The results, as expected, showed uniform and complete removal of free phosphorus when using the powder and paste; but when using the solutions, results were variable, and removal was often incomplete.

Proposals for the Treatment of Phosphorus Burns

As a result of the foregoing considerations we put forward the following rationale of treatment:

Stage I.—Remove the bulk of the free phosphorus and superficial acid with alkaline powder (Formula 2A). Under no circumstances must liquid suspected of containing phosphorus be dabbed with a handkerchief or other dry material. If water has been used to extinguish the phosphorus, sprinkle the powder on to the wet surface, add more water if necessary, and work up to a medium thick paste, covering the whole of the burn and the surrounding area if contamination is suspected. Wash or wipe off and reapply so long as marked effervescence continues. Drying must be avoided at this stage. The advantages claimed over the corresponding stage in the official treatment are: portability; greater concentration and osmotic effect (drawing acid products of combustion from the wound); and the prevention of displacement of unignited phosphorus to other parts of the body or clothes.

Stage II.—Immobilize the whole of the remaining free phosphorus by the use of glycerin-copper sulphate paste (Formula 3 or 4). Apply sparingly but thoroughly, and rub well in. Remove by washing or wiping as soon as no smell of phosphorus is perceptible in the wound. The advantages claimed over the same stage in the official treatment are: portability, concentration, rapidity, osmotic effect, and completeness of action.

Stage III.—Neutralize the deep-seated penetration of acid with alkaline powder (Formula 2A). Reapply the powder, work up to a paste with water, bandage, and dampen bandage occasionally. Do not allow bandages to dry. The importance of this stage has apparently not been previously recognized.

Stage IV.—Remove to hospital labelled "Phosphorus Burn."

Regarding subsequent hospital treatment we would suggest that all forms of coagulation therapy (tannic acid, picric acid, silver nitrate, etc.) be avoided in view of the obvious necessity for not "sealing in" the acid products. The very painful nature of phosphorus burns and the tendency to delayed healing may possibly result from the inadequate treatment of this acid concentration, and the alkalinity of the tissues may not be sufficient to deal with it. It is suggested, therefore, that continuation treatment should be alkaline in character, and might consist ideally of the envelope treatment with sodium hypochlorite solution.

Summary

Serious weaknesses of the official treatment of phosphorus burns are pointed out.

A rational first-aid treatment is described which can be issued in a simple portable form suitable for inclusion as standard equipment for use in the field.

Continuation treatment in hospital is also discussed.

We acknowledge with thanks the facilities generously provided by the Crookes Laboratories, which have made this investigation possible.

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THE RELATIVE NEEDS OF YOUNG MALE AND FEMALE RATS FOR VITAMIN A

BY

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It has been shown (Coward *et al.*, 1931) that if young rats were given a diet deficient in vitamin A until their reserves of that factor had been exhausted, and were then given relatively small doses of vitamin A, the response of the male rats (increase in weight in five weeks) was less than that of the female rats. When the dose of vitamin A given was rather larger the responses of the two sexes were equal, and when larger still the response of the male rats was greater than that of the female rats. This was depicted graphically by drawing the curves of response of male and female rats respectively to five graded doses of cod-liver oil, the average increase in weight of each group of rats being plotted against the logarithm to base 10 of the dose of oil given. The curve of response of the male rats was steeper than that of the female rats, and the two lines crossed at the point where the increase in weight was 13.7 gm. and the daily dose of the sample of cod-liver oil given was about 1.2 mg. (equal to about 1.8 international units of vitamin A, as determined later). The smaller response of the male rats to low doses of vitamin A indicated that the male rat was more sensitive to a shortage of this factor than was the female rat.

All the tests of low doses of vitamin A which had up to that time been carried out in our laboratory were then examined for the number of male and female rats that had died in each group, every member of any one group having received the same dose of cod-liver oil. It was found that, in 23 groups, 57 out of 288 male rats and 37 out of 277 female rats had died. Dr. J. H. Gaddum supplied a calculation based on the numbers of groups in which more males than females and more females than males, respectively, had died. The numbers were 17 and 5, and 1 in which the mortalities of male and female rats were equal. His value of χ^2 was 6.26. The table of χ^2 shows that the probability of this value of χ^2 being due to chance is between 1/50 and 1/100 (Fisher and Yates, 1938). Hence it was concluded that there was a strong probability that young male rats are more sensitive to a shortage of vitamin A than young female rats.

Since the publication of that paper many more determinations of vitamin A have been carried out in this laboratory by the same method, and it seemed worth while to examine some of our recent work to obtain more information on this point. It happened that we had used a particular sample of cod-liver oil as a standard of reference for determining the vitamin A (carotene) content of a large number of fruits and vegetables, and the international standard of reference (carotene) for determining the vitamin A content of a large number of substances containing vitamin A of animal origin. Thus it was possible not only to obtain the further information required on the relative needs of male and female rats for vitamin A but also, by separating the groups given vitamin A of animal origin from those given vitamin A (β -carotene) of vegetable origin, to see whether this difference in response between male and female rats applied to both sources of the vitamin.

Calculations of tests which fell naturally into four groups have been made. They are summarized in Table I and also put out separately, for several results all supporting the same conclusion are often more convincing than one total result.

M. Josephi, J. Mehlman, and D. Hager (*J. Pediat.*, 1941, **19**, 823) record a case of influenzal meningitis in a male infant aged 7 months who made an uncomplicated recovery after treatment with large doses of sulphapyridine and a minimal amount of serum. Culture of the spinal fluid showed *B. influenzae*.

