would be found to be again lowered, which, indeed, proved to be true. Finally, during convalescence, both pH and buffer values became normal.

The loss of buffer for alkali observed in two instances of febrile typhoid (Cases 8 and 9) is in accord with the tendency toward an alkalosis which we have observed in several cases of this disease.

TABLE 7.—ACIDOSIS, NINE CASES; TWENTY-FOUR DETERMINATIONS

	Blood					
	Acid		Alkali		Total	
	Extremes	Averages	Extremes	Averages	Extremes	Averages
Entire group (9 cases; 24 determinations)	[illegible]	0.08	[illegible]	[illegible]	[illegible]	0.17
During stage of true acidosis ([illegible] cases; 16 determinations)	[illegible]	0.075	[illegible]	0.059	[illegible]	[illegible]
During stage of compensation (6 cases; 8 determinations)	[illegible]	0.1	[illegible]	0.1	[illegible]	[illegible]

TABLE 8.—SUMMARY OF ACID, ALKALI AND TOTAL BUFFER VALUES FOR THE VARIOUS CASE GROUPS EXPRESSED AS GROUP AVERAGES

		Blood		
		Acid	Alkali	Total
Normal individuals		0.18	0.18	[illegible]
Miscellaneous cases with normal pH, showing normal buffer values		[illegible]	0.17	[illegible]
Cases with normal pH showing diminished buffer values		0.19	0.06	0.2[illegible]
Acidosis	True	0.077	0.050	0.1[illegible]
	Compensated	0.10	0.10	0.2[illegible]

4. *Acidosis.* (Tables 6 and 7.) From the tables it is evident that cases of acidosis have less buffer for both acid and alkali during the stage of uncompensated acidosis (increased pH of the blood) than during the stage in which the tension of alveolar carbon dioxid is diminished, but the pH of the blood is normal (stage of compensation). Despite the return to normal of pH and buffer values, the clinical evidences of acidosis may persist (Cases 4 and 8).

EXPERIMENTAL CONSIDERATIONS

Inasmuch as the buffer value of blood is fairly constant in health, and decreased in acidosis, the question obviously arises as to whether buffer can be supplied. The possibility of accomplishing this with