having taken her blood pressure as soon as the purpuric rash appeared. There was no corresponding rash on the opposite forearm or any sign of bleeding elsewhere. The pregnancy appeared to be normal; the vertex was presenting and the head was still free. Blood pressure was 110/70, and no albuminuria was found.

The patient was brought into hospital, where she was seen in consultation with Professor Stanley Alstead, under whose supervision full blood investigation was carried out. The most significant finding here was a platelet count of 101,500 per c.mm.-i.e., about 40% of the accepted minimum standard. There were some red blood cells in the urine, singly and in small clumps, and clotting time was prolonged to 14 minutes. In a few days, however, without any specific treatment, the rash disappeared and no new ecchymoses were observed. The pregnancy seemed to be progressing normally. There was no evidence of toxaemia, so it was decided to await events. She was discharged from hospital and returned to her home.

A week before term full blood investigation was repeated and appeared to be normal in all respects. The platelet count was 562,320 per c.mm. and the clotting time 6 minutes 45 seconds. The foetal head was by this time engaged in the pelvis, and the pregnancy seemed to be normal. As a precautionary measure it was decided that she should have a small transfusion of fresh blood at the onset of labour and that vitamin K should be given at the same time. A week later there was a small "show," and a slow transfusion of 250 ml. of Group IV blood was given together with vitamin K ("kapilon," 4 ml.). A few hours after this labour began; it was completed in about three hours with the delivery of a live female child weighing 8 lb. Immediately after the birth of the shoulders ergometrine, 0.5 mg., was given intravenously and the placenta and membranes were delivered complete without any apparent haemorrhage. There were no lacerations and the uterus contracted well. After the completion of labour bleeding was normal in amount and gave rise to no anxiety. The puerperium was uneventful, and on the fifth day the platelet count was found to be 340,000, the other blood findings also being normal. The child made good progress, being fully breast-fed, and did not seem to be affected by purpura, its platelet count on the twelfth day being 346,000. Both mother and child were discharged home in good condition on the 14th day, and subsequent progress has been uneventful.

## Discussion

In 63 previously reported cases of purpura haemorrhagica complicating pregnancy the mortality in both mother and foetus has been alarmingly high, the most recently recorded figure for the former being 55%. The commonest cause of death is prolonged haemorrhage after the completion of the third stage. The case quoted above was mild and transient. Special precautions were taken before and after labour; no undue haemorrhage was observed and the puerperium was normal in all respects. The infant was unaffected. This case confirms Patterson's opinion that there are two different types of case-namely, the chronic, which is associated with all the dangers giving rise to high mortality, and the acute, in which the abnormality may pass off before labour begins and there is no particular danger. The prognosis is good in the acute case unless the acute phase and the onset of labour should unfortunately coincide, but it is not possible to give a prognosis regarding recurrence in a subsequent pregnancy. In view of the very small number of cases recorded it is unlikely that recurrence is common.

### Summary

Purpura haemorrhagica is a rare complication of pregnancy. The literature, in which there are records of 63 cases, has been reviewed. A further case is now added.

In the case recorded the purpura was acute and transient, and labour was unattended by any complication.

Treatment should be directed to restoring the blood picture to normal before the onset of labour. If this is impossible special measures for the control of post-partum haemorrhage may be necessary.

The prognosis in the acute cases is good, but there is grave danger of maternal death in the chronic cases.

The foetus is commonly involved, and may die of haemorrhage or of prematurity associated with the early onset of labour in a considerable proportion of these cases.

I am indebted to Dr. H. J. R. Kirkpatrick, Dr. Elemer Forrai, and Dr. Edgar Moyes for the blood investigations in this case, and to Professor Stanley Alstead for his advice in the management of it.

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# POLIOMYELITIS EFFECT OF EXERTION DURING THE **PRE-PARALYTIC STAGE**

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A history of great physical exertion immediately preceding the onset of severe paralysis in attacks of poliomyelitis has often been recorded, but until recently no statistical evidence was available to determine whether such instances were more frequent than chance would dictate. Russell (1947) made a very careful statistical analysis of the degree of physical activity and resulting paralysis in 44 cases of poliomyelitis, and concluded: "Physical activity of any

TABLE	I.—Effect	of Physical	Activity	during	the Pre-Par	alytic Stage
on the	Severity o	of Paralysis i	n 30 Cas	es of Po	liomyelitis.	P indicates
		ons	et of Pa	ralysis		

