human and bovine serum albumin on the blood volume after acute blood loss in normal men, reported no change in the serum or urinary potassium concentration, but their albumin solutions were not salt-poor. In 5 of the 7 patients in whom we injected salt-poor albumin a significant fall in plasma sodium occurred. In the single patient in whom potassium was measured, no change was noted. It should be emphasized that the fall in sodium concentration was most marked immediately after infusion, at which time the plasma volume had increased by a volume more than 3 times that of the fluid injected. The magnitude of the fall in sodium concentration was much less than could be accounted for by hemodilution with a sodium-free solution, hence the fluids responsible for the dilution probably contained sodium but in lower concentration than in plasma.

Because intracellular fluid is poor in sodium and rich in potassium, it is logical to assume that much of the diluting fluid must have come from this source, in which case one might expect a rise in plasma potassium. The latter was measured in 2 cases, but no change was found. However, renal clearance of potassium is so rapid that this discrepancy may not be significant. Indeed, Stewart and Rourke,[6] in the experiments cited earlier, found an increase in potassium output in the urine after hemorrhage. The red cells themselves might be assumed to be the source of the diluting fluid, but this could be true only if a rapid two-way shift of fluid were involved, inasmuch as no change in the size of red cells was observed after the injection of albumin.[2]

Summary. 1. Following phlebotomy in each of 4 normal male subjects, there was a transient fall in the plasma sodium concentration.

2. Following injections in 4 patients of a 25% solution of salt-poor human albumin solution, a similar fall in the plasma sodium concentration was noted.

3. It is concluded that sodium-poor fluid is added to the plasma, presumably from the intracellular space, following hemorrhage, as well as following injection of salt-poor human albumin.

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Environmental Conditions Which Initiate Sweating in Resting Man.

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During the course of studies[1,2,3] on the influence of environmental conditions upon the rate of water loss from the skin of man, observations were made to learn the environmental temperature and relative humidity which would initiate sweating in normal man.

Eleven subjects of both sexes, varying in ages from 12 to 59 years, were permitted to rest quietly on a hospital type of bed in an air-conditioned room. They were clothed only in a cotton gown. The rate of water loss from the skin of the epigastrium and volar surface of the right forearm was determined by a method previously described.[4]

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[2] Winsor, Travis, and Burch, George E., *Arch. Int. Med.*, 1944, **74**, 428.

[3] Burch, George E., and Winsor, Travis, *Arch. Int. Med.*, 1944, **74**, 437.

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TABLE I. Results of Studies to Learn Environmental Conditions Necessary to Initiate Sweating in Normal Man Resting in a Hospital Type Bed.*

Subject	Age	Sex	Color	Before sweating				Sweating			
				Temp. (C)	Relative humidity (%)	Rate of water loss mg/cm²/10 min.		Temp. (C)	Relative humidity (%)	Rate of water loss mg/cm²/10 min.	
						Epigastrium	Forearm			Epigastrium	Forearm
1	22	F	C	35	85	6.0	4.2	37.8	60	19.8	8.9
2	50	F	W	32.2	54	7.1	6.8	34.4	50	8.7	9.5
3	25	F	W	34.4	28	7.4	4.5	36.7	26	13.3	6.3
4	40	M	W	30.0	62	5.8	6.1	33.8	58	7.3	7.0
5	12	F	C	32.2	90	4.5	7.5	34.4	70	7.5	5.9
6	31	F	C	32.2	80	5.6	5.4	35.5	56	11.6	4.5
7	49	F	C	31.1	42	5.2	5.5	33.3	42	20.0	18.1
8	34	M	C	36.1	26	4.6	3.6	38.9	28	7.5	4.1
9	19	M	C	38.9	9.5	5.5	4.1	38.9	9	6.9	4.3
10	17	M	W	33.3	22	7.0	2.8	35.5	20	11.4	11.0
11	59	M	W	33.3	26	4.0	4.2	34.4	26	5.9	6.0
Mean				33.5	47.9	5.7	5.0	35.8	40.5	10.9	7.8
Max.				38.9	90	7.4	7.5	38.9	70	20.0	18.1
Min.				30.0	9.5	4.0	2.8	33.3	9	5.9	4.1

* The water lost before sweating is lost by diffusion. The onset of sweating in subjects Nos. 5 and 6 showed local variations.

