TABLE I.
Comparison of Results after Oral and Intravenous Administration of Alcohol Showing Alcohol Remaining in Gastro-Intestinal Tract.
Dose, 3 gm. per kg.; time, 2 hours.

	—Intravenous—		—— Oral ——			
	Dog 20	Dog 22	Dog 4	Dog 17	Dog 18	Dog 19
Wt. (kg.)	6.4	12.0	5.0	10.2	8.9	5.9
Alcohol administered (gm.)	19.2	36.0	15.0	30.6	26.7	17.7
Wt. of:						
Stomach contents (gm.)	4	8	146	31	156	13
" tissue "	78	125	69	120	144	96
†Gut, upper half "	141	184	324*	234	210	164
† " lower " "	114	219		135	175	146
Conc. of alcohol:						
Stomach contents (mg./gm.)	3.53	3.34	5.56	8.04	15.70	7.52
" tissue "	2.78	2.59	3.09	3.70	7.26	3.20
†Gut, upper half "	2.97	2.99	2.51*	3.13	3.28	3.18
† " lower " "	2.91	2.98		2.59	2.65	3.27
Blood	3.72	3.82	3.37	3.20	4.14	3.85
‡Unabsorbed alcohol:						
Stomach contents (gm.)			0.38	0.14	1.92	0.06
" tissue "			0.05	0.12	0.66	0.05
Gut, upper half "				0.04	0.06	0.03
" lower " "				—0.05	—0.05	0.05
Total unabsorbed "			0.43	0.25	2.59	0.19
% unabsorbed			2.8	0.8	9.7	1.0
% absorbed			97.2	99.2	90.3	99.0

†Including contents.
*Total gut and contents analyzed together.
‡ Weight of organ or contents X (concentration found minus average concentration from intravenous administration.)

90.0; average, 93.4. These figures do not support the conclusions of Haggard and Greenberg, whose calculations for absorption are: One-half hour, 48.8%; one hour, 67.5%; 1½ hours, 75.7%; and 2 hours, 83.7%.[8]

8049 C

Lipoid and Hemorrhagic Changes in Adrenal Cortex Following Traumatic Shock.*

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It has been shown that if profound and fatal shock is to be obtained in the intact dog by traumatization, the severity of tissue

[8] Haggard, H. W., and Greenberg, L. A., *loc. cit.*, Fig. 2, Curve B.

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injury must be very much greater than is required to induce shock symptoms in the equally healthy and vigorous animal lacking adrenal glands.[1] Evidence is presented indicating that the extreme susceptibility of the adrenalectomized dog to shock following trauma is largely due to the absence of one of the mechanisms concerned with the maintenance of normal blood volume, namely, the adrenal cortex. Data are presented here which show that the adrenal cortex of intact dogs dying from traumatic shock presents histological evidence of considerable impairment.

The lipoid studies were made on frozen sections of the adrenals variously stained with 2% osmic acid, Scharlach R, and Sudan III. For the study of adrenal cortical hemorrhages and other histological details, paraffin sections were stained with iron hematoxylin and eosin.

The dogs in both the normal and experimental groups were rigorously selected and only mature, well-nourished animals were employed. Deep nembutal and ether anesthesia was used and care was taken that the animals suffered no pain. Shock was induced by traumatization of the muscles of the hind limbs.[1] Periodic blood-pressure readings were taken by direct needle-puncture into the femoral artery and used as a criterion for the presence and duration of shock.[2] Following the temporary fall and rise of blood-pressure characteristic of primary shock, a subsequent and more gradual fall in pressure to 50 mm., or less, indicated the unquestioned presence of secondary shock.