	Age	Sex	Day After Onset of Pre-paralytic Symptoms						Paralysis	Residual Paralysis	
No.			D+1	D+2	D+3	D+4	D+5	D+6	D+7	on Admission	at 3 Months
1 2 4 5 6 10 11 15 16 17 18 10 21 22 23 26 27 28 31 35 36 37 38 41 42	$\begin{array}{c} 8\\ 33\\ 1\\ 7\\ 26\\ 3\\ 1\\ 4\\ 23\\ 7\\ 5\\ 34\\ 3\\ 12\\ 17\\ 2\\ 17\\ 1\\ 1\\ 4\\ 9\\ 28\\ 5\\ 5\\ 34\\ 35\\ 22\\ 30\\ 35\\ 21\\ \end{array}$	M F M F F F F F F F F M M M F M F M F F F F F M M F M M	iii * * *2 <sup>-</sup> N2 - <sup>-</sup> N1 - <sup>1</sup> N * *2 <sup>N</sup> N * *1 - <sup>1</sup> N1 <sup>*</sup> *3 2 - 3 3	iii 1 2 2 ii 2 P 3 1 1 ii " P ii " 1 2 ii 1 ii " 3 2 ii 2 3	221P21N  3P2NPN  NPPPN * PPN-13PP1	211   N 1 N   P   2P   N   N       N ::     P - P     N	P1P   PPP     2     2     1   1   PPN       1   1   P	P                 P   P       P       P         P         P         P         P             P	<b>P</b>	Сввссооскосовеквеввеесвеккокк	B A B B A B B C F B B C A D F A E A A B A C B A A F F C F F

Degree of physical activity.—Nil = in bed.  $1 = \text{not more than } \frac{1}{2}$  day light work (e.g., resting in house, with short walks). 2 = Average light work (e.g., secretarial, housework, school). 3 = Average or heavy manual work (e.g., factory, labourer, labourer, labourer, labourer. athletic sports).

athletic sports). Severity of paralysis.—A = No paralysis. B = No severe paralysis, probable recovery to full function. C = Moderate paralysis.—i.e., moderate multiple paralysis or severe paralysis of a few muscles in one limb. D = Bilateral severe paralysis at any level including trunk, or gross paralysis of one limb. E = Severe and extensive paralysis such as trunk and both lower limbs, or severe paralysis of all limbs. F = Fatal.

kind during the pre-paralytic stage increases the danger of severe paralysis. Complete physical rest in bed during the whole of the pre-paralytic stage seems to protect the patient (rom severe paralysis."

A similar analysis of cases of poliomyelitis occurring in Cornwall during 1947-8 is shown in Table I. The cases are selected in so far that only those giving a clear history of a pre-paralytic meningeal stage of the illness are included. The standards adopted for the criteria of meningeal involvement, the severity of physical activity, and the degree of paralysis are the same as used by Russell, differing only in that the degree of paralysis in my cases is estimated after three months' treatment instead of on admission. Two of the cases (Nos. 34 and 38) had travelled to Cornwall by road during the pre-paralytic stage of the illness. In both cases the patient had personally driven his car 250 miles or more.

TABLE II.—Showing Maximum Physical Activity in any one Day following First Meningeal Symptoms and Severity of Paralysis

Paralysis at	No. of	Physical Activity, Showing Maximum Activity in 24 Hours							
3 Months	Cases	Nil (Bed)	1 Slight	2 Moderate	3 Severe				
None (A) . Slight (B) . Moderate (C) . Severe (D) . Very severe (E) . Death (F) .	. 9 . 9 . 4 . 1 . 1 . 6	6 1 1 1 1 1		$\begin{array}{c} 2\\ 5\\ -\\ -\\ -\\ 1 \end{array}$					

Table II shows the maximum activity in a single day and the severity of the resultant paralysis. It will be noted that patients who took to their beds with the onset of initial symptoms proved to be relatively mild cases, six of the ten cases having no residual paralysis after three months of treatment, whereas severe activity in the pre-paralytic stage of the disease led to a very grave prognosis: five of the six fatal cases had undertaken severe mental or physical strain in the days immediately before the onset of paralysis.

 TABLE III.—Showing Aggregate Physical Activity in Pre-paralytic Stage and Severity of Paralysis

Paralysis at 3 Months		No. of Cases	Physical Activity-Total Incidence								
			0	1	2	3	4	5	6	7	8
None Slight Moderate Severe Very severe Death	(A) (B) (C) (D) (E) (F)	9 9 4 1 1 6	6 1 1 1 1 -		$\frac{\overline{3}}{1}$		2 	1   1		 	

Table III shows the degree of paralysis plotted against the aggregate of physical activity during the pre-paralytic period. It will be seen that the maximum exertion in any one day is a more important indication of the prognosis than the aggregate of physical activity during the period.

