zuzuschreiben ist. Es wurden deshalb Untersuchungen durchgeführt, in welchen an Stelle des Extraktes ein synthetisches Oxytocin-Präparat (Syntocinon) zur Verwendung gelangte, das von anderen Prinzipien der NH frei ist.

In drei Versuchsserien wurden insgesamt 128 ausgewachsene Ratten von gleichem Gewicht verwendet, die eine salzarme Nahrung erhielten. In einer Versuchsreihe bekamen die Tiere Leitungswasser, in den beiden anderen eine 2,5promillige Kochsalzlösung zu trinken. In jeder Versuchsserie wurden die Tiere in 4 Gruppen von je 32 eingeteilt. Am Versuchstage erhielt die erste Gruppe subkutan eine ACA-Injektion, die zweite Gruppe die gleiche Dosis ACA und gleichzeitig 100 mE Syntocinon, die dritte Gruppe 100 mE Syntocinon und die vierte Gruppe (die Kontrolle) das Lösungsmittel von ACA. Die Urinmenge der Tiere wurde von 4 h vor bis 6 h nach der Injektion alle 2 h gemessen und in jeder Urinprobe Natrium und Kalium mittels Flammenphotometer bestimmt. Die ACA-Dosen pro Ratte betrugen 3,2 mg, 6,4 mg und 40 mg. Jede Dosis des ACA und des ACA in Verbindung mit Syntocinon wurde an 32 Tieren geprüft. Zwischen zwei Experimenten wurden Intervalle von 3 bis 4 Ruhetagen eingeschaltet.

Die Wirkungen des ACA und des Syntocinon (einzeln und zusammen verabreicht) auf die Diurese und die Na- und K-Ausscheidung können den Abbildungen 1 und 2, die einige Experimente wiedergeben, entnommen werden. Die stärkste Wasser- und Elektrolytausscheidung wurde immer bei denjenigen Tieren registriert, die ACA zusammen mit Syntocinon erhielten. Genau wie bei der Verwendung der NH-Extrakte summiert sich die Wirkung des Syntocinon und die von ACA, und zwar sowohl bei den Tieren, die Wasser zu trinken bekamen, als auch bei denjenigen, welchen die NaCl-Lösung verabreicht wurde. Die Verstärkung der ACA-bedingten Wasser- und Natriumausscheidung durch 100 mE Syntocinon ist praktisch die gleiche bei Verwendung von 3,2 mg, 6,4 mg oder 40 mg ACA pro Ratte. Die Einwirkung auf die K-Diurese war im allgemeinen weniger regelmässig als die auf die Na-Diurese. Die steigernde Wirkung von Syntocinon auf die Ausscheidung von Wasser und der Elektrolyten Na und K (Berde und Cerletti[2]; Rosas *et al.*[3]) wurde in diesen Untersuchungen auch bestätigt.

H. Croxatto und
E. Labarca

Physiologisches Laboratorium des Pädagogischen Institutes der Chilenischen Staatsuniversität, Santiago de Chile, 4. Juni 1958.

Summary

Changes in water, sodium, and potassium excretion following administration of synthetic oxytocin (Syntocinon)—alone or in combination with acetazolamide— resemble the changes that occur after an equivalent dose of neurohypophysial extract containing oxytocin. Since the action of Syntocinon on water and sodium excretion is manifest when carbonic anhydrase in kidney tissue is fully inhibited by acetazolamide, it may be assumed that the mechanism whereby Syntocinon increases sodium excretion is independent of carbonic anhydrase. Oxytocin does not affect ultrafiltration in the glomeruli (Krause[4]) and would therefore appear to modify the reabsorption or excretion process in the tubuli.

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Paper Electrophoresis of Soluble Proteins from Regenerating Rat Liver[1]

Previous researches showed that the soluble proteins of the regenerating rat liver exhibit the same electrophoretic pattern as the normal ones, thus ruling out the presence of a preferential reserve protein pool among the soluble liver proteins (Sorof, Claus, and Cohen[2]). However, these electrophoretic analyses did not take into account the behaviour of the other complex protein groups, like the carbohydrates bound ones, which appear to play an important role during any tissue proliferation (Gersh and Catchpole[3], Catchpole[4], Eugel[5]).

