research in general practice, and shortage of time and other factors make even simple clinical investigations difficult to undertake.

In recent years Mackintosh (1954), Fry (1958), and other writers have felt that co-operation between hospital out-patient departments and family doctors should make possible certain types of clinical investigation that would be difficult for either to accomplish alone.

One field in which a combined approach seems worth considering is the clinical assessment of some of the numerous new drugs now available. There are many difficulties in the way of a controlled clinical trial of a drug in general practice. Ethical considerations are involved, and our experience is that patients are more willing, and even keen, to participate in an experiment involving new remedies when it is carried out under the guidance of a hospital clinic with special experience in that field. On the other hand, it is desirable of most remedies that they should be effective and practicable for use in general practice, and it would be helpful if methods of assessment could be devised which were known to give similar results under hospital and general practice conditions.

It is perhaps unnecessary to emphasize that such investigations must be planned in detail in advance, and that a certain sacrifice of time by the participants is inevitable. Where subjective responses of patients are being assessed, or where objective changes of minor degree may be significant, current practice demands that bias of both patient and physician be minimized by the use of the double-blind technique.

This paper describes a simultaneous controlled clinical trial in the hospital out-patient department and in general practice of the effect of certain regimes of treatment for hypertension. On the whole the results of the two assessments show a remarkable agreement.

The tabulated blood-pressure readings were studied to find whether there was any general trend to higher readings in one or other assessment. There is no doubt that a single estimation of blood-pressure at a diagnostic consultation in hospital often gives a much higher reading than the usual level recorded by the family doctor. Yet in this investigation the blood-pressure readings during placebo therapy did not show any general tendency to higher readings at hospital.

In a few subjects there was a substantial and consistent discrepancy between the two sets of readings—for example, Case 5 showed much higher readings in hospital throughout the whole period, and Cases 8 and 15 the converse, particularly for systolic pressure. These facts, and the rather wide scatter of individual readings, indicate once more the need for a fairly large number of blood-pressure recordings in each phase of such an investigation. And it is clear that a smaller number of patients would be unlikely to give a conclusive result.

Simple investigations of this kind, involving close co-operation between hospital and family doctors, can be educative to both and may produce useful information.

Summary

A controlled clinical trial is described comparing reserpine with a smaller dose of reserpine plus rescinnamine in hypertension.

The results were assessed independently at the hospital clinic and by the patients' family doctors, working in co-operation.

Rescinnamine is unlikely to offer any useful advantage over reserpine in the treatment of hypertension.

Clinical investigations involving co-operation between hospital and family doctors have possibilities that should be explored further.

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MONOPLÉGIA AND HORNER'S SYNDROME FROM PRESSURE PALSY

REPORT OF A CASE AND ANATOMICAL DISCUSSION

BY

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Transient paralyses of the median and ulnar nerves sustained by prolonged pressure during narcotic-induced sleep, and particularly after bouts of acute alcoholism, are relatively commonly observed occurrences. Likewise, the condition of "Saturday night paralysis," involving the radial nerve in the upper part of the arm, is a well-known clinical entity. But, as Walshe (1958) points out, it is rare for the nerve of a healthy subject to sustain such pressure palsy, the majority of such patients being chronic alcoholics with an already debilitated peripheral nervous system.

The majority of brachial-plexus-pressure palsies reported in the literature are cases sustained during anaesthesia. Wood-Smith (1952) describes a lesion of C 5 and C 6 roots sustained during operation, and cases are also reported by Raffan (1950), Shaw (1953), and Westin (1954). The *Lancet* (1950) had a leading article on this subject; and, in particular, the hazards of the Trendelenburg position have been well covered. Ewing (1950) stressed the mechanism of abduction of the arm in the Trendelenburg position, causing traction injuries. Traction was incriminated also in the earlier series of Clausen (1942). Kiloh (1950) felt that both traction and pressure were of significance. But reports of cases arising apart from operative anaesthetic hazard are few and far between; the case of "pack palsy" reported by Bom (1953) in the Korean war is a rarity.

The occurrence, therefore, of an extensive lesion of the cervical and brachial plexus due to direct pressure

in the neck is of some rarity, as well as providing an interesting illustration of the anatomy of the region. Such a case is reported below.

Case Report

A married woman aged 39 was admitted to Lancaster Moor Hospital in December, 1959, after an attempt at suicide by coal-gas poisoning. The psychiatric history being largely irrelevant to the present consideration, it is not discussed in detail, but it is of interest to note that, in association with an anxiety-depression of some months' standing, the patient had anorexia, and had lost a good deal of weight, being only 6 st. 9 lb. (42.2 kg.) on admission. She was of slim build, and had a somewhat drooping shoulder-girdle. Details of the suicidal attempt were not available at the time of admission, but routine physical examination revealed an almost complete monoplegia involving the left arm; anaesthesia, partial or complete, involving the cutaneous distribution of all the cervical nerves, including the brachial plexus; and left Horner's syndrome. The detailed physical findings were as follows.