A definite depletion in adrenal cortical lipoid occurs in dogs dying from traumatic shock as shown in Table I. Three dogs (11T, 12T, 13T), whose blood-volumes were temporarily increased and survival periods lengthened by single intravenous injections of saline showed marked lipoid depletion of the adrenal cortex at autopsy. Two dogs (14T, 15T), receiving both saline and cortical hormone were relieved of shock symptoms but nevertheless showed marked depletion of cortical lipoid following a 2-day survival period. Apparently the rise in blood-pressure above shock level resulting from saline or cortical hormone administration, is not accompanied by an immediate restoration of lipoid in the adrenal cortex. Lipoid depletion was most marked in the reticular and inner fascicular zones of the cortex. Extreme depletion of lipoid from the glomerular zone was noted in the adrenals of the 2 dogs whose survival periods were lengthened by saline and cortical hormone.

[1] Swingle, W. W., and Parkins, W. M., *Am. J. Physiol.*, 1935, **111**, 426.
[2] Parkins, W. M., *Am. J. Physiol.*, 1934, **107**, 518.

TABLE I.
Lipoid Depletion in the Adrenal Cortex Following Trauma.

Dog No.	Survival after trauma	Glomerular lipoid	Outer fascicular lipoid	Inner fascicular lipoid	Reticular lipoid	Remarks		
1N		+++	++++	++++	+++	Normal		
2N		+++	++++	++++	+++	"		
3N		+++	++++	++++	+++	"		
4N		+++	++++	++++	+++	"		
5N		+++	++++	++++	+++	"		
6N		+++	++++	++++	+++	"		
8T	5 hrs.	+++	+++	++	++	Trauma		
9T	6 "	+++	+++	++	+++	"		
10T	8 "	++	+++	++	+	"		
11T	12 "	+++	++	++	+	"	(saline)	
12T	21 "	+++	++	++	+	"	"	
13T	9 "	++++	+++	+	+	"	"	
14T	47 "	+	+++	++	++	"	"	and extract
15T	46 "	+	+++	++	++	"	"	and extract

+ practically no lipoid.
++ lipoid droplets all small.
+++ packed with small droplets plus some large droplets.
++++ packed with small droplets plus many large droplets.

Secondary shock may result in hemorrhagic changes in various organs,[3] including the adrenals.[4] Hemorrhages of the adrenal cortex have also been noted in the rat following pituitarectomy[5] and are said to occur following other conditions such as severe burns and the injections of various toxins. In view of the importance of the adrenal cortex as a factor in the maintenance of normal blood-volume[6] the occurrence of hemorrhagic changes in the adrenal cortex of the shocked dog becomes significant since such changes would undoubtedly interfere with its normal function. Table II indicates that hemorrhages may be expected to appear in the adrenal cortex of dogs 4 or more hours following trauma. Apparently the degree of trauma, rather than the survival period *per se*, determines the time of appearance and the severity of cortical hemorrhages. The hemorrhagic areas were usually confined to the reticular and fascicular zones. Occasionally hemorrhages were seen in the glomerular zone. The condition varied from a few blood points in the reticular zone to that pictured in Fig. 1, in which the entire reticular zone, a large part of the fascicular zone, and portions of the glom-

[3] Moon, V. H., and Kennedy, P. J., *Arch. Path.*, 1932, **14**, 360.

[4] Brooks, B., and Blalock, A., *Annals Surg.*, 1934, **100**, 728.

[5] Perla, D., PROC. SOC. EXP. BIOL. AND MED., 1935, **32**, 655.

[6] Swingle, W. W., Vars, H. M., and Parkins, W. M., *Am. J. Physiol.*, 1934, **109**, 488.