After the subjects had rested for at least 60 minutes under comfortable environmental conditions (temperature and relative humidity approximately 20.4°C and 50% respectively) the room temperature was elevated 3°C at a time, and the relative humidity was changed in a variable fashion. After the subjects had been exposed for 15 minutes to the new room conditions their rate of water loss was measured. This procedure was continued until an atmospheric condition was reached which was associated with a definite increase in rate of water loss. Occasionally, as a check, the room temperature or humidity or both were lowered, the rate of water loss was measured, the temperature or humidity or both were raised again, and another water collection was made.

Results are summarized in Table I. Sweating was initiated by a temperature of about 36°C when the relative humidity was about 40%; with a low relative humidity, a higher air temperature was required. Sweating began at a temperature of 39°C, even though the relative humidity was as low as 9%. In one patient sweating was cyclic, *i.e.*, it alternately occurred and ceased for 15-minute periods, even though the room conditions remained unchanged. These observations suggested that sweating cooled the subject sufficiently to abolish the need for this mechanism for a certain length of time. He accumulated heat, sweated again, cooled his body, and then stopped sweating. This cyclic sweating conserves water and electrolytes.

Such observations indicate that environmental temperature of 34.4°C and relative humidity of about 50% are essentially the threshold level for sweating in normal man resting in bed. Increased exercise and rate of heat production are accompanied by a proportionate lowering of the threshold.

When the studies were prolonged, subjects became restless and irritable, and "nervous" or "psychogenic" sweating resulted, which made it impossible to observe the thermal sweating.

Summary. Observations were made on 11 subjects of varying ages to discover the environmental conditions necessary to initiate

sweating in man. These studies, performed in a room designed for close control of temperature and humidity, indicate that an environmental temperature of 34.4°C and relative humidity of about 50% are essentially the threshold level for sweating in normal man resting in bed. Exercise and increased rate of heat production are associated with a proportionate lowering of the threshold.

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Utilization of Glutamic Acid in the Presence of High Levels of Pteroylglutamic Acid.

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It has been recently suggested that pteroylglutamic acid (PGA) may function as a metabolic antagonist for glutamic acid.[1,2] The statement was made that "folic acid may competitively interfere with the nutrition of the spinal cord just as certain vitamin deficiencies in experimental animals may be caused by closely related chemicals. Thiamine deficiency, for example, may be induced by the administration of pyrithiamine, and pantothenic acid deficiency by pantoyltaurine." It was further suggested that PGA could interfere with the metabolism of the central nervous system *in vivo* or *in vitro*.[1]

The analogy between folic acid and such "anti-vitamin" compounds as pyrithiamine appears to be inappropriate. Folic acid is a member of the vitamin B complex, and is itself inhibited by "anti-vitamin" compounds which are comparable to pyrithiamine and pantoyltaurine. These compounds include "methyl folic acid,"[3,4] pteroylaspartic acid,[5] 4-amino pteroylglutamic acid,[6] certain pteridines,[7] a sulfonyl-substituted benzimidazole analogue of PGA,[8] and N^{10}-methyl pteroic acid.[9]

Since representative species of bacteria require both folic acid and glutamic acid, it is obvious that folic acid does not competitively interfere with the metabolism of glutamic acid by these bacteria. However, because the response to glutamic acid obtained with *L. casei* and *S. faecalis* R is so quantitative and sensitive, an experiment was made to determine whether massive amounts of PGA would slow up growth on suboptimal levels of glutamic acid. A culture medium deficient in glutamic acid[10] was used and PGA was added at levels of 0.01 μg, 1 μg, 10 μg, to 3 series of tubes containing varying amounts of glutamic acid. The results are illustrated in Fig. 1.

The growth curves indicate that increasing the level of PGA 1000-fold had no inhibitory

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