The Lymphatic Drainage of the Peritoneal Cavity with Reference to the Treatment of Ascites by Lympho-Venous Shunt

by G. SZABÓ

National Institute of Traumatology, Mezo Imre ut 17, H–1430 Budapest (Hungary).

The abdominal cavity contains normally only a few millilitres of fluid, but under pathological conditions vast effusions may accumulate and this may represent a serious clinical problem. The peritoneal fluid is not sequestrated but is in a steady dynamic exchange with the plasma. For the understanding of the formation and resolution of peritoneal effusions, protein absorption and transport from the abdominal cavity is of considerable interest. Protein from the peritoneal fluid enters by diffusion into the blood capillaries[1], but due to the concentration gradient a net absorption into the circulation is obviously out of question. Actually, the protein molecules are absorbed into the lymphatic capillaries and subsequently transported by the lymph vessels. The right duct carries 3 to 5 times more protein from the peritoneal cavity than the thoracic duct[1–3].

In postsinusoidal stasis due to systemic congestion or cirrhosis, most of the excess filtrate formed in the liver is removed by the lymphatic vessels joining the thoracic duct[4]. The contribution of the right duct is insignificant[5]. In dogs with caval constriction, a constant formation of drops of fluid was observed on the surface of the liver[6,7]. The fluid leaving the sinusoids and oozing from the surface of the liver has a high protein content[7]. The observation of augmented liver lymph flow, the high protein content of this lymph and of the ascites and the exuding of a protein-rich fluid from the liver suggests that in ascites some of the water and most of the protein of the peritoneal fluid are derived from the liver[7].

In patients with Laennec's cirrhosis, the thoracic duct is greatly dilated, contains fluid under pressure and when it is vented a great amount of lymph flows from this vessel[4,8,9]. A functional resistance has been assumed at the veno-lymphatic junction, leading to an impairment of flow of the excess lymph[10,11]. The cannulation of the thoracic duct or the construction of a surgical lymphato-venous shunt was advocated for the treatment of the ascites[12–14]. A number of favorable reports appeared on the value of this intervention[15–17], but various criticisms were also advanced. It was stated that there is no pressure difference across the thoracic duct-vein junction; accordingly, there is no evidence to support the presence of a relative obstruction[18,19]. An elevated pressure inside the occluded thoracic duct bears no evidence of the presence of a pressure gradient at the junction[20]. In dogs with inferior cava vein constriction, a significant pressure difference was observed, however, in the intact lymphatic system between the cervical thoracic duct and the innominate vein[14]. After the cannulation of the thoracic duct the lymph flow, tremendously augmented in this condition, decreases rapidly, indicating an initial elimination of the sequestrated fluid[1]. Similarly, the thoracic duct, which is enermously dilated in patients with cirrhosis, collapses as soon as it is vented[9].

The cannulation and external drainage of the distended thoracic duct reduces the increased plasma volume and, probably mostly by this effect, also central and hepatic venous pressures. The consecutive reduction of sinusoidal pressure leads to decreased filtration and diminished escape of the filtrate into the peritoneal cavity. The observed rapid decrease of ascites volume is not a direct consequence of the above occurences. Lymphatic absorption of ascites is but little affected, because the peritoneal cavity is drained mainly not by the thoracic duct but by the right lymph trunk. The formation of peritoneal fluid is, however, diminished and, mainly as a result of the decreased systemic venous and capillary pressures, water absorption through the peritoneal blood capillaries is increased.

The external drainage of the thoracic duct is obviously only a temporary measure; it was proposed as a preliminary operation, to reduce portal pressure before the construction of a portocaval venous shunt[21,22]. More permanent relief of ascites can be

[1] G. SZABÓ, Z. MAGYAR and P. SERÉNY, Acta med. hung., in press (1976).
[2] G. SZABÓ and Z. MAGYAR, Acta med. hung. *30*, 303 (1973).
[3] G. SZABÓ and Z. MAGYAR, Res. exp. Med. *161*, 335 (1973).
[4] R. E. HYATT and J. R. SMITH, Am. J. Med. *16*, 434 (1954).
[5] G. SZABÓ, Z. MAGYAR and F. JAKAB, Res. exp. Med., *166*, 193 (1975).
[6] W. VOLWILER, J. L. BOLLMAN and J. H. GRINDLAY, Proc. Staff. Meet. Mayo Clin. *25*, 31 (1950).
[7] R. E. HYATT, G. H. LAWRENCE and J. R. SMITH, cited in R. E. HYATT and J. R. SMITH, Am. J. Med. *16*, 434 (1954).
[8] A. E. DUMONT and J. H. MULHOLLAND, Ann. Surg. *156*, 668 (1962).
[9] L. LÉGER, , M. PRÉMONT, P. DEVISSAGUET, Presse méd. *70*, 164 (1962).
[10] R. R. BRADHAM and T. TAKARO, Surgery *64*, 643 (1968).
[11] G. SZABÓ, Z. MAGYAR, P. SERÉNYI, Acta med. hung. *27*, 257 (1970).
[12] A. E. DUMONT and M. H. WITTE, Surg. Gynec. Obstet. *122*, 524 (1966).
[13] A. E. DUMONT and M. H. WITTE, Am. J. Surgery *172*, 401 (1966).
[14] J. R. CUETO and A. CURRIE, Ann. Surg. *165*, 408 (1967).
[15] H. V. SCHREIBER, W. KOCH, H. V. ACKEREN, TH. GEORGI and K. SCHILLING, Dt. med. Wschr. *93*, 195 (1968).
[16] P. SERÉNYI, Zs. MAGYAR and G. SZABÓ, Khirurgiya *3*, 100 (1970).
[17] R. A. BHALEARO, D. E. TRIVEDI and P. K. SEN, Surgery *69*, 569 (1971).
[18] W. O. WARREN, J. J. FOMON and C. A. LEITER, Surgery *63*, 7 (1968).
[19] H. M. SHIZGAL and J. R. GUTELIUS, Ann. Surg. *169*, 519 (1969).
[20] G. SZABÓ and Zs. MAGYAR, Am. J. Physiol. *212*, 1469 (1967).
[21] W. F. BOWERS, W. M. MCKINNEN, J. M. MAVINO and J. T. CULVERWELL, J. int. Coll. Surg. *42*, 71 (1964).
[22] J. R. CUETO, N. TAJEN and R. A. CURRIE, Arch. Surg. *96*, 9 (1968).

