No poisonous glands, or ducts leading from such glands, were found at the base of the spines or along its course, either during gross dissection or histologic examination. Injections of soluble dyes (bromphenol blue and methyl blue) around the base of the spines (both in vivo and after dissection) resulted in no diffusion of the dye along the course of the spine either with or without the aid of gravity. The morphologic structure of the spine is essentially the same in the newborn dogfish pup as it is in the adult dogfish.

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SPLENIC VEIN BLOOD FLOW AND HEMATOCRIT RESPONSES TO SYMPATHOMIMETIC DRUGS IN Squalus acanthias

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The following observations show that in contrast to mammals Squalus acanthias does not possess a readily mobilizable reserve of normal erythrocytes which are released into the general circulation in response to sympathetic receptor stimulation. The effects of two classic sympathomimetic drugs, L-epinephrine and isoproterenol, on arterial pressure (dorsal and/or ventral aorta), hematocrit (systemic or splenic vein), splenic vein blood flow and heart rate were observed both before and after sympathetic alpha or beta receptor blockade in 23 lightly anesthetized (pentobarbital sodium, 20 mgm/Kgm) fish whose gills were perfused with seawater. Central venous hematocrits (sinus venosus) were obtained from 6 fish 1-10 minutes after injection (ventral aorta) of L-epinephrine or isoproterenol (2×10^{-5} mgm/Kgm). The spleens of 4 fish were exposed and stimulated electrically (0.5-20 volts, 2-50 cycles/sec) without observable evidence of a contractile response.

Central venous sampling revealed no significant change in hematocrit in response to L-epinephrine or isoproterenol although ventral aortic pressure increased to 149% of control.

Tables 1 and 2 show the responses to L-epinephrine and isoproterenol before and after blockade by phentolamine, an alpha blocker, and propanolol, a beta blocker. No significant effects on heart rate were observed. L-epinephrine increased arterial pressure significantly both before and after alpha or beta receptor blockade. Splenic vein hematocrit was significantly decreased by L-epinephrine. This effect was blocked by phentolamine but not by propanolol. Phentolamine blocked the decrease in splenic vein flow elicited by L-epinephrine but propanolol did not.

Isoproterenol, a beta receptor stimulator, decreased arterial pressure. Propanolol, the beta receptor blocker, reversed this effect, but the isoproterenol response was not affected by phentolamine. A barely significant decrease in splenic vein hematocrit occurred in response to isoproterenol in the presence of phentolamine block. Isoproterenol tended to increase splenic vein blood flow, but the response was too variable to make the result significant. However, after beta blockade (propanolol) a significant increase in splenic blood flow was observed. However, control arterial pressure and blood flow were so very low that the slight increase in actual arterial pressure and blood flow which occurred resulted in a large percentage increase.

The decrease in the hematocrit of splenic vein blood following L-epinephrine injection is of considerable increase because in mammalian species an increase is observed (to 85-90%) in

Table 1

EFFECT OF L-EPINEPHRINE BEFORE AND AFTER AUTONOMIC BLOCK

Treatment	N	Dorsal aortic press.		Splenic vein hct.		Splenic vein blood flow	
		Maximum response at time (t)	Recovery at time (t)	Maximum response at time (t)	Recovery at time (t)	Maximum response at time (t)	Recovery at time (t)
		% of cont.	% of cont.	% of cont.	% of cont.	% of cont	% of cont.
L-epinephrine 1×10^{-4}/KGM injected dorsal aorta	12	285 ± 34.3* (5') $p > 0.01$	196 ± 30.6 (30') $p > 0.01$	74 ± 5.01 (3') $p > 0.01$	101 ± 13.6 (30') p = N.S.	36 ± 10.9 (3') $p > 0.01$	132 ± 31.2 (30') p = N.S.
Phentolamine 1 MGM/KGM followed 5-10' later by L-epinephrine 1×10^{-4}/KGM	8	356 ± 87 (5') $p > 0.01$ --- 325 ± 74.5 (10') $p > 0.01$	188 ± 18.4 (30') $p > 0.01$	127 ± 38 (10') $p > 0.1$	141 ± 80 (30') p = N.S.	173 ± 35.1 (2') $p > 0.05$ --- 72 ± 41 (5') p = N.S.	240 ± 17.8 (30') $p > 0.02$
Propanolol 1 MGM/KGM followed 5-10' later by L-epinephrine 1×10^{-4}/KGM	6	205 ± 16 (2') $p > 0.01$ --- 161 ± 17 (10') $p > 0.05$	142 ± 13 (30') $p > 0.0<$	55 ± 8 (5') $p > 0.01$	96 ± 11 (30') p = N.S.	36 ± 15 (3') $p < 0.02$	20 ± 12 (30') $p > 0.01$

*Standard error.