The present study, carried out by a suitable method of preparation and analysis of the liver proteins, deals with the soluble proteins and glycoproteins changes occurring during the liver growth after the partial hepatectomy.

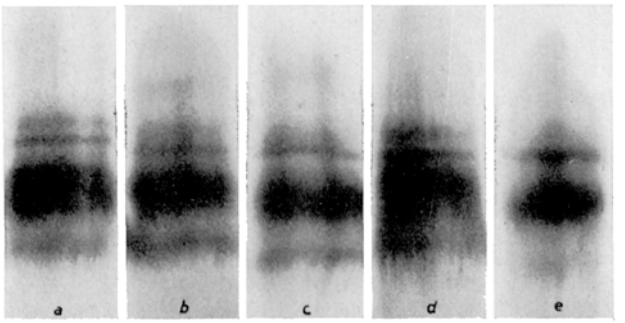

Fig. 1.—Paper electrophoretic pattern of soluble proteins of resting and regenerating rat liver. (a) Resting; (b) 24 h; (c) 60 h; (d) 240 h; (e) 30 days after the partial hepatectomy.
Staining: Amidoschwarz 10 B.

16 male albino rats, Wistar strain, fed a standard diet, were hepatectomized according to Higgins and Anderson[6]. Four rats at a time were killed 24–60–240 h and 30 days after operation; the liver was quickly taken out and perfused with a Ringer-phosphate solution both through the thoracic aorta and the inferior vena cava in order to remove completely the blood proteins. The soluble proteins were extracted by the technique of Adjutantis[7], as modified by Ciaranfi and di Sabato[8], who minimized the protein denaturation by using ultrafiltration instead of dyalisis. Paper electrophoresis was carried out as described by Ciaranfi and di Sabato[8]. The strips were stained either with Amidoschwarz 10B or with the periodic acid-leucofuchsin technique, according to Köiw and Grönwall[9].

Soluble proteins, 24–60–240 h and 30 days after partial hepatectomy, present essentially the same electrophoretic pattern as those of the resting rat liver (Fig. 1). However, a faint albumin-like peak appears 24 h after operation and disappears almost completely 60 h after the hepatectomy. These results are in agreement with those

[1] This investigation was aided by a grant from C.N.R., Rome, and was done during the tenure by one of us (G. G.) of a Scholarship from C.N.R., Rome.

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previously reported[2]. The faint albumin-like peak exhibited by the early regenerating liver may be due to: (a) increased production of albumin; (b) reduced release of albumin into the blood stream; (c) depolimerization of large molecules into smaller ones, as suggested by di Sabato[10] for cell vacuolation, a regressive process also appearing in the regenerating liver[11].

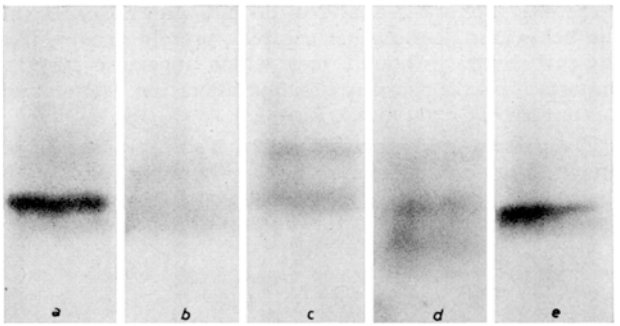

Fig. 2—Paper electrophoresis pattern of glycoproteins of resting and regenerating rat liver. The symbols are the same as in Fig. 1. Staining: periodic acid-Schiff[9].

Because of the virtual identity of the normal and regenerating liver protein patterns, evidence is given that no single protein could be regarded as a reserve material chemically different from the basal structural proteins (Luck[12]). Furthermore, since it is well known that the neoplastic liver growth is accompanied by a marked reduction in the amount of slowly moving proteins and by a large increase in the fast-moving ones[2], our results show that the liver regeneration is quite a normal growth in spite of the biochemical similarities with the liver tumors[13].

Evidence is given in Figure 2 that the liver soluble glycoproteins markedly decrease 24 h after operation. 60 and 240 h later, two faint peaks, and may be a third one, appear in the electrophoretic pattern. 30 days after hepatectomy, the glycoproteins are again normalized. This behaviour most likely accounts for a decrease of the ground substance of the liver connective tissue, secondary to the stress induced by the surgical trauma[4]. It may also be suggested that during the liver regeneration the more complex conjugated proteins synthesis is sacrificed on behalf of the synthesis of the bulk of the proteins necessary to the parenchimal re-building.

