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Shock Associated with Deep Muscle Burns.*

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Current views are still at variance as to whether surface burns lead to shock through drainage of fluid through the burned surface or through the formation of toxic substances in the damaged tissue. The question has also been raised whether any toxic substance that may be formed is not due solely to bacterial infection. In order to study this subject further we have observed the course of events after production of extensive aseptic deep muscle burns in the hind legs of anesthetized dogs by means of strong diathermy currents. In such preparations the volume of fluid which accumulates at the sites of the burns can be fairly exactly measured by immersion of the extremities.

Methods. Dogs, anesthetized with initial injections of 4 mg per kg of morphine and 175 mg per kg of sodium barbital, were used. The diathermy current from a Victor spark gap high frequency oscillator was led through moulded metal electrodes applied to the shaved skin with an intervening layer of electrocardiographic paste. The temperature of the deep structure, recorded with a Leeds and Northrup micromax by means of an iron-constantin needle thermocouple, was raised to 66° to 91°C during application of the diathermy current for 0.5 to 1.5 hours in the different experiments. About a third of the thigh muscles, or the thigh and calf muscles of one leg or the thigh and calf muscles of both legs were burned. The amount of fluid accumulating at the site of the burn was measured by recording the displacement of water when the legs were immersed separately in a tall cylindrical vessel until the symphysis pubis and iliac crest rested upon the mouth of the vessel. The opposite extremity was elevated about 40° above the horizontal and extended. The depth to which the skin entered the water was marked with quick drying paint. Two to four immersions were performed before burning and repeated again after death or, if the dog survived, at approximately 24 or 48 hours after burning.[1]

Results. In one group of experiments, 5 dogs were kept supine on animal boards during and following the burning in order that mean arterial pressure could be continuously recorded. These dogs were given small supplementary injections of sodium barbital from time to time. All died in 19 to 35 hours with a progressively declining arterial pressure and a rising hematocrit reading. Two of three controls observed simultaneously died, but in these the arterial pressure remained stable at the control level until within 0.5 and 1.5 hours before death. The results in these 8 dogs are summarized in Table I.

In a second group of experiments, 5 dogs were anesthetized and burned and were immediately placed in recovery cages. Water was withheld for the first 30 hours. Five control dogs were similarly treated except that they were not burned. All of the dogs survived 24 to 48 hours. Table II shows that the measured gain in volume of the legs of the burned dogs at this time was 21 to 31 ml/kg of dog. Since the dogs were all apparently in good condition, 9 of the 10 dogs were reanesthetized with 10 to 14 mg/kg of sodium pentobarbital and their mean arterial pressure recorded. The arterial pressure of the burned dogs ranged from 130 to 167 and that of the control dogs from 170 to 210. In order to see whether these dogs might be in a prodromal stage of shock[2] we studied their resistance to blood loss by comparing the volume of blood which had to be removed from the burned dogs with

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[1] For further details see Green, H. D., Dworkin, R. M., Antos, R. J., and Bergeron, G. A., *Am. J. Physiol.*, 1944, **142**, 494.

[2] Green, H. D., *Anesthesiology*, 1942, **3**, 611.

TABLE I.
Trauma by Deep Muscle Burn—M.A.P. Recorded Continuously.

Traumatized dogs							Control dogs					
					Hematocrit reading						Hematocrit reading	
No.	Exp. No.	Survival time, hr	Measured limb vol. gain, ml/kg	MAP recorded for hr	Control	Max.	No.	Exp. No.	Survival time, hr	MAP recorded for hr	Control	Max.
1	KF-2	19	0	until died								
2	" 3	25.5	10	" "	38	57						
3	" 4	35	11	" "	53	59	1	KF-5	22	until died	51	64
4	" 7	23.5	18	" "	53	63	2	" 6	17	" "	55	69
5	" 8	25.5	3.5	" "	52	69	3	" 9	Survived	27	48	48

TABLE II.
Trauma by Deep Muscle Burn—Dogs in Recovery Cages.