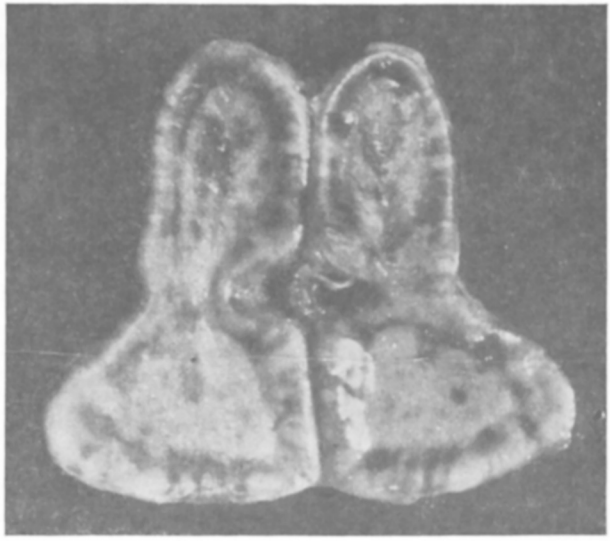

FIG. 1.
Hemorrhages of the adrenal cortex of a dog surviving 8 hrs. following very severe trauma. The dark areas represent gross hemorrhages.

erular zone, were hemorrhagic. In this, and other severe cases, hemorrhagic areas were also present in the medulla.

That the extent of hemoconcentration is reflected in the time of appearance and the severity of hemorrhagic changes in the adrenal cortex is illustrated in Table II. Dogs were subjected to intraperitoneal injections of glucose (100 cc. 5.5% solution/kilo) which was withdrawn by paracentesis after 5 hours.[7] The adrenals showed congestion of the capillaries of the cortex. In one case (28) receiving 1720 cc. of glucose slight, but definite, hemorrhages were seen in the cortex at the end of the 5-hour period.

Intraperitoneal injections of glucose followed by paracentesis after 3 hours and subsequent trauma caused gross hemorrhages of the adrenal cortex. The hemorrhages appeared sooner and were much more striking than those observed in dogs subjected to trauma alone. It is noteworthy that the dogs subjected to intraperitoneal glucose administration required considerably less traumatization for the induction of shock than those subjected to trauma alone. The marked hemorrhagic condition shown in Fig. 1 was occasionally obtained by the infliction of very severe trauma, whereas a comparable condition was commonly observed in glucose-injected dogs which were subsequently mildly traumatized.

[7] Gilman, A., *Am. J. Physiol.*, 1934, **108**, 662.

TABLE II.
Hemorrhagic Changes in Adrenal Cortex Following Trauma, Intraperitoneal Injections of Glucose, and Glucose Injections Plus Trauma.

Trauma only			Glucose*		Glucose plus trauma†		
Dog No.	Survival after trauma	Hemorrhages in cortex	Dog No.	Hemorrhages in cortex	Dog No.	Survival after trauma	Hemorrhages in cortex
20	5 hrs.	mild	26	congestion	31	3 hrs.	severe
21	6 "	none	27	"	32	6 "	"
22	8 "	severe	28	mild	33	5 "	"
23	5 "	mild	29	congestion	34	9½ "	"
24	11 "	"	30	"	35	4 "	"
25	4 "	slight			36	1 "	mild

* Glucose left 5 hrs. in peritoneal cavity.
† Glucose left 3 hrs. in peritoneal cavity.

In addition to the lipoid and hemorrhagic changes mentioned, evidence of nuclear vacuolization, particularly in the fascicular zone, and the presence of large numbers of mononuclear phagocytes were noted in the adrenals of the shocked dogs.

The data indicate that following trauma and the onset of secondary shock the adrenal cortex is subjected to severe functional strains leading to a marked depletion of lipoid, gross hemorrhages into the gland and vacuolization of cells. These changes are indicative of marked reduction in the functional efficiency of the cortex in this syndrome.

8050 C

The Endometrial Mole.

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It is well known since the classical experiments of Loeb that the uterine mucosa is sensitized by the corpus luteum hormone in such a manner that it reacts to traumatization with the formation of a deciduoma, a tumor closely resembling in its structure the maternal part of the placenta. It has been noted, however, that while deciduomata form readily during the first part of gestation, they will not form during the later stages of pregnancy although active corpora lutea are present in the ovary. We have repeated and confirmed these observations on rats, placing silk threads into the uterus as a

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