was of a lower order of magnitude than that encountered in pantothenic acid, pyridoxine (1), and pteroylglutamic acid deficiencies. It seems possible that the increased susceptibility of vit. A-deficient animals to disease processes may be attributed in part to their decreased antibody-synthesizing ability.

With respect to vit. D-deficiency, it is generally agreed that rachitic animals are no more susceptible to infection than controls receiving adequate doses of this vitamin(15). In addition, studies(20,21) of the ability of rachitic animals to produce antibodies following antigen injection have indicated that there is no impairment of this function. In agreement with these observations we found no significant difference between antibody levels of vit. D-deficient rats and those of control animals.

The experiments reported in this paper complete an extensive study of the effect of the following deficiency states upon the antibody response of the albino rats (Sprague-Dawley strain) to the antigenic stimulus of Group O, Rh positive human erythrocytes: pantothenic acid, pyridoxine, riboflavin(1), biotin, thiamine(2), pteroylglutamic acid, niacin-tryptophan, vit. B_{12}, vit. A, and vit. D. Identical immunological procedures were employed throughout these investigations. From our results it is possible to classify roughly the effects of these deficiencies upon circulating antibodies into 3 groups as follows: Group I. Severe impairment of antibody response—pantothenic acid, pyridoxine, and pteroylglutamic acid deficiencies. Group II. Moderate impairment of antibody response—riboflavin, thiamine, biotin, vit. A, and niacin-tryptophan deficiencies. Group III. No impairment of antibody response—vit. B_{12}, and vit. D deficiencies.

Summary. 1. Hemagglutinin production in response to inoculation with human erythrocytes has been investigated in pteroylglutamic acid, niacin-tryptophan, vit. B_{12}, vit. A, vit. D-deficient rats and their respective controls. 2. Severe impairment of antibody response was observed in the pteroylglutamic acid-deficient rats. Moderate impairment was demonstrated in vit. A and niacin-tryptophan deficiencies. Vit. B_{12} and vit. D deficiencies had no effect on antibody formation. 3. A rough classification of the effect of 10 deficiency states upon antibody production is presented.

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Relative Effectiveness of Pantothenic Acid and Panthenol in Stimulating Antibody Response of Pantothenic Acid-Deficient Rats.* (18837)

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Panthenol, (α,γ-dihydroxy-N-(-3-hydroxypropyl)-β,β-dimethyl butyramide) the alcohol analogue of pantothenic acid, was demonstrated by Pfaltz(1) to be approximately as effective as pantothenic acid in enhancing growth and curing pantothenic acid deficiency symptoms of the rat. Later Burlet(2) showed that panthenol was oxidatively converted to

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2. Burlet, E., *Ibid.,* 1944, v14, 318.

TABLE I. Relative Effectiveness of Pantothenic Acid and Panthenol in Stimulating Antibody Response of Pantothenic Acid Deficient Rats.

Pantothenic acid deficient Wt (g)			Pantothenic acid controls Wt (g)			Panthenol controls Wt (g)		
Initial	Final*	Titer	Initial	Final*	Titer	Initial	Final*	Titer
39	85	0	47	261	1:5120	46	247	1:5120
44	74	0	47	257	5120	48	278	2560
48	115	0	46	260	5120	47	278	2560
42	78	0	48	288	5120	46	247	5120
40	78	0	44	262	5120	44	248	5120
41	85	0	48	287	2560	47	255	5120
44	74	0	44	267	5120	45	256	5120
41	110	0	46	270	2560	48	284	5120
44	73	0	46	260	5120	46	254	2560
36	61	0	45	261	2560	44	246	5120
40	77	0						
41	83	0						
34	79	0						
41	89	0						
37	58	0						
47	78	0						
Avg 41	81	0	46	267	1:4352	46	259	1:4352

* At time of bleeding.

pantothenic acid in both rats and humans. Recently, Rubin and his co-workers(3,4) on the basis of urinary excretion studies have concluded that panthenol is more available to rats and humans than calcium pantothenate when fed at high dosage levels. On the other hand at the lowest level tested, the availability of the two substances was found to be essentially the same.

