there was a rapid nuclear uptake by mononuclear leukocytes. 4. DCVC did not inhibit incorporation of H^3-thymidine in either blood from DCVC-treated calves or in normal blood incubated in presence of DCVC. 5. Abolishment of DNA synthesis in leukocytes or leukopoietic tissue does not appear to be a primary biochemical lesion through which DCVC induces fatal blood dyscrasia in calves.

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Analgesia Enhancing Effect of Thalidomide. (25602)

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Since it has been reported that addition of thalidomide* (N-phthalyl-glutamic acid imide) to the classical "APC" combination enhanced its clinical analgesic effect(1), it was considered interesting to study this effect experimentally on human subjects.

Method. The amount of electricity necessary to elicit a painful sensation was determined 3 times at 15 minute intervals before, and 9 times at 20 minutes after oral medication. The current was applied as square waves of 1 msec. duration every half second. The stimulus was increased by 0.01 volt at every impulse until the subjects indicated feeling pain. Temporal clues during this procedure were avoided. The method has been described(2,3). Each of the 10 subjects served for 5 experimental sessions, with at least 48 hours intervening between any 2. The sequence of medication was by "latin-square" design and "double-blind" discipline prevailed. The medications, prepared to look alike were: A. 2 APC, = 7 gr. acetylsalicylic acid, 5 gr. acetophenetidin, 1 gr. caffeine; B. 25 mg thalidomide + 2 APC; C. Placebo; D. 32 mg Codeine sulfate + 2 APC; E. "Dry-run" (no capsules).

Results. For analysis, each postmedication threshold was expressed as its increment from its average premedication voltage on the same day. The average threshold increments of the 10 subjects, by interval after and type of treatment, are presented in Table I.

An analysis of variance, performed on the detailed aggregated data (of which Table I is a summary), is summarized in Table II. This analysis compares the net (or average or areas under the curve) increments following each treatment, in terms of variance attributable to subject-treatment interaction. No significant difference was found between the average

* "Kevadon", Trademark of Wm. S. Merrell Co.

TABLE I. Average Increments of Experimental Pain Thresholds of 10 Subjects (in Centivolts), by Time Postmedication and Treatment.

Min. after medication	Treatment A	B	C	D	E
20	6.0	.9	.2	3.9	.3
40	7.2	9.1	2.9	7.3	1.1
60	5.7	12.0	2.6	10.2	1.2
80	8.4	10.9	2.8	10.8	1.4
100	6.7	7.0	4.6	8.2	−.2
120	5.1	8.2	2.0	8.7	−.1
140	3.5	3.5	3.3	6.9	.7
160	2.3	4.6	1.5	5.0	1.2
180	−.1	3.0	.6	2.9	1.8
Total	44.8	59.2	20.5	63.9	7.4

postmedication increments which followed the control treatments C and E.

No significant difference was found between the average increments which followed the 3 treatments: A, B, and D. However, increments of the 3 treatments (A, B, and D) were significantly ($P<.01$) greater than the controls (C and E).

TABLE II. Summary of Analysis of Variance.

Source	Degrees freedom	Mean square	"F" ratio
Subjects	9	333.39	2.22*
Treatments	(4)	(667.33)	4.44†
Controls (C, E)	1	95.34	.63
Treats. (A, B, D)	2	110.05	.73
Controls *vs* treats.	1	2353.86	15.67†
Subj. × treats.	36	150.20	2.84†
Intervals	8	168.70	5.09†
Subj. × intervals	72	33.14	.63
Treats. × "	32	36.04	.68
Residual	288	52.93	

* $P<.05$. † $P<.01$.

Since it is apparent in Table I that time-response characteristics were not the same after each treatment, a separate analysis of variance was performed on all the data available within each postmedication interval.

The results of these comparisons of particular interest here are those which relate the increments after one compound to another. The only statistically significant differences obtained ($P < .05$-$.01$) indicated that the threshold was elevated above the dry run by APC + thalidomide (B) from 40 through 120 minutes after ingestion and by APC + Codeine (D) from 60 through 140 minutes postmedication. Increments after these 2 combinations (B and D) were significantly superior to placebo (C) induced increments at the 60 and 80 minute observations.

Conclusion. Because 2 APC + 25 mg thalidomide or + 32 mg Codeine sulfate elevated the threshold to experimentally induced pain of 10 human subjects above threshold increments which followed placebos or no treatment while 2 APC alone did not, it is concluded that the analgesic potential of APC is enhanced by added compounds in the quantities employed.

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Survival of Dogs Following Section of Carotid and Vertebral Arteries.* (25603)

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The functional effects following acute arrest of the carotid and vertebral arterial blood supply to the brain aroused the interest of several investigators during 19th and part of present century(1-7). Effects of deprivation of blood flow by these routes have been observed in animals, including horse, dog, cat, rabbit, monkey and goat(2,8,9). With few exceptions(1,2), these experiments were acute in nature, the animal either succumbing, or sacrificed soon after its termination. The

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