Motor Lesion.—Slight movement of the fingers and a little flexion at the wrist were possible, but no active movement of extension of the wrist or fingers, no pronation-supination movement, and no flexion-extension at the elbow. Little movement of the shoulder-joint itself could be made, but the shoulder-girdle could be raised by the trapezius; there was some activity in the pectorals, and the rhomboids and serratus anterior were intact. All the tendon reflexes of the upper limb were abolished.

Sensory Lesion.—There was diminished sensation to pain and touch over the back of the head, the side of the neck extending just over the angle of the jaw, and the whole of the arm. Sensation was unimpaired over the pectoral region, and the lines of demarcation along the angle of the jaw superiorly and the clavicular region inferiorly were clearly indicated. The demarcation in the midline of the neck was fairly distinct, but over the parietal and occipital scalp the borderlines of sensory impairment were not so precise. Subjective paraesthesiae described as "tingling numbness" were felt over the head and neck on the affected side, but the limb was described merely as "numb."

Autonomic Lesion.—There was meiosis of the left pupil, slight ptosis, and dryness of the skin. Enophthalmos was not apparent.

Local Examination of Neck.—This revealed a small, slightly tender, firm, immobile, but not very discrete swelling at the posterior border of the sternomastoid at about the mid-point, and apparently arising from the substance of, or from beneath, that structure.

Discussion with the patient and her husband revealed that she went to put the kettle on at 9.30 a.m. On an impulse she did not light the gas, and her husband found her at 12.30 p.m. in the gas-filled kitchen, slumped unconscious

with her neck over the hard back of a chair. He found her neck red and swollen along the line of contact. The subject in the accompanying illustration shows the position in which the patient was found.

By next day the sensory loss had almost entirely recovered, although some paraesthesiae were still complained of. Considerable motor recovery had occurred, the flexors and extensors of the wrist and fingers having regained full power, and there being some function of extension at the elbow. There was still, however, complete loss of power in the biceps and supinator, and in the deltoid, supraspinatus, and infraspinatus; and Horner's syndrome was unaltered.

Treatment was instituted with passive and active movements and galvanism. Further improvement was slower, and when the case was last reviewed there was still a complete palsy of the deltoid and spinati. The progress of the clinical picture is summarized in the Table.

Summary of Clinical Progress

	On Admission	Next Day	After 2 Weeks	After 5 Weeks
Deltoid	0	0	0	0
Supraspinatus	0	0	0	0
Infraspinatus	0	0	0	2
Biceps	0	0	3	5
Supinator	0	0	0	5
Triceps	0	4	4	5
Pectorals	2	4	4	5
Flexors of wrist and fingers	2	5	5	5
Extensors of wrist and fingers	0	5	5	5
Sensory impairment ..	Present	Doubtful	Absent	Absent
Horner's syndrome ..	"	Present	Not noted	"

The grading of muscle power numbered 0-5 is that recommended by the Medical Research Council: 0=Complete paralysis. 1=Flicker of voluntary action. 2=Sufficient to move joint. 3=Action against gravity. 4=Action against gravity and resistance. 5=Full power.

Discussion

In the position in which the lesion was sustained, the adducted left arm exaggerated the depression of the shoulder-girdle in this patient, who was already of asthenic "drooping" stature. A period of anorexia resulting in loss of weight meant that there was very little superficial fascia protecting the structures in the posterior triangle of the neck. These factors would approximate even more closely the crowding together of the three trunks of the brachial plexus before the ramification to form the cords occurs, and would tend to drag the trunks out from their protected site between the scalenus anterior and the scalenus medius. An injury at this site would explain the sparing of the longitudinal cervical musculature, the serratus anterior, rhomboids, and phrenic contribution (all derived from roots of the plexus) while involving the suprascapular (derived from the upper trunk of the plexus) and all the branches of the plexus below the trunks. The presence of the swelling at the posterior border of the sterno-mastoid overlying this point would suggest that this was the site of maximum injury.

Emerging more superficially, but still at approximately the same site at the mid-point of the posterior border of the sterno-mastoid, a spray of cutaneous nerves spreads out fanwise—the three supraclaviculars, the anterior cutaneous of neck, and the greater auricular. The lesser occipital is exposed a little higher, but in a linear lesion it is understandable how all of these might be affected. This area also corresponds to one of the "trigger points" illustrated by Reichert (1955), which may account for some of the initial diffuse paraesthesiae described.

It is not quite so easy to appreciate how the cutaneous sensation of the occiput comes to be involved, since this is supplied by the greater occipital nerve from the posterior primary ramus of C 2. However, the sensory

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