Since in the course of our studies on the relationship of nutrition to immune response, we have occasionally noted that the process of antibody synthesis is a more sensitive criterion of dietary adequacy than that of growth or symptomology(5), it seemed of interest to determine whether panthenol is as effective as calcium pantothenate in preventing the observed suppression of circulating antibodies in pantothenic acid-deficient rats(6,7).

Experimental. Thirty-six male weanling albino rats were distributed into 3 groups as indicated in Table I. Except for the omission of sulfasuxidine, the basal diet was identical with that employed in the preceding paper(5) for the production of a pteroylglutamic acid deficiency. All animals received a daily vitamin pill supplying the following vitamins: thiamine, 40 γ; riboflavin, 60 γ; pyridoxine, 50 γ; niacin, 100 γ; biotin, 1 γ; and pteroylglutamic acid, 1 γ. The pantothenic acid-deficient rats received no further vitamin supplement while the pantothenic acid and panthenol controls received a daily intraperitoneal injection of 100 γ of calcium pantothenate and a molar equivalent dosage of panthenol (86 γ), respectively. All animals were fed the basal diet *ad libitum*. Immunization was instituted during the 5th week of the experiment. The antigen, immunization procedure, and the determination of serum antibody titers were identical with those described in the preceding paper(5).

Results. The data obtained are presented in Table I where it may be seen that panthenol was equally as effective as pantothenic acid in promoting both normal growth and attainment of a high serum antibody concentration. These results are in accord with previous studies by other workers(1,2,4) demonstrating the quantitative conversion of panthenol into pantothenic acid by the rat.

3. Rubin, S. H., Cooperman, J. M., Moore, M. E., and Scheiner, J., *J. Nutrition*, 1948, v35, 499.

4. Rubin, S. H., Drekter, M. E., and Pankopf, R., *Ibid.*, 1950, v40, 265.

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Summary. 1. Panthenol was found to be equally as effective as pantothenic acid in promoting antibody synthesis in pantothenic acid-deficient rats.

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Experimental Production of Post-Tonsillectomy Bulbar Poliomyelitis.* (18838)

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Poliomyelitis occurring within 30 days after tonsillectomy is predominantly bulbar(1). When compared with the incidence of the bulbar form in patients who have not had the operation within the same period of time, the difference is greater than can be explained on the basis of pure chance and indicates a causal relationship between operation and bulbar localization. This is particularly true of children, as shown by the two examples from the literature given in Table I.

In human bulbar poliomyelitis, including the post-tonsillectomy cases, the muscles of deglutition and phonation, supplied by the X cranial nerve and the nucleus ambiguus, are practically always involved, the facial muscles (N.VII) in about half, and those supplied by the V and XII nerves in lesser numbers(4). The nuclei of supply in all these are in the pons or medulla. Post-tonsillectomy bulbar poliomyelitis occurs almost exclusively during epidemics of the disease, and the onset of symptoms according to Aycock's(1) compilation nearly always occurs 6-24 days after operation, with the peak at 14-16 days. These figures agree closely with the incubation period of poliomyelitis in general, suggesting that virus may be introduced at the time of operation. Only rare cases have been reported in which the onset was less than 4 days after operation. Previous attempts(5-7) to reproduce experimentally the sequence of bulbar paralysis after tonsillectomy have nearly always failed, paralysis when produced usually having been spinal in type. In most of these studies, notably that of von Magnus and Melnick(7) exposure of the throat to the virus has been made *after* rather than before operation. Unpublished observations in our laboratory have shown that in order consistently to produce infection in divided nerve it is necessary to apply virus to the central cut end before it is sealed off by blood.

In the present study we have explored two hypotheses: (1) that virus already in the pharynx is directly introduced into the peritonsillar musculature, which is unavoidably traumatized during the human operation, and

TABLE I. Bulbar Poliomyelitis in Poliomyelitis Cases With and Without Recent Tonsillectomy.

	Poliomyelitis			
	Bulbar	Other forms	% bulbar	P*
A.(2) 0-10 years				
Tonsillectomy	24	12	66.7	
No tonsillectomy	771	6063	11.6	<.001
B.(3) 3-7 years				
Tonsillectomy	12	4	75	
No tonsillectomy	96	395	19.5	<.001

* From chi square of differences between bulbar and other forms. In both series P indicates that probability of differences occurring by chance are less than 1 in 1000.

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