G. Guidotti and E. Clerici

Institute of General Pathology, University of Milan (Italy), June 5, 1958.

Riassunto

Gli autori hanno studiato il comportamento delle proteine e delle glicoproteine solubili del fegato rigenerante di ratto mediante l'elettroforesi su carta. La colorazione con Amidoschwarz mostra che il comportamento elettroforetico delle proteine di fegato rigenerante è praticamente sovrapponibile a quello del fegato normale. La colorazione secondo Köiw e Grönwall dimostra che le glicoproteine diminuiscono nei primi giorni dopo l'atto operatorio, tornando però alla normalità circa 30 giorni dopo l'epatectomia.

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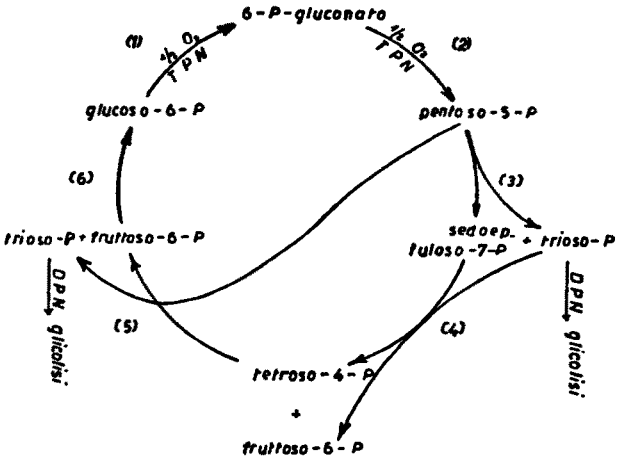
Eptoformazione non ossidativa nel muscolo scheletrico

Con il termine di «eptoformazione non ossidativa», Bonsignore *et al.*[1] hanno indicato la formazione, esclusivamente anaerobia, in assenza di TPN, di un eptosofosfato – probabilmente sedoeptuloso-7-fosfato – a partire da glucoso-6-fosfato, ad opera della frazione solubile di omogenati di fegato di ratto.

Normalmente invece, come è noto, lo stesso sedoeptuloso-7-fosfato viene considerato un prodotto della degradazione dei pentosi, a loro volta formatisi aerobicamente dagli esoso-fosfati, mediante l'intervento degli enzimi glucoso-6-fosfato- e 6-fosfogluconatodeidrasi (TPN specifici) della via ossidativa diretta. Attraverso la formazione intermedia di eptoso, i pentosi possono poi essere convertiti anaerobicamente ad esosi. Questa conversione, come è sommariamente indicato nello schema del ciclo di Horecker qui sotto riportato, è catalizzata dagli enzimi transchetolasi e transaldolasi.



(1) Glucoso-6-fosfato-deidrasi; (2) 6-fosfogluconato-deidrasi; (3) transchetolasi; (4) transaldolasi; (5) transchetolasi; (6) esosofosfatoisomerasi.

Per spiegare la «eptoformazione non ossidativa», Bonsignore *et al.*[1] ammettono appunto l'intervento catalitico di questi enzimi transchetolasi e transaldolasi in senso inverso a quello riportato nello schema, cioè nel senso della trasformazione anaerobia degli esosi (glucoso-6-fosfato e fruttoso-6-fosfato) a pentosi.

Nella presente nota dimostriamo che la eptoformazione anaerobia a partire da glucoso-6-fosfato (e da fruttoso-6-fosfato) ha luogo anche ad opera di estratti enzimatici di muscolo scheletrico. Nel muscolo scheletrico, sprovvisto, completamente[2] o quasi [3], delle attività deidrasiche della via ossidativa diretta (glucoso-6-fosfato- e 6-fosfogluconato-deidrasi), la dimostrazione di tale «eptoformazione non ossidativa» potrebbe assumere particolare importanza, in quanto potrebbe indicare la via seguita da questo tessuto per la biosintesi dei pentosi.

L'estratto enzimatico è stato preparato da muscolo scheletrico di ratto con il metodo di Glock e McLean[3], seguito da Bonsignore *et al.*[1]. Il metodo consiste essen-

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