TABLE I.—*Relative Needs of Male and Female Rats for Vitamin A*

Tests	All Groups			Groups given Vitamin A of Animal Origin			Groups given Vitamin A (β -carotene) of Vegetable Origin		
	No. of Groups in which died:			No. of Groups in which died:			No. of Groups in which died:		
	More Males	More Females	χ^2	More Males	More Females	χ^2	More Males	More Females	χ^2
Fruits and vegetables from Ceylon compared with a sample of cod-liver oil (Z) as standard	78.0	46.0	8.26	29.5	16.5	3.67	48.5	29.5	4.63
Routine tests carried out under war conditions, mostly vegetable products compared with inter. standard	27.5	7.5	11.43	9.0	3.0	3.0	18.5	4.5	9.14
Research tests carried out under war conditions, mostly vegetable products compared with inter. standard	57.0	7.0	39.19	12.0	1.0	8.31	45.0	6.0	29.82
The last 200 groups in tests carried out before the war, mostly animal products compared with inter. standard	137.5	62.5	28.13	91.5	41.5	18.80	47.0	20.0	10.88
For all tests	300.0	123.0	74.06	142.0	62.0	31.37	159.0	60.0	44.75

The significance of the values of χ^2 found above may be judged from the following figures quoted from the χ^2 table. The probability that the difference between males and females in their need for vitamin A is due to chance is 0.10 when $\chi^2=2.706$; 0.05 when $\chi^2=3.841$; 0.02 when $\chi^2=5.412$; 0.01 when $\chi^2=6.635$; 0.001 when $\chi^2=10.027$.

A probability of 0.10 (or more) of the result being due to chance is generally considered a pretty good one, and no

TABLE II.—*Deaths under 1 Year per 1,000 Live Births*

Period	Deaths		Ratio M/F	Period	Deaths		Ratio M/F
	Males	Females			Males	Females	
1891	164	133	1.23	1915	123	96	1.28
2	162	132	1.23	6	102	80	1.28
3	173	143	1.21	7	108	85	1.27
4	150	124	1.21	8	108	86	1.26
5	176	144	1.22	9	100	78	1.28
6	161	134	1.20	1920	90	69	1.30
7	170	141	1.21	1	93	72	1.25
8	175	145	1.21	2	87	66	1.32
9	177	148	1.20	3	78	60	1.30
1900	169	139	1.22	4	85	65	1.31
1	166	136	1.22	5	84	66	1.27
2	147	118	1.25	6	79	61	1.30
3	145	118	1.23	7	79	60	1.32
4	159	131	1.21	8	74	56	1.32
5	141	115	1.23	9	83	65	1.28
6	145	120	1.21	1930	68	51	1.33
7	130	104	1.25	1	75	57	1.32
8	133	107	1.24	2	73	56	1.30
9	120	97	1.24	3	72	55	1.31
1910	116	94	1.23	4	65	51	1.28
1	142	117	1.21	5	64	50	1.28
2	106	84	1.26	6	66	50	1.32
3	120	96	1.25	7	65	50	1.30
4	116	93	1.25	8	60	46	1.30

significance would be attached to a result giving this probability. A probability of 0.01 to 0.05 of the result being due to chance is not good, and the result would be accepted as fairly dependable. A probability of 0.001 of the result being due to chance is almost *nil*, and the result would be accepted as almost certainty. Most of our separate values are highly significant, and the values of the totals leave no room at all for doubt of the fact that young male rats are more sensitive to a shortage of vitamin A than young female rats. The further analysis of the results shows also the same difference between male and female behaviour whether the vitamin A is of animal or of vegetable origin. Hence young male and female rats seem to have equal ability to convert carotene into vitamin A.

It is tempting to speculate on the possibility that a low intake of vitamin A by human babies may have a greater influence on male infants than on female infants in their mortality from, say, 0 to 1 year of age. The numbers of deaths under 1 year of age per 1,000 live births of male and female infants published by the Registrar-General are shown in Table II together with the ratio male/female deaths for each year from 1891 to 1938. The numbers of deaths per 1,000 live births have fallen dramatically in that time for both males and females, and this fall must have

been due to the influence of a very great many factors. One point, however, is of particular interest: the ratio of male/female deaths, already high in the 1890's, rose steadily from about 1900 to 1920, since when it has remained nearly constant. Assuming that all forms of extra care lavished on infants in the last fifty years have been given to male and female infants equally, is it possible (a) that the extra cod-liver oil given since prenatal and child clinics have been operating has been sufficient to bring about some improvement only, which, according to the rats, would be greater in the females than in the males; and (b) that by giving abundance of vitamin A, which would mean more cod-liver oil for boys than girls, the ratio of male/female deaths would be reduced to unity?

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PUERPERAL CEREBRAL THROMBOPHLEBITIS TREATED BY HEPARIN

BY

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Thrombophlebitis of the cerebral veins and sinuses has been recognized for many years as a rare and fatal complication of the puerperium. Von Hösslin (1904, 1905) reported a large series of cases in which the nervous system was involved during pregnancy and the puerperal state: these included five fatal cases of cerebral thrombophlebitis. Subsequently, no doubt, other examples of the condition have been recorded as cases of "late eclampsia," but interest seems to have waned until the publications of Symonds (1940), Martin (1941), and Martin and Sheehan (1941). In particular, Symonds points out that it is possible to diagnose the condition during the life of the patient, and says that, in his opinion, the prognosis is not so gloomy as a perusal of the literature would suggest.

Aetiology

The thrombosis has been termed "primary" in Gowers's sense, in that obviously it does not arise out of a local infective condition, but is rather the result of an increased coagulability of the blood. Fresh light has been shed on the problem by the experimental work of Batson (1940), who demonstrated that it was possible for emboli to pass from the pelvic veins via the vertebral venous plexuses to the cerebral veins. The factor favouring this condition was a rise in the pressure in the inferior vena cava.