loss of this area was not so well defined, and, in any case, there are known to be filamentous connexions between the greater and the lesser occipital nerves, the latter being derived from the anterior primary ramus of the same segment. Again, another possibility is direct involvement, since, as Wright and Brady (1958) point out, the greater occipital nerve rests on the vertebral arch of the axis, in a vulnerable position, unprotected by an intervertebral foramen.

Deep and internal to the swelling found in this case is the site of the superior cervical ganglion of the sympathetic system, lying on the lower part of the longus capitis muscle. It is suggested that pressure on this structure was responsible for Horner's syndrome, thus completing the clinical picture observed.

Remembering that the patient was breathing a proportion of carbon monoxide for a period of three hours, we are justified in assuming that there was a neurotoxic element at work lowering the threshold for neuronal damage for the more direct factors at work.

Summary

A case is described of an extensive cervical and brachial plexus injury arising as a result of linear pressure on the neck during a coma produced by coal-gas in the course of a suicidal attempt. It is noted that most of the literature on pressure palsies of the brachial plexus is concerned with cases sustained as an operative anaesthetic hazard.

An attempt is made to correlate the clinical findings with the regional anatomy. The posture and habitus of the patient appeared to be of contributory significance, as well as the mode of suicidal attempt.

I wish to thank Dr. Stanley Smith for permission to publish details of this case, and Dr. F. Hillman for the photograph.

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"The progress which the Elderly Invalids' Fund has made year by year was well maintained in 1959. A total of 135 invalids received financial assistance compared with 92 in 1958. As this increase in the number of patients could have been achieved simply by helping more temporary cases and fewer permanent ones, the better way of measuring the progress is in terms of patient weeks, which amounted to 2,086 in 1959 compared with 1,290 in 1958. This is equivalent to supporting 40 invalids throughout the year compared with 25 in 1958. Unfortunately, we cannot show a proportionate increase in our income, with the result that this increased work and commitments have caused us a deficit of £387 9s. 3d. . . . Expenditure during the year amounted to £5,205 compared with £4,850 in 1958. Income at £4,817 was £182 less than the previous year. . . . Attention can again be drawn to the low administrative costs—£115. There is still no paid staff and expenses are only for printing, stationery, and postage. These have cost more than in 1958, but with increasing work the use of more writing paper, envelopes, and stamps is inevitable." (*Elderly Invalids' Fund Annual Report, 1959*).

POST-MORTEM SERUM CHOLESTEROL LEVELS

BY

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It has been shown that there is a higher serum cholesterol associated with coronary artery disease (Oliver and Boyd, 1953; Dock, 1958), and its estimation is a recognized procedure in patients with this condition.

The purpose of this investigation was to discover if the level of the serum cholesterol altered after death. If it could be established that hours or even days after death the serum cholesterol had not altered appreciably from the level just before or at death it was thought that post-mortem estimation could be taken to indicate the serum cholesterol during life and could perhaps be used in the correlation of serum cholesterol levels and the state of the arteries as found at necropsy.

Method

Two preliminary observations were made. Firstly, blood taken from patients by venepuncture was divided into two parts and the serum cholesterol level estimated on one part while the second sample was left for four days in the specimen refrigerator at a temperature of 5° C., and a second cholesterol estimation was performed. No significant difference was found between the two results (Table I). Secondly, it had been

TABLE I

Specimen	Original Serum Cholesterol (mg./100 ml.)	Serum Cholesterol Level After 4 days (mg./100 ml.)
1	165	160
2	203	210
3	122	117
4	118	110
5	392	403
6	372	362
7	247	243
8	366	363
9	207	204
10	250	254
11	216	219
12	199	201

found that when samples of blood taken at post-mortem examination were cultured the majority in varying degrees grew *Escherichia coli* and "coliforms." Blood of known cholesterol content was therefore inoculated with these organisms and kept for the remainder of the time in the post-mortem room refrigerator at 5° C.

At 48 hours and after six days the cholesterol was again estimated, and there was found to be no significant change (Table II).

TABLE II

Specimen	Original Serum Cholesterol Level (mg./100 ml.)	48 Hours After Inoculation (mg./100 ml.)	6 Days After Inoculation (mg./100 ml.)
1	156	152	151
2	192	169	167
3	179	167	167
4	125	109	105
5	337	348	345
6	158	148	155
7	168	162	154
8	112	108	109
9	380	363	360
10	343	339	331
11	423	420	418
12	381	373	367

As the levels did not appreciably change the following procedures were undertaken. Blood samples were taken from very ill patients and the serum cholesterol levels estimated. If in any case death did not occur within 24 hours the blood was discarded and a fresh specimen taken. In one case blood was taken just after death.