Table 2

EFFECT OF ISOPROTERENOL BEFORE AND AFTER AUTONOMIC BLOCKADE

Treatment	N	Dorsal aortic press.		Splenic vein hct.		Splenic vein blood flow	
		Maximum response at time (t)	Recovery at time (t)	Maximum response at time (t)	Recovery at time (t)	Maximum response at time (t)	Recovery at time (t)
		% of cont.	% of cont.	% of cont.	% of cont.	% of cont.	% of cont.
Isoproterenol 1×10^{-4}/KGM	11	41 ± 7.8* (30') p > 0.01		92 ± 5.5 (5') p > 0.2	94 ± 3.3 (30') p > 0.1	163 ± 58 (2') p = N.S.	
						88 ± 31 (10') p = N.S.	123 ± 59 (30') p = N.S.
Phentolamine 1 MGM/KGM followed by Isoproterenol 1×10^{-4}/KGM	7	38 ± 13 (3') p > 0.01	None	86 ± 4.8 (30') p > 0.05		101 ± 16 (2') p = N.S.	
						85 ± 15 (5') p = N.S.	38 ± 16 (30') p > 0.02
Propanolol 1 MGM/KGM followed by Isoproterenol 1×10^{-4}/KGM	6	221 ± 66 (5') p > 0.05	196 ± 42 (30') p > 0.05	137 ± 23 (5') p = N.S.	107 ± 6 (30') p > 0.1	461 ± 98 (5') p > 0.05	221 ± 39 (30') p > 0.05

*Standard error.

the dog). Indications are that the dogfish spleen does not sequester erythrocytes. Hence, the decrease in hematocrit may be due to a very rapid increase in plasma volume.

Phentolamine apparently is not as an effective alpha blocker in the dogfish as in mammals. Propanolol, on the other hand, effectively blocks the vasodepressor effect of isoproterenol. These observations suggest that qualitative differences in the organization of autonomic control of the circulation may exist between this species and mammals.

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HEMODYNAMIC RESPONSES OF Squalus acanthias TO IMPOSED STRESS

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Six fish Squalus acanthias, 2-6 Kgm, were lightly anesthetized with pentobarbital sodium (20 mgm/Kgm). Short cannulae or catheters connected to P23AA Statham gages were placed in the ventral and dorsal aortae while the gills were perfused with seawater. Arterial pressures were recorded (Electronics-for-Medicine IR-4) by a relatively high fidelity recorder system (resonant frequency of catheter-transducer greater than 25 cps). Responses to injections of L-epinephrine, saline, $CaCl_2$ and isoproterenol in random order were observed. The object of the experiments was to produce a variety of cardiovascular loadings which would elicit cardiovascular reflexes or reveal physical characteristics of the arterial system of this species. The responses have been interpreted in relation to typical responses in mammalian species.

Injections (dorsal aorta) of L-epinephrine (2×10^{-5} mgm/Kgm); saline, (30 ml in 5 ml) increments/30 seconds); and $CaCl_2$ (1 ml, 2M), increased the foot to peak time of both ventral and dorsal aorta pressure pulses. The delay in transmission time of the pressure pulse between ventral and dorsal aorta (mean distance approximately 40 cm) was decreased by all three interventions. Systolic, diastolic and pulse pressure increased consistently in response to L-epinephrine injection and saline loading. However, in response to L-epinephrine average dorsal aortic systolic, diastolic and pulse pressure increased more than ventral aortic pressure (ventral/dorsal aortic systolic pressure increases were 12.0 and 15.7 mmHg respectively; diastolic, 9.4 and 11.3 mmHg; pulse pressure, 2.8 and 4.2 mmHg). But in response to saline loading ventral aortic pressures increased more than dorsal aortic pressures (ventral/dorsal systolic pressure increases were 10.8 and 7.0 mmHg, respectively; diastolic, 2.5/2.2 mmHg, pulse pressure, 8.5/5.1 mmHg). Consideration of these results and application of mammalian hemodynamic concepts result in tentative conclusions that: the ventral aortic system is less compliant than the dorsal aortic system (response to saline loading); that the dorsal aortic system perhaps vasoconstricts to a greater degree than the ventral aortic system in response to L-epinephrine (greater elevation of pressure); but that the predominant effect of both L-epinephrine and saline loading was to increase cardiac stroke volume (increase in pulse pressure). The latter point leads to consideration of the effect of L-epinephrine and saline loading on cardiac cycle length; saline loading consistently (all 6 trials) increased cycle length; L-epinephrine injection resulted in four instances of increased cardiac cycle length and two decreases. Obviously, the increase in pulse pressure