## Summary

The effect of physical or mental exertion during the preparalytic stage of poliomyelitis on the ultimate prognosis is analysed in thirty cases of the disease which occurred in Cornwall during 1947-8.

The findings support those of Russell (1947)—namely, that severe physical activity during the pre-paralytic stage is associated with grave prognosis. Severe mental strain, such as driving a car over long distances, appears to be equally disastrous, whereas paralysis tends to be mild in cases confined to bed during the pre-paralytic stage.

I wish to thank Dr. J. G. M. Molony for granting me facilities to examine these cases during their stay in the County Isolation Hospital.

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# Medical Memoranda

## A Case of Tetanus Treated with *d*-Tubocurarine Chloride

The use of curare in tetanus was described by Hunter (1878) and later by Hale-White (1901). More recently Florey, Harding, and Fildes (1934), Cole (1934), Mitchell (1935), West (1936), Isacson and Swenson (1941), and Cullen and Quinn (1943), using various preparations of curare, were of the opinion that beneficial effects were produced by the use of the drug in this disease. It is felt that the following case history may be of interest now that a derivative of curare (*d*-tubocurarine chloride) is readily available.

#### CASE HISTORY

A lad aged 16 was admitted on April 23, 1948. Pain and stiffness in the lumbar muscles and jaw had started 36 hours previously. Owing to repeated injuries at football the incubation period of the disease is uncertain. On admission his temperature was  $97.2^{\circ}$  F. (36.2° C.), pulse 90, and respiration rate 24. His general condition was good and he weighed 8 stone (50.8 kg.). On his right shin there was a scabbed sore  $\frac{1}{2}$  in. (1.9 cm.) in diameter, together with a few scratches. Risus sardonicus and trismus were present, and he was unable to separate his incisor teeth more than half an inch (1.25 cm.). There was marked hypertonus of his spinal and abdominal muscles, with slight opisthotonos, and hypertonus of the muscles of his right leg.<sup>9</sup> He was given 240,000 units of antitetanic serum—200,000 units intravenously and the rest intramuscularly. On the day after admission the wound on his shin was excised.

For nine days from the time of admission the patient was kept more or less continuously under the influence of bromethol or paraldehyde. Bromethol per rectum was used for the first three days, an initial dose of 5.1 ml. of bromethol fluid being followed by a maintenance dose of between 2 and 3 ml. Following this, paraldehyde, in a 10% solution in saline, was given per rectum for six days in doses of 2 to 4 dr. (8-15 g.). The frequency of dosage with each drug (about every six hours) was governed by the patient's degree of somnolence. In all, 21.1 ml. of bromethol fluid and 57 dr. (200 g.) of paraldehyde were given.

From the start of treatment, in view of the anticipated prolonged sedation, 5% carbon dioxide inhalations for a few minutes, and coughing exercises, were given six-hourly.

During his first night in hospital, and again on the second day when his wound was being dealt with, the patient had a cramp-like exacerbation of his muscular spasm. By the third day his condition had deteriorated in spite of sedation. His body musculature was more rigid, and he was having a good deal of pain. He had a further exacerbation of spasm and was complaining of some difficulty in breathing.

It was then decided to try the effect of *d*-tubocurarine chloride. As we were uncertain of the degree of muscular paralysis which would be required to give relief, we brought to the ward a Drinker respirator and anaesthetic trolley with accessories. *d*-Tubocurarine chloride in solution was given slowly by the intravenous route, and after a dose of 8 mg. the patient's face relaxed, he remarked that his muscle pains were quite easier, there was some diminution in the spasm of his lumbar and abdominal muscles, and he was able to separate his incisor teeth 1 in. (2.5 cm.) compared with  $\frac{1}{3}$  in. (0.8 cm.) beforehand. Immediately afterwards a further 6 mg. of the drug was given by subcutaneous injection. After this we gave *d*-tubocurarine chloride in solution by intramuscular injection in doses ranging from 7.5 to 12.5 mg. every few hours, the frequency of administration being adjusted so as to control the pain and exacerbations of spasm. The patient received *d*-tubocurarine chloride for six and a half days at intervals of from two to six hours, the total amount given being 252 mg.

During this time he remained free from exacerbations of spasm and comparatively free from pain, except when the action of curare was wearing off. At these times severe interscapular, lumbar, and abdominal pain would recur, associated with increased hypertonus, cyanosis, and a rise in the pulse rate from about 80 to between 120 and 150 a minute.

By the ninth day in hospital the pain, spasm, and cramps were diminishing, and we stopped the administration of sedatives and curare. In the ensuing few days the residual stiffness wore off and he felt well and hungry. He lost 4 lb. (1.8 kg.) in weight during his illness.

On June 20 he wrote to us saying that he felt fit and was back at school.