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The metabolism of the purines in man.

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Adenine, guanine, hypoxanthine and xanthine were fed at intervals to two subjects living on a constant purine-free diet. The effects of the administration of these purines (1 to 1.5 grams) on various metabolic functions, especially the partition of nitrogen in the urine, was reported. The output of urinary purines is summarized here:

	Purine fed.	Nitrogen in purine nuclens fed grams.	Increase in nitrogen eliminated as			
			Uric acid.		Purine bases.	
			gram.	per cent.	gram.	per cent.
WWH	Hypoxanthine,	0.387	.248	64	.015	4
	Xanthine,	0.369	.196	53	.009	2
	Guanine,	1.114	.347	31	.037	3
	Adenine,	0.414	.153	37	.015	3.6
JFL	Hypoxanthine,	0.387	.219	56	.009	2
	Xanthine,	0.369	.170	46	.004	1
	Guanine,	1.114	.217	19	.030	2.7
	Adenine,	0.414	.126	30	.014	3

From the numerous data reported, the authors conclude that all of the familiar purines may lead to an increase in exogenous uric acid in the urine of man, with (quantitatively) little influence on the elimination of purine bases. In contradiction to the recent suggestion of Plimmer, Dick, and Leib,[1] they interpret their protocols to support the view that uric acid is a stage in the metabolism of exogenous purines, rather than an expression of leucocyte metabolism.

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The distribution of blood in shock.

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An effort has been made to determine the percentage of blood in different organs before and after shock. About fifteen animals

[1] *Jour. of Physol.*, 1909, xxxix, 98.

(dogs) have been used so far. Under Grehant's anesthetic, the arterial blood pressure was determined. Then, under conditions of high blood pressure, certain organs, or parts of organs, were suddenly ligatured or clamped off from the circulation, and then removed with their blood content. The animal was then allowed to go gradually into a state of "shock" (for our purposes, indicated by a low pressure) or was rapidly reduced to that state by concussion or burning. Then companion organs or parts were similarly clamped off and excised. The organs were weighed, cut up into fine pieces and extracted, the blood content being determined by Welcker's method. In taking the abdominal organs, we usually proceeded in the following order: (1) a small loop of intestine clamped off or ligatured suddenly, avoiding large arteries and veins; (2) a portion of one lobe of the liver, using a large clamp suddenly applied; (3) one pole of the spleen, (4) one kidney. After "shock" the same order was followed. So far, in most of our work the same animal has served for "before" and "after" determinations. This perhaps introduces doubt as to the condition of abdominal organs if shock were produced before opening the abdomen. We shall extend the experiments with variations as soon as a large series of "before" percentages is available for averaging.

Results.—Leg: three experiments; less percentage of blood after shock in all.

Thyroid: two experiments; less after shock in both.

Intestine: six experiments; less after shock in four cases; more in two. These two were early experiments, in which we were not so careful to exclude large vessels.

Liver: seven experiments; less after shock in six cases; more in one. The one case is doubtful, as 52 per cent. of blood was indicated, probably due to an error in weighing or other manipulation.

Spleen: eight experiments; less after shock in seven; equal in one.

Kidney: nine experiments; less after shock in eight; more in one.

The differences are often extreme. In one case the kidney excised before shock contained six times as much blood as its companion excised after shock.

It seems that the anemic condition always observed in the skin in shock is also found in the organs generally. Further experiments are being performed to ascertain the location of the blood.

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The fundamental conditions of surgical shock.

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Death in shock may be either from failure of respiration, or from failure of the circulation. In Crile's experiments, and in my own, the former mode of death was much more common than the latter. As I have recently shown, deaths of this type occur also in human beings after intense pain. The excessive breathing induced by pain diminishes the carbon dioxide content of the blood and tissues. This acapnia is the cause of the depression of all functions so characteristic of shock. Finally, apnœa vera occurs in exactly the same manner as in a normal man after voluntarily forced breathing.

If death from apnœa is prevented by supplying artificial respiration, as in the majority of Crile's experiments, or by continual afferent irritation, as in my own, the circulation fails. Crile proved that this is not heart failure. Seelig and Lyon have proved that it is not vaso-motor failure, but that on the contrary the peripheral arteries are in intense constriction. Malcolm has suggested that the volume of the blood is diminished because of a passage of serum into the tissues. Sherrington and Copeman observed a considerable increase in the specific gravity of the blood even before arterial pressure had fallen to a low level. The balance between the water content of the blood and of the tissues is probably in part dependent upon their relative carbon dioxide contents. Acapnia may alter the tonus of the veins, or the relative osmotic pressure of the blood and the tissue fluids, or the imbibition tension of the colloids of blood and tissues. *Thus acapnia diminishes the volume of the blood.*

I find that acapnia induced by excessive artificial respiration, or by excessive natural breathing during stimulation of afferent nerves (*i. e.*, trauma), involves a lowered venous pressure and