Title: EFFECTS OF THE SUGIURA'S ESOPHAGEAL TRANSECTION ON THE POSSIBLE MECHANISMS OF HYPOXEMIA IN HEPATIC CIRRHOSIS.

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Introduction: The mechanisms of hypoxemia frequently observed in patients with hepatic cirrhosis have not been established. An attempt was made to identify the cause of hypoxemia by examining the effects of the esophageal transection plus paraesophageal devascularization (Sugiura's method) on pulmonary function and cardiovascular dynamics in patients with hepatic cirrhosis and portal hypertension.

Methods: Fourteen consenting patients were studied before and after the operation. All patients studied had the classical clinical and laboratory evidence of hepatic cirrhosis and portal hypertension. None of these patients showed any evidence of active cardiorespiratory disease. None had any significant amount of ascites at the time of the study. A few days before the operation, closing capacity (CC) was measured by a single breath nitrogen washout method while they were in sitting position. FRC was also measured and CC/FRC was calculated. The day before the operation, a tripple lumen thermodilution pulmonary catheter was inserted in all patients and measurements and calculations were made for cardiac index (CI), $\dot{Q}s/\dot{Q}t$, $P(A\text{-}a)O_2$ and other hemodynamic parameters. The esophageal transection and paraesophageal devascularization were performed through a left thoracotomy by the Sugiura's method under general anesthesia. One to three months after the operation when patients fully recovered, they were re-admitted and the above measurements were repeated. The results were compared between the preoperative and the postoperative values by student's t-test. Correlation coefficients were calculated between the preoperative values in PaO_2 on room air, $\dot{Q}s/\dot{Q}t$, $P(A\text{-}a)O_2$, CI and CC/FRC. Correlation coefficients were also calculated between the changes in these values after the operation.

Results: Before the operation, the patients had increased $\dot{Q}s/\dot{Q}t$ and $P(A\text{-}a)O_2$. The mean CC/FRC value was 116 ± 16.0% of the predicted value. Mean PaO_2 on room air while patients were in supine position was 75.7 ± 6.0mmHg. There were no correlations between the preoperative PaO_2 value and the preoperative values of $\dot{Q}s/\dot{Q}t$(n=14, r=0.1826), $P(A\text{-}a)O_2$(n=14, r=0.3877) or CC/FRC(n=9, r=-0.3847). After the operation, PaO_2 on room air increased significantly by 9% while $\dot{Q}s/\dot{Q}t$, $P(A\text{-}a)O_2$ and CC/FRC decreased significantly by 16.8%, 23.6% and 10.3% respectively. There were no significant correlations between the increase in PaO_2 and the decrease in $\dot{Q}s/\dot{Q}t$(n=14, r=-0.1183), $P(A\text{-}a)O_2$ (n=14, r=-0.1382) or CC/FRC(n=9, r=0.4236). The decrease in $\dot{Q}s/\dot{Q}t$, $P(A\text{-}a)O_2$ or CC/FRC also did not correlate with each other. CI decreased by 17% after the operation. The decrease in CI correlated with the decrease in $\dot{Q}s/\dot{Q}t$ (n=14, r=0.8467). Pulmonary capillary wedge pressure (PCWP) decreased by 40.7% while pulmonary vascular resistance (PVR) increased by 18.6% after the operation.

Discussion and Conclusion: Although PaO_2 on room air increased and CC/FRC, $\dot{Q}s/\dot{Q}t$ and $P(A\text{-}a)O_2$ all decreased significantly after the esophageal transection and paraesophageal devascularization, the increase in PaO_2 did not correlate with the decrease in CC/FRC, $\dot{Q}s/\dot{Q}t$ or $P(A\text{-}a)O_2$. It has been suggested in the past that increased CC, intrapulmonary shunt or impaired hypoxic pulmonary vasoconstriction (HPV) may be the mechanisms for the hypoxemia associated with hepatic cirrhosis. However, Funahashi et al[1] did not find a consistant relationship between hypoxemia and CC in their patients with hepatic cirrhosis. Our results were in agreement with their finding. Although we did not study the pulmonary vascular response to hypoxia in our patients, Naeije et al[2] did not find the increased frequency of impaired HPV in their patients with hepatic cirrhosis. Our results and their recent reports suggest that none of increased CC, intrapulmonary shunt and impaired HPV can be considered as the sole mechanism to explain the hypoxemia associated with hepatic cirrhosis.

References:

1. Funahashi A, et al: Hypoxaemia and cirrhosis of the liver. Thorax 31:303-308, 1976
2. Naeije R, et al: Hypoxic pulmonary vasoconstriction in liver cirrhosis. Chest 80:570-574, 1981

Table:

	Pre-operation	Post-operation	% Change	P
$\dot{Q}s/\dot{Q}t$ %	18.0 ± 5.1	14.9 ± 3.4	-17.2	< 0.001
CC/FRC %	119 ± 10	107 ± 17	-10.3	< 0.05
$P(A\text{-}a)O_2$ mmHg	220 ± 34	168 ± 43	-23.6	< 0.001
PaO_2 mmHg	75.7 ± 6.6	82.5 ± 6.3	+9.0	< 0.01
C.I. L/min/m²	4.03 ± 0.44	3.43 ± 0.34	-14.9	< 0.001
M.P.A.P. mmHg	15.3 ± 1.2	10.6 ± 1.8	-30.7	< 0.001
P.C.W.P. mmHg	11.3 ± 1.7	6.7 ± 2.3	-40.7	< 0.001
P.V.R. dyne-sec/cm⁵	56.3 ± 24.2	66.3 ± 29.2	+17.8	< 0.001

(Mean ± SD)