

nolic -OH groups) as compared to the weaker hydrogen bond-forming power of the androgenic hydrogen (e.g. secondary alcoholic hydrogen).

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Effect of Altitude Anoxia in Provoking Relapse in Malaria

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The opinion is widely held that anoxia tends to precipitate relapse in individuals with latent malaria (1). This view, founded on clinical observation over a period of many years, received additional support during World War II when, with the increase in both the incidence of malaria and air travel, relapses of malaria were observed to occur following aerial flight. Recently, Gajewski and Tatum (2), studying the phenomenon of relapse in avian malaria, succeeded in inducing relapse in from 2 to 7 days in all of more than 100 canaries with latent *P. cathermerium* infection by exposing the birds continuously to an oxygen tension of approximately 75 mm. Hg.

In the present study an attempt was made to induce relapse in human subjects using a short, yet moderately severe, anoxic assault such as might occur during high-altitude flight. Fifty overseas returnees from various Army Air Force installations who gave histories of two or more recent attacks of malaria were the subjects. They were exposed for 1 hour in a low-pressure chamber to a simulated altitude of 18,000 feet without supplementary oxygen (oxygen tension, approximately 80 mm. Hg.).

Since the subjects' statements were the sole source of information in obtaining data as to the number, circumstances, and dates of their previous attacks, it

was not possible in all cases to separate reinfections from relapses; therefore, only the total number of previous attacks was recorded in each case. While the subjects were at the simulated altitude, continuous oximeter readings of oxyhemoglobin concentration in the blood were made. Thick blood smears were made immediately before and immediately after the chamber flight, and daily thick blood smears were made for 5 days thereafter. The subjects remained in the hospital under observation for a minimum of 6 days following the chamber flight, and at the time of hospital discharge were instructed to report subsequent relapses. All subjects, with the exception of one individual who had finished quinine treatment of his last relapse only 3 days before the chamber run, had discontinued atabrine or quinine administration 15 days or more prior to the anoxic episode.

The mean oximeter reading of the group was 76.4 per cent. None of the 50 subjects experienced relapses within 7 days of the chamber flight. Eight of the subjects, however, had relapses at periods varying from 8 to 35 days following the flight. None of the subjects included in the series had positive blood smears before entering the chamber or during the succeeding 5-day period when daily blood examinations were made. Data pertaining to the 8 subjects who relapsed subsequent to the 7-day period are shown in Table 1.

TABLE 1
DATA ON 8 CASES WHICH RELAPSED LATER THAN 7 DAYS
AFTER THE ANOXIC EXPERIENCE

Case No.	No. of previous attacks	Days since last attack	Days since last atabrine	Relapses		
				Days after chamber flight	Type	Days after last attack
3	7	25	17	8	Vivax	33
6	2	36	31	8	..*	42
8	6	12	3 (quinine)	23	..*	33
13	4	33	17	18	Vivax	51
42	9	26	15	30	..*	56
43	2	26	18	35	Vivax	61
44	5	52	46	17	Vivax	69
49	7	42	35	18	Vivax	60

* Type not reported.

In summary, neither relapse nor parasitemia was observed in a group of 50 individuals giving histories of recent malaria within a period of 7 days following exposure to the anoxia produced by a 1-hour stay at 18,000 feet in a low-pressure chamber.

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