Traumatized dogs									Control dogs						
				A	B										
No.	Exp. No.	Time when bled, hr	MAP	Measured limb vol. gain, ml/kg	Bleeding vol., ml/kg	A + B, ml/kg	Vol. blood held out, ml/kg	Survival, hr	Exp. No.	Time when bled, hr	MAP_1	MAP_2	Bleeding vol., ml/kg	Vol. blood held out ml/kg	Survival, hr
1	KF-10	—	—	—	—	—	—	Killed 3rd day	KF-11	24	200		44	21	Survived
2	" 12	30	145	31	24	55	—	Killed end of bleeding	" 13	30	215	175	49	29.5	"
3	" 15	27	130	20.5	17	37.5	—	" " "	" 14	27	170	160	35	26	"
4	" 17	26	158	21.5	18.5	40	9	Killed 5th day	" 16	26	210	178	36	21.5	"
5	" 18	47	167	29	19.5	48.5	—	Killed end of bleeding	" 19	47	185	110	35	29	"

Footnotes for Tables I and II: Time measured from start of burning of trauma dog. Control dogs were anesthetized and observed simultaneously with the burned dogs. Limb volume gain and bleeding volume are expressed in ml/kg of body weight. MAP = mean arterial pressure. In Table II, MAP_1 is the pressure at the start of bleeding, MAP_2 is the pressure which existed when a volume of blood, equivalent to the volume of edema in the burned dogs, had been removed. Bleeding volume is the volume of blood which had to be removed in order to lower mean arterial pressure to, and to keep it at 50 mm Hg for 20 min. After establishing the "Bleeding Volume," part of the withdrawn blood was reinjected. The amount of blood which was not reinjected is indicated in the column labeled "Vol. of Blood Held Out." It will be noted that this is approximately the volume of the edema in the corresponding burned dog.

that which had to be removed from the control dogs in order to reduce their arterial pressures to 50 mm Hg. The results, summarized in Table II, show that the volume of blood which could be removed from the burned dogs was significantly lower than that of the control dogs. The sum of the local fluid accumulation in the burned legs plus the bleeding volume was in every case, however, equal to or greater than the bleeding volume of the corresponding control.

Because of the difficulty in caring for dogs with such severely burned legs, three of the burned dogs were killed immediately after the bleeding, and the remaining two were sacrificed on the third and fifth days. Only part of the withdrawn blood was reinjected into the controls, 21 to 29.5 ml/kg of body weight being withheld. All survived indefinitely.

Conclusions. It was not found possible to produce a progressive decline in arterial pressure, leading to death in lightly anesthetized dogs by means of deep muscle burns alone. A prodromal state of shock was produced, however, since the resistance of these dogs to bleeding was significantly less than that of control dogs similarly anesthetized but not burned. Since the volume of blood which had to be removed from the burned dogs in order to lower their mean arterial pressure to 50 mm Hg, plus the volume of edema in the legs, was as large as or larger in each case than the bleeding volume of the corresponding control dogs, it is concluded that the accumulation of fluid at the site of the burn is the principal factor responsible for the prodromal stage of shock in the burned dogs. Association of the extensive muscle destruction with restriction of movement and with prolonged moderately deep anesthesia converts the prodromal stage into outspoken shock with death of the dog even when the accumulation of fluid at the site of the burn is quite small.

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Functional Polarization in Retinal Development and Its Reestablishment in Regenerating Retinæ of Rotated Grafted Eyes.*

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The retina of adult salamander eyes always degenerates and is replaced by regeneration from surviving peripheral cells when the eye is grafted,[1,2,3] even repeatedly,[4] or when its blood supply is temporarily interrupted.[5] If the optic nerve is cut without interfering with the blood supply the retina survives and the nerve immediately regenerates.[5] This offers unusual opportunity to test the functional quadrants of the retina.

When Sperry first reported[6] reversed visuomotor responses in salamanders after optic nerve regeneration in eyes rotated *in situ*, I examined the effects of various types of rotation on vision in 100 grafted adult *Triturus viridescens* eyes in which a new functioning retina could be tested.

Normal visuomotor responses in salamanders are demonstrated when the animal moves toward, snaps at and follows a lure approaching any of the 4 quadrants in the field of vision. The animal also automatically moves

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1 Stone, L. S., and Zaur, I. S., *J. Exp. Zool.*, 1940, **85**, 243.

2 Stone, L. S., and Ellison, F. S., PROC. SOC. EXP. BIOL. AND MED., 1940, **45**, 181.

3 Stone, L. S., and Cole, C. H., *Yale J. Biol. and Med.*, 1943, **15**, 735.

4 Stone, L. S., and Farthing, T. E., *J. Exp. Zool.*, 1942, **91**, 265.

5 Stone, L. S., and Chace, R. R., *Anat. Rec.*, 1941, **79**, 333.

6 Sperry, R. W. (Abst.), *Anat. Rec.*, 1942, **84**, 20.