

Serum Lipid Concentration in Blood from Afferent and Efferent Pulmonary Vessels in Man. (21151)

ALVAR SVANBORG. (Introduced by D. D. Van Slyke.)

From the Departments of Clinical Biochemistry and of Medicine, Karolinska sjukhuset, Stockholm, and Sahlgrenska sjukhuset, Göteborg, Sweden.

In experiments on dogs Abelous and Soula (1) observed that serum from the right heart had a higher cholesterol content than serum from the left heart. Furthermore, they found an autolytic decrease in the cholesterol content in extirpated lung tissue that had been protected from bacterial decomposition under storage. In their opinion these observations indicated that breakdown of cholesterol takes place normally in the lung tissue. Bouisset and Soula(2) demonstrated that the decrease in serum cholesterol in the passage through the lungs increased during hyperventilation. Since that time numerous writers have reported similar results. A review of the literature may be found in Shillito *et al.*(3). Bugnard(4) believed he could verify the observations of earlier investigators of a decrease in the cholesterol concentration in serum in passage through the lungs, but contended that this decrease was due to a migration of cholesterol into the blood corpuscles. This cholesterol migration from the serum to blood corpuscles he considered regulated by changes in the pH of blood (Bugnard(4)). Other investigators have reported contradictory results. Markowitz and Mann(5) were unable even after increased fat administration to demonstrate differences in the total lipid concentration in afferent and efferent pulmonary vessels in dogs. Shillito *et al.*(3) reported similar results in serum cholesterol investigations in dogs.

Determinations of the lipid concentration in serum from the right heart have not heretofore been carried out in man. Such an investigation is reported in the present work. Blood was collected for analysis from the brachial artery by puncture and from the pulmonary artery at heart catheterization. The patients studied exhibited no signs of pulmonary disease. Two patients were investigated in conjunction with abdominal operations. One

patient was catheterized because of suspected heart disease, but the findings at the examination were normal. Six patients had heart diseases; 4 of these without right-to-left shunt. For comparison 2 cases of heart disease with right-to-left shunt were included. The diagnoses appear in the table.

Methods. Total lipid content determination. The serum was dropped into absolute alcohol, heated to just under the boiling point. The precipitate was removed by filtration and Soxhlet extracted with absolute alcohol for 5 hours. The alcohol was evaporated, and the lipids were redissolved in chloroform. From several portions of this chloroform solution, the lipids were determined by means of evaporation and weighing of the residue. Cholesterol and lipid phosphorus concentrations were determined according to methods described in an earlier publication (Svanborg(6)).

Results. The results are presented in the table. No tendency to a decrease in lipid concentration in the passage of blood through the lungs was demonstrated. The differences obtained lie completely within the limits of error of the method.

Discussion. It has been shown repeatedly in *in vitro* experiments that lung tissue is able to break down lipids (Falk(7); Geyer, *et al.*(8); Alfin-Slater, *et al.*(9); Meyer, *et al.*(10)). It is impossible, however, to draw definite conclusions from these *in vitro* experiments concerning the magnitude of the lipid breakdown in the lungs *in vivo*.

As pointed out in an earlier work (Svanborg(6)), it is frequently impossible to assess the quantitative enzyme activity of an organ solely by determination of differences in substrate concentration in blood from afferent and efferent vessels. It is necessary to take into consideration the possibility of enzyme activity of opposite effect in the organ, the possible accumulation of substrate in the

TABLE I. Total Lipid Cholesterol and Lipidphosphorus Concentration in Blood from Pulmonary Vessels.

Case	Pulmonary artery			Brachial artery			Diagnosis
	Cholesterol, mg/100 ml	Lipid P, mg/100 ml	Total lipids, mg/100 ml	Cholesterol, mg/100 ml	Lipid P, mg/100 ml	Total lipids, mg/100 ml	
H.N.	200	5.9	590	190	5.9	670	Peptic ulcer
P.L.	300	11.3	1250	330	11.1	1080	" "
R.P.	220	6.1	890	200	5.6	950	Normal case
J.S.	200		790	210		680	Ess. pulm. hypertension
E.K.	180	5.4	810	210	6.2	830	Pulm. artery stenosis
P.M.	200	6.9	770	190	6.5	690	Mitral valve disease
E.A.	220	7.1	880	220	7.7	1170	<i>Idem</i>
K.E.	180	7.0	650	160	7.5	630	Pat. duct. art.
S.M.	180	5.3	460	180	5.4	580	Atr. sept. defect

organ, the loss of substrate through lymph circulation, and mucosal secretion, among other factors.

It is important to establish that even if an appreciable lipid breakdown normally occurred in the lungs, the decrease in lipid concentration in blood in the passage through the lungs would be small since the rate of circulation through the lungs is extremely high. Actually, it may be estimated that this decrease in concentration cannot conceivably be sufficient to permit demonstration with the available methods of lipid concentration determination.

Consequently, the results presented here do not preclude the possibility that a lipid breakdown normally takes place in the lung tissue.

Summary. 1. Numerous earlier investigators have maintained that they demonstrated a lower serum cholesterol concentration in blood from efferent than from afferent pulmonary vessels in experimental animals. These observations have been interpreted as indications of cholesterol breakdown in lung tissue. 2. Cholesterol, lipid phosphorus and total lipid concentrations were determined in blood from the left heart (brachial artery) and the right heart (pulmonary artery) in 9 patients. No

decrease in lipid concentration in serum in the passage of blood through the lungs was demonstrated. 3. The difficulties in attempting to evaluate the quantitative enzyme activity of an organ solely by determination of the substrate concentration in afferent and efferent blood vessels is discussed.

1. Abelous, J.-E., and Soula, L.-C., *Compt. rend. Soc. de biol.*, 1921, v85, 6.
2. Bouisset, L., and Soula, L.-C., *Compt. rend. Soc. de biol.*, 1928, v99, 1206.
3. Shillito, F. H., Bidwell, E. H., and Turner, K. B., *J. Biol. Chem.*, 1936, v112, 551.
4. Bugnard, L., *Compt. Rend. Soc. de biol.*, 1929, v102, 369, 550.
5. Markowitz, C., and Mann, F. C., *Am. J. Physiol.*, 1930, v93, 521.
6. Svanborg, A., *Acta med. Scandinav.*, 1951, v141, suppl. 264.
7. Falk, K. G., *The Chemistry of Enzyme Action*, The Chemical Catalog Co., New York (1924)
8. Geyer, R. P., Matthews, L. W., and Stare, F. J., *J. Biol. Chem.*, 1949, v180, 1037.
9. Alfin-Slater, R. B., Denel, H. J., Jr., Schotz, M. C., and Shimoda, F. K., *Federation Proc.*, 1950, v9, 144.
10. Meyer, J. R., Siperstein, M. D., and Chaikoff, I. L., *J. Biol. Chem.*, 1952, v198, 105.

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