expected from a lymphato-venous anastomosis. If the main obstacle to the elimination of the excess lymph is a relative constriction at the orifice of the thoracic duct, then the transsection of the duct and the construction of a shunt with a new, wider opening may warrant a better lymph drainage. The advantage of this intervention would be that there is no water, electrolyte and, most importantly, protein loss from the organism. This is also the main ground for the criticism against the shunt operation. If there is no fluid loss systemic venous and sinusoidal pressures will not decrease and consequently ascites formation is not reduced[20,12]. In dogs with congested inferior cava vein, the venous pressure does not decrease significantly after the construction of a cervical lymphato-venous shunt, but the mean pressure in the thoracic duct decreases[14], signaling that there is no longer any obstacle to lymph flow, and consequently that the shunt has had a favorable effect. The effect is based mainly on the facilitation of the lymphatic transport of the excess capillary filtrate. In view of the encouraging clinical results, this intervention is indicated for the relief of ascites in some patients with liver cirrhosis. In an unpublished series of experiments it was proved that an ascites induced by the supradiaphragmatic constriction of the vena cava inferior can to a great extent be prevented by administration of the benzopyrone-preparation Venalot®.

Conclusion

by M. Földi

Research Laboratories Schaper & Brümmer, P. O. Box 61 1160, D-332 Salzgitter 61 (German Federal Republic, BRD).

The aim of the present report has by no means been to achieve complete covering of the subject. The intention of the initiator, Professor Mislin, was to create a Review of the current trends in Lymphology.

Canalicular lymphatic drainage and synergistic extralymphatic cellular plasma protein mastering are vital in the maintenance of the internal milieu of mesenchymal tissues. As these tissues are scattered, there is no organ the function of which can be understood – either in health, or in disease – if the lymphatic system is not taken into consideration.

SPECIALIA

The Structure of Isocedrolic Acid Isolated from *Juniperus squamata* Lamb.

Y. H. Kuo, S. H. Hsieh, S. T. Kao and Y. T. Lin

Department of Chemistry, National Taiwan University, Roosevelt Road Section 4, Taipei (Taiwan 107, China), 2 February 1976.

Summary. Isocedrolic acid isolated from *Juniperus squamata* Lamb. was established as 8s-hydroxycedrane-12-carboxylic acid by chemical and physical evidence.

Material and methods. In a previous paper[1], we described the isolation of α-cedrol, 8s, 14-cedrandiol, and a new compound 4-ketocedrol **1** from neutral fraction of a *n*-hexane extract of wood of *Juniperus squamata* Lamb. Now we isolated isocedrolic acid **2a** together with cedrolic acid[2], hinokiic acid, and widdringtonia acid II[3] from the acidic fraction of the same extract. Isocedrolic acid was discovered in *Juniperus procera*[4] in 1961, but its structure was still obscure. This communication describes the structure elucidation.

Result and discussion. Isocedric acid **2a**, m.p. 259 to 261 °C, $C_{15}H_{24}O_3$, $[\alpha]_D$ −24.8 (c. 0.5 in CH_3OH), exhibits IR-absorption bands at 3320, 3100, 2500, and 1670 cm^{-1}. It shows NMR-spectrum signals at τ_{CDCl_3} 9.04 and 8.85 (each of 3H, s, $=C(CH_3)_2$) and 8.74 (3H, s, $=C(OH)CH_3$). The structure of **2a** was suggested to be a derivative of cedrol by the similarity of its NMR-spectrum pattern with that of α-cedrol, except for the carboxylic acid group instead of a secondary methyl group. **2a** gave an amorphous product **3** by heating in 99% formic acid

[1] Y. H. Kuo, I. C. Yang, C. S. Cheng and Y. T. Lin, Experientia *32*, 686 (1976).
[2] K. H. Baggaley, H. Erdtman and T. Norin, Tetrahedron *24*, 3399 (1968).
[3] J. Runeberg, Acta chem. scand. *14*, 1985, 1991 and 1995 (1960).
[4] E. Pettersson and J. Runeberg, Acta chem. scand. *15*, 713 (1961).