Bodies were left at a temperature of $+5^{\circ}$ C. in the post-mortem room and at varying times blood samples were taken. This sampling presented no difficulty: a 20-ml. syringe and a 5-in. (12.5 cm.) No. 14 gauge needle was used to take the blood from a femoral vein or artery, from the heart by substernal puncture, or direct from the heart at necropsy. There was usually a great deal of haemolysis, but by long centrifugation enough serum was always obtained.

The estimations were performed by the colorimetric method of Sackett (1925).

The results (Table III) in 26 cases show that up to 60 hours after death there is no large fluctuation in the cholesterol estimation, and in two cases at 90 hours after death the level was quite near that found before death.

TABLE III

Age	Disease or Injury	Hours before Death Sample Taken	Cholesterol (mg./100 ml.)	Cholesterol (mg./100 ml.) Hours after Death			
				0-30	30-60	60-90	90+
21	Subarachnoid haemorrhage	18	187	190			
60	Crush injuries	19	139	137	134		
51	Cerebral haemorrhage	22	362	348			
56	Bronchopneumonia	23	90		91		
50	Subarachnoid haemorrhage	16	221		250		
74	Steatorrhea	12	37		48		
41	Chronic nephritis	12	113		112		
9	Influenza	1	82	81			
32	Cerebral haemorrhage with hypertension	8	231		214		
60	Neoplasm pancreas	12	89		80		
46	Subarachnoid haemorrhage	18	362	379			
28	Multiple injuries	1	146	148			
69	Influenza	7	208	218	187		195
39	Fractured skull	At death	116		125		
68	Pneumonia	"	53		58		
66	Influenza	"	76		76		
23	Carcinoma of bladder	At death	187				183
2	Carcinoma of prostate	2 hours after death	147		157		
68	Cerebral haemorrhage	8	234		241		
61	Pulmonary abscess	At death	42		46		
61	Cor pulmonale	"	88			96	
70	Mesenteric thrombosis	3	95		94		
38	Multiple injuries	1	110		108		
54	Cor pulmonale	9	83		86		
53	Cerebral haemorrhage	6	138	135	132		
60	Nephrotic nephritis	3	620	620			

Summary

The serum cholesterol was estimated on 26 patients shortly before and at varying times after death. The ante-mortem and post-mortem levels were close.

The post-mortem serum cholesterol may be taken as an indication of the level just before death.

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D

RAPID METHOD FOR ESTIMATION OF CARBON MONOXIDE IN BLOOD

BY

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A simple, rapid, and accurate method of measuring the carbon monoxide content of blood is required for investigation of subjects exposed to that gas. Analysis of alveolar gas which has reached equilibrium with the pulmonary capillary blood (Sjöstrand, 1948) is simple and accurate but time-consuming. This paper describes a rapid method based on Sjöstrand's method and compares its accuracy with the results of direct blood-gas analysis.

Method

The subjects were six healthy adults (five male and one female) aged 26 to 34 years, and the procedure was as follows:

Each subject washed out the nitrogen from his lungs by breathing 100% oxygen from a simple open circuit (Fig. 1) for a period of three minutes. At the end of this time he was instructed to take a maximum inspiration and hold his breath. The three-way tap (T) was then turned and he exhaled through a carbon dioxide absorber, previously washed out with oxygen, into an empty bag and rebreathed from this bag for a further three minutes. The contents of the bag were then analysed for oxygen by the Haldane method and for carbon monoxide, using an infra-red meter. From these figures the basal blood carboxyhaemoglobin saturation (COHb%) was calculated, using Dahlström's (1955) equation, namely:

$$\text{COHb}\% = \frac{M \times 100 \times p_{\text{CO}}}{p_{\text{O}_2} + (M \times p_{\text{CO}})}$$

where $M=231$ and p_{CO} and p_{O_2} are the partial pressures of carbon monoxide and oxygen in the equilibrated bag.

During the last minute of the rebreathing period 7 ml. of venous blood was withdrawn for the direct measurement of the carbon monoxide content of the blood by the method of Lawther and Apthorp (1955). The oxygen capacity of the blood of each subject was measured by the method of Van Slyke, and from these

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FIG. 1.—Breathing circuit. The mouthpiece (M) is fixed to a three-way tap (T) which can be turned so that the subject first washes out his lungs with oxygen and then rebreathes from the 5-litre bag through the CO_2 absorber.