TABLE I.
Influence of Tagathen on Production of Histamine-Induced Ulcers in Guinea Pigs.

Treatment*	No. animals	Animals showing gastric pathology	
		No.	%
Kale†	11	0	0
Kale† + histamine‡	45	44	98
Histamine‡	25	23	92
No Vit. C + histamine‡	7	5	71
20 mg Vit. C + histamine‡	6	5	83
4 " tagathen§	6	0	0
4 " " + histamine‡	6	0	0
6 " " " "	6	1	17
0.1 " " " " + kale†	6	6	100
0.5 " " " " " "	6	6	100
1 " " " " " "	18	10	56
2 " " " " " "	12	7	58
4 " " " " " "	50	24	48
6 " " " " " "	19	9	47
8 " " " " " "	6	1	17
12 " " " " " "	5	1	20

* All groups received basal diet as given except as noted.
† Kale—fed *ad libitum*.
‡ Histamine diphosphate (0.6 mg) in distilled water injected i.m./pig 2×/day.
§ Tagathen in distilled water orally/pig 2×/day.

histaminic to prevent histamine-induced ulcers. It would appear that such a reduction in ulcer production in so large a group of animals clearly demonstrates the potent anti-ulcer properties of this compound.

In addition to the above reported experiments we have made preliminary investigations on the influence of desiccated thyroid, thiouracil, dried liver cake, 15-unit liver extract, insulin, thyroxine, tannic acid, 2,4-dinitrophenol and casein digests on the incidence of histamine-provoked gastric lesions.[8] None of these was found to have any marked preventive or provocative activity.

Summary. Conditions have been established for the production of gastric lesions in guinea pigs by injection of aqueous histamine without considerable loss of animals due to fatal bronchial spasm.

Tagathen causes a very pronounced reduction in the incidence of gastric lesions produced by this method.

In view of the favorable results obtained, it would seem that a study of the mechanism involved in the anti-ulcer effect produced by antihistaminic compounds of this type, should be made.

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17119 P. Blood Flow and Oxygen Consumption of the Brain in Coarctation of the Aorta.*

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Recent developments making possible measurements of the blood flow to the brain[1,2] and myocardium[3] in man[4] enable the clinical in-

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vestigator of the hemodynamics in coarctation of the aorta to make for the first time an accurate estimate of the pressure-flow relationship in these regions. Reports concerning the circulatory alterations in this malformation have not established decisively whether or not vascular resistance is increased uniformly throughout the body.[5-10] Bing suggests that there is no generalized elevation of peripheral vascular resistance.[11] This preliminary report is to present evidence showing that cerebral blood flow and oxygen consumption in two patients with coarctation are significantly increased above normal.

Studies in this laboratory over the past four years have indicated that there is increased resistance to blood flow through the brain in arterial hypertension of unknown cause, this region sharing in equal measure the generalized increase in vascular tone which is associated with essential hypertension.[12,13] We

TABLE I.

Cerebral Circulatory Data with Normal Circulation,[2] Essential Hypertension, and Coarctation of the Aorta.

Group	Cerebral blood flow, cc/100 g/min.	Blood pressure, mm. Hg. Indirect Brachial Right Obs.	Right Mean	Left Obs.	Left Mean	Direct Femoral mean	Cerebral vascular resistance MABP/CBF	Cerebral arteriovenous O_2 diff. vol. %	Cerebral oxygen consumption cc/100 g/min.
Young normal ♂ (34 observations, 14 subjects)	54	107/69	82			86	1.6	6.3	3.3
Coarctation group, mean	*78			*†115			1.5	6.0	*4.6
S.S. 18 WM	87	130/105	113	180/100	127	107	1.5	6.0	5.2
R.F. 20 ♀	68	150/80	103			84	1.5	5.9	4.0
Essential hypertension (45 observations, 45 patients)	54					*156	*3.1	6.5	3.2

* Denotes statistically significant difference from normals.

† This is the average value of mean brachial artery pressure for the two coarctation patients where the higher brachial artery pressure of S.S. (127) was chosen as the one more closely approximating the value of mean carotid artery pressure.

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have had the opportunity to make measurements of the cerebral circulation in two patients with hypertension limited to the upper part of the body by reason of their having coarctation of the aorta. Our results indicate that the cerebral vascular resistance in these patients is lower than that found in essential hypertension.

Methods. Cerebral blood flow, cerebral oxygen consumption, mean femoral pressure and brachial artery auscultatory pressure measurements were made in two patients with coarctation and in forty-five with essential hypertension.[2] The mean pressure of the coarctation patients was calculated by using the diastolic pressure plus one third the pulse pressure as obtained by brachial artery auscultation.[14] The pressure in the carotid artery was assumed to be the same as the brachial artery pressure. The cerebral vascular resistance for the essential hypertension patients was calculated as previously described.[2]

Results. These are presented in Table I. Cerebral blood flow and calculated mean carotid pressure are significantly increased when compared with the mean values of the young normal male.[2] These data indicate that the cerebral vascular resistance in coarctation is normal, and is not increased as in essential hypertension. Cerebral oxygen consumption is also increased with cerebral arteriovenous oxygen difference not significantly different from the normals or hypertensives.

Discussion. Because S.S. had different blood pressures in each arm the higher pressure (127 mm Hg) is assumed to be more representative of the pressure in the cerebral arterial circuit. These pressure values probably underestimate mean carotid artery pressure by about 5 mm Hg because brachial artery auscultation is a lateral artery pressure measurement. This does not take the coarctation cerebral vascular resistance values out of the normal range.

Summary. 1. Measurements of cerebral blood flow, oxygen consumption and cerebral vascular resistance were made in two patients with coarctation of the aorta. The resistance to blood flow through the brain is found to be within the normal range.

2. This is in contrast with the increased cerebral vascular resistance of essential hypertension.

[14] McLeod's Physiology in Modern Medicine, 8th edition, edited by Philip Bard, Chap. 21, p. 314.

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