUCTION OF GLYCOSURIA IN NORMAL RATS BY MEANS OF ADRENOCORTICOTROPHIC HORMONE[1]

THE production of glycosuria and hyperglycemia in normal rats by the administration of large amounts of 17-hydroxycorticosterone and 17-hydroxy-11-dehydrocorticosterone has been reported.[2, 3] We have been able to duplicate these effects with pure adrenocorticotrophic hormone.

Normal male rats of the Sprague-Dawley strain having an initial weight of 300 grams were force-fed a fluid diet which represented approximately 15 grams of available carbohydrate per day. The adrenocorticotrophic hormone was prepared by the procedure described by Li, Evans and Simpson.[4] Following a control period of ten days the hormone was injected subcutaneously in amounts of 1 mg every two hours until a total of 7 mg per day had been administered. Five rats were injected with the hormone for ten days. As shown in Fig. 1, four of the five animals developed glycosuria on the second day following the beginning of injection and it was continued as long as the hormone was given. One of the rats did not develop glycosuria, although it showed a marked hyperglycemia following feeding. Two of the rats were killed at the end of the injection period. Weights for pairs of adrenal glands of 146.5 and 131.1 mg were recorded. Control weights averaged 36 mg with a

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