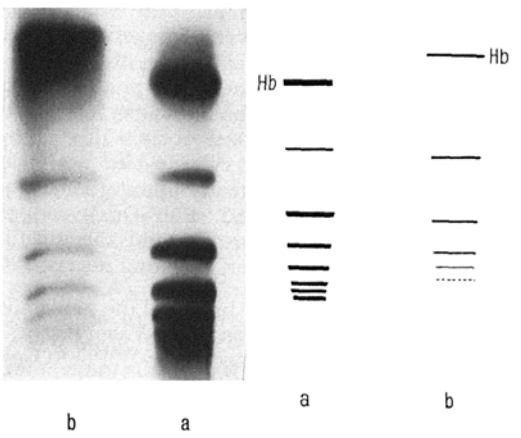
Haptoglobinuria Following Muscular Exercise

Muscular exercise is accompanied by an increase in urinary protein excretion. Though albumin represents the major fraction, we have been able to reveal about fifteen proteins of plasma origin in urine obtained after physical effort[1]. Among these, our attention has been drawn to the haptoglobin, the presence of which has been suggested by PATTE et al.[2] and then established by BERGGÅRD[3] in normal urine. Only the monomeric fraction of haptoglobin has been found in normal urine[4]. In orthostatic, lordotic[5] and pathologic proteinuria[6] the haptoglobin 1-1 form or the more anodic fractions of type 2-1 is found only. As we can consider exercise proteinuria as an intermediate form between normal and pathology, we have investigated the haptoglobin type present in urine after an intense physical effort (9 km cross-country run).

The urine is concentrated within a Visking membrane $^{8}/_{32}$ in. under reduced pressure (see [1]). Not being able to obtain large individual volumes, we have had to combine different samples, and thus it is on a urinary pool (10 g% protein) that the analyses were performed. After addition of haemoglobin and starch gel electrophoresis with the discontinuous buffer of POULIK[7], the haptoglobin-haemoglobin complex is coloured by benzidine. Haptoglobin 2-1 serves for comparison.

The Figure shows the pattern after staining. Urine possesses haptoglobin type 2-1, although in an incomplete form. The monomeric band is the most coloured and therefore quantitatively the most important fraction. Polymers are also present, at least for the anodic first four bands. The last polymers (2 bands), with higher molecular weight, stay absent from urine.



Electrophoretic pattern of haptoglobin 2-1 in urine after physical exercise. a: Serum haptoglobin 2-1, as control. b: Urine.

From this investigation it appears that haptoglobin type 2-1 is present in urine after exercise in a form approaching the complete structure: 5 bands on 7 are visible. It thus seems that the glomerular passage of this glycoprotein is facilitated in exercise proteinuria compared to physiologic urine[4] and nephrotic syndrome[6]. On the contrary, this haptoglobin 2-1 excretion may be compared to orthostatic and lordotic proteinuria[5]. On the other hand, cerebrospinal fluid also contains the lighter polymers of haptoglobins 2-1 and 2-2[8].

The results show that the permeability of the meninges and renal glomerulus plays an important part during the transfer of proteins. The increased glomerular permeability consecutive to muscular exercise permits the heavier haptoglobin 2-1 polymers to pass into the urine. The average molecular weight of haptoglobin 2-1 being about 220,000[9], it appears that the glomerular permeability limit during physical effort is situated around 350,000 molecular weight. The presence of fibrinogen in urine, in small quantity, corroborates this assertion (unpublished results)[10].

Résumé. L'urine recueillie après effort physique intense contient l'haptoglobine de type 2-1 sous une forme proche de sa structure complète. Les cinq bandes les plus anodiques sont visibles dans l'urine d'effort, quoiqu'à un taux inférieur à celui d'un sérum normal.

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Effects of Anoxia on Excitability, Refractoriness, and Contractility in Isolated Rabbit Atria

Introduction. The effects of anoxia on isolated rat atria[1] Purkinje and papillary fibers[2,3] have been shown to include decrease in amplitude and duration of the action potential, prolongation of conduction time and decreased amplitude of contraction. BURN and HUKOVIC[4] have reported that anoxia facilitates the development of fibrillation and hence it is said to increase 'excitability'.

The following experiments were designed to study directly the effects of anoxia on diastolic excitability, refractoriness and contractility in isolated rabbit atria. The results show that changes in neither excitability nor refractoriness can account for increased susceptibility to fibrillation.

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