THE EFFECT OF LACTATE ON BILE SECRETION OF ANAESTHETIZED DOGS

In contradiction to the reports of earlier workers[1-3] that adrenaline reduces bile secretion[4] we have observed that it first increases and then decreases the rate of hepatic bile secretion. Lundholm[5] has recently suggested that the inhibitory action of adrenaline on smooth muscles is due to the lactic acid which is formed by glycogenolysis after the administration of adrenaline. It is well known that adrenaline increases the glycolysis in liver. Sahyum and Webster[6] have reported that liver glycogen is first decreased and then increased after the administration of adrenaline. Kato and Kinura[7] have shown that adrenaline increases hepatic vein blood lactic acid concentration more than that of peripheral or portal blood. The present work was undertaken to investigate the possibility of this lactic acid playing any role in the hepatic secretory response to adrenaline.

Healthy adult dogs of either sex (8–10 kg.) were used for the study, the procedure for collection of bile was described by Ramprasad and Sirsi.[8] After cannulating the bile duct the bile was allowed to flow for about thirty minutes since within this period the rates of bile flow become steady. Then two control samples of bile were collected at 15 minute interval and the infusion of lactate (6 to 7 mg./min./kg.) was started through the femoral vein in one set of five dogs and through the portal vein in another set of five dogs. Infusion was continued for one hour and four bile samples were collected at 15 minute intervals during the infusion. After discontinuing the infusion four more bile samples were collected at the same intervals. Since the hypertonic solutions have been shown to reduce bile secretion[9] the isotonic solution of sodium lactate (1·85% w./v.) was employed for infusion. Similar experiments were performed on three dogs with the exception that no infusion was given. This served as control. All the bile samples collected were analysed for bile acids,[10] bilirubin[11] and cholesterol.[12]

The results are summarised in Table I. In control experiment no significant alteration in the rate of bile secretion was observed; however, the concentration of bile acid in bile, progressively declined over a period of two hours. The concentration of cholesterol in bile decreased only slightly while that of bilirubin remained unaltered.

TABLE I

Effect of lactate on the excretion of bile and its constituents

Time in min.	Rate of bile secretion mg./min./kg.			Total bile acid excretion in mg./15 min.			Total cholesterol excretion in mg./15 min.			Total bilirubin excretion in mg./15 min.		
	C	LF	LP	C	LF	LP	C	LF	LP	C	LF	LP
15	5·2	5·50 ↓	3·80 ↓	11·99	10·43 ↓	10·0 ↓	0·59	0·77 ↓	0·72 ↓	1·07	1·00 ↓	1·08 ↓
30	5·16	4·85	3·66	11·23	10·82	10·5	0·58	0·68	0·77	1·05	1·37	1·33
45	5·06	5·85	4·08	9·08	11·34	11·6	0·53	0·99	0·81	1·04	2·27	1·85
60	5·13	6·10	4·06	9·06	10·16	11·6	0·54	1·09	0·81	1·02	2·51	2·08
75	5·16	6·25 ↑	4·53 ↑	9·14	9·56 ↑	12·7 ↑	0·54	1·14 ↑	1·08 ↑	1·00	2·17 ↑	2·58 ↑
90	5·60	6·45	3·86	9·20	8·82	10·8	0·59	1·29	1·03	1·09	2·24	2·64
105	5·60	5·80	3·20	9·23	7·61	9·0	0·54	1·02	0·63	1·09	1·94	1·82
120	5·63	5·75	3·30	7·73	6·59	9·2	0·49	0·83	0·65	1·02	1·95	1·65
135	5·65	5·20	3·43	6·88	7·17	9·0	0·49	0·72	0·68	1·10	1·89	1·28

C = Control; LF = Lactate infusion through femoral vein; LP = Lactate infusion through portal vein;
↓ = Infusion start; ↑ = Infusion stop.

The infusion of lactate through the femoral or portal vein resulted in the increased rate of bile secretion. The analysis of bile (collected before, during and after infusion) shows that excretion of bile acids, cholesterol and bilirubin gradually increased during the period of infusion as compared to the control. These effects were more marked when the infusion was given through the portal vein. After discontinuing the infusion these changes gradually returned to normal.

In the control experiment the excretions of cholesterol and bile acid were reduced by about 8·4 and 23·8% respectively at the end of one hour. In the dogs receiving the lactate infusion through femoral vein the excretion of bile acid was reduced only by 8·5% while that of cholesterol and bilirubin was increased by 48 and 110% respectively at the end of the same

period. In dogs receiving the lactate infusion through the portal vein the excretion of bile acid, cholesterol and bilirubin increased by 27, 40 and 138% respectively at the end of same period.

The intravenous infusion of lactate results in the increased rate of secretion of bile and excretion of bile acid, cholesterol and bilirubin showing thereby that the hepatic secretory response to lactate is an augmentory one. This response is more pronounced if the lactate infusion is given through the portal vein suggesting that the factors involved in such response are intrahepatic. Schwiegh[13] has reported that lactate augments the hepatic circulation which is in agreement with the report that lactate is a powerful vasodilator.[14] Increased circulation through the liver results in the increased bile secretion. It is therefore probable that the augmentory hepatic secretory response to lactate is due to the increased circulation through liver. The increase in the excretion of bile constituents namely bile acids, bilirubin and cholesterol during lactate infusion shows the increased hepatic cellular activity.

Adrenaline causes increased glycolysis in the liver which results in the increased lactic acid level in the hepatic blood.[7] Increased intrahepatic circulation has also been reported to follow the adrenaline injection.[15] It is possible that this hemodynamic response of liver is related to the metabolic response to adrenaline.

In summary, the effect of the infusion of isotonic solution of sodium lactate through femoral and portal vein on the rate of secretion and composition of bile has been studied in anæsthetized dogs. The infusion of lactate through femoral or portal vein results in increase in the rate of the secretion with concomitant increase in the excretion of bilirubin, cholesterol and bile acids through it. These changes were more marked when infusion was given through the portal vein.

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A NOTE ON THE STRATIGRAPHY AND MICROFAUNA OF THE KIRTHAR BEDS OF THE JAISALMER AREA

THE presence of Kirthar beds from Rajasthan was first noted by Singh.[1] He recorded its occurrence from a locality near Kolayat in Bikaner area and extended the paleogeographical coastline of the Kirthar sea (previously limited only to western extremity of western India, Singh[2]) further east to near Bikaner. Subsequently Chatterji[3] reported the occurrence of another outcrop of the Kirthars from the Jaisalmer District and thus further extended the Kirthar coastline up to Jaisalmer, southwest of Bikaner. Chatterji[3] (*op. cit.*) recognised two distinct horizons of the Eocene beds in the Jaisalmer area—one containing *Nummulites atacicus* Leymerie and *Assilina granulosa* (d'Archiac) of Laki age and the other, an upper one, containing *Dictyoconoides cooki* (Carter) *Alveolina oblonga* d' Orbigny along with *Nummulites atacicus* and *Assilina granulosa* of Lower Kirthar age. The exact locality at which the Kirthar beds occur was, however, not given by Chatterji.

Recently Mathur and Evans[4] have given a complete stratigraphical succession along with a geological map of the Jaisalmer District. They assign the beds exposed at Bandah to Kirthars and the beds at Khuiala (a locality five miles south-east of Bandah) to Lakis.

The object of this short note is to give in brief the little-known stratigraphy and the important microfauna of the Kirthar beds exposed at Bandah (70° 21′ E : 27° 11′ N.), district Jaisalmer. The stratigraphical succession as exposed at Bandah is given below. The Kirthar beds are divisible into four units on the basis of their contained fauna and lithology.