INFLUENCE OF INTRA-ARTERIAL HYDROCORTISONE ON ADRENERGIC RESPONSES IN THE HAND

BY

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The profound influence exerted by the adrenal cortex on vascular activity has been abundantly demonstrated in laboratory animals, and there are good clinical grounds for inferring a similar relationship in man. In hypertensive patients who subsequently develop Addison's disease or who are treated by bilateral adrenalectomy a fall in blood pressure has been noted (Perera, 1945; Thorn *et al.*, 1952; Arnott *et al.*, 1957). When such patients are given an excess of deoxycortone or cortisone the blood pressure may rise even to hypertensive levels (Loeb *et al.*, 1939; Thorn *et al.*, 1942; Roth *et al.*, 1943). After total adrenalectomy the circulatory responses to adrenaline and noradrenaline are severely impaired, both in the experimental animal (Ramey *et al.*, 1951) and in man (Howland *et al.*, 1956), but these deficiencies may be corrected by the administration of cortisone. In the present investigation the influence of hydrocortisone on peripheral adrenergic activity has been studied in healthy adults; constrictor responses in the hand to adrenaline and noradrenaline were measured before and immediately after an intra-arterial infusion of hydrocortisone.

Subjects and Methods

Repeated tests were performed on 22 healthy young adults—medical students and colleagues—between the ages of 18 and 28, all but two of whom (I.D. and E.P.) were men.

Throughout every experiment the subject rested on a couch in a laboratory kept at $23 \pm 1^\circ \text{C}$., while the blood flow in both hands was measured by venous occlusion plethysmography every half-minute under standard conditions (Duff, 1952).

Through a needle inserted under local analgesia into the brachial artery, saline (0.9% w./v. NaCl) was infused continuously at the rate of 3 ml./min. The hand receiving this infusion (usually the right) was designated the "test" hand, the opposite (left) being the "control" hand. At regular four-minute intervals plain saline was alternated with saline solutions of known concentrations of various hormones. In the first half of each experiment the effects of successive infusions of adrenaline and noradrenaline at dose levels of 1/16 and 1/8 $\mu\text{g./min.}$ were measured, each hormone infusion being preceded by a four-minute infusion of plain saline. Next, hydrocortisone was given by the intra-arterial infusion, in most experiments at the rate of 100 $\mu\text{g./min.}$ for six minutes, and in the remainder at 15, 50, or 200 $\mu\text{g./min.}$ for four or six minutes. The total amounts given were between 60 and 1,200 $\mu\text{g.}$ In the second half of each experiment the various doses of adrenaline and noradrenaline were given in the same concentrations as before. The order in which these hormones were

infused was varied in some experiments and kept constant in others; the results did not appear to be influenced by the sequence in which the infusions were given.

Synthetic L-adrenaline bitartrate (B.D.H.) and synthetic L-noradrenaline bitartrate (Bayer) were diluted with physiological saline (0.9% w./v. NaCl) solution, to which ascorbic acid had been added (1 mg./litre saline) to prevent oxidation (Gaddum *et al.*, 1949). In different tests two preparations of hydrocortisone were used—water-soluble hydrocortisone free alcohol in glycols (Roussel Laboratories) and a free alcohol 50% ethanol preparation (Boots). The activity of each excipient solvent was also tested in a series of control experiments.

The mean change in flow in the test hand during each individual infusion of adrenaline, noradrenaline, or hydrocortisone was estimated from the measurements of blood flow during the control and experimental periods. The results were analysed by a method that made due allowance both for incidental fluctuations in the circulation through the control hand and for bilateral changes in flow unrelated to the test infusions (Duff, 1955a). The results obtained in a typical experiment are illustrated in Fig. 1.

FIG. 1.—Constrictor effects of adrenaline (A/16, A/8) and noradrenaline (N/16) infusions before and after the intra-arterial infusion of 400 $\mu\text{g.}$ of hydrocortisone (Hc).

Results

Effects of intra-arterial adrenaline before and after intra-arterial hydrocortisone

(a) *Adrenaline 1/16 $\mu\text{g./min.}$* —The initial infusion of this concentration of adrenaline generally caused a slight reduction in flow in the test hand during most of the four-minute infusion period. The flow in the other hand usually stayed around the same average level as that during the previous saline control period, but sometimes changed in a haphazard manner unrelated to the level in the test hand. The results of 16 experiments were analysed to reveal the independent change in flow in the test hands attributable to the adrenaline (Table 1, a). The mean effect was a reduction of flow of 29%. After hydrocortisone had been infused into the artery the second infusion of adrenaline (1/16 $\mu\text{g./min.}$) caused an independent reduction of flow in the test hand which was usually greater than before, averaging 41% (Table 1, a). The difference between these values is significant ($t=2.9$, $P<0.02$).

(b) *Adrenaline 1/8 $\mu\text{g./min.}$* —With the higher dose of adrenaline the first infusion usually caused a greater reduction in flow, which in 14 experiments averaged 36%. After the hydrocortisone had been given this dose of adrenaline now caused an even greater reduction in flow, averaging 52% (Table I, b). Here again the difference between the two values (36% and 52%) is significant ($t=4.5$, $P<0.001$).

Thus after the hydrocortisone had been infused the vasoconstriction to adrenaline was significantly greater at both dose levels.

Effects of intra-arterial noradrenaline before and after intra-arterial hydrocortisone

(a) *Noradrenaline 1/16 $\mu\text{g./min.}$* —In 15 experiments the flow was reduced during the first infusions by an average amount of 26% (Table II, a). After the hydrocortisone infusions the noradrenaline was found to cause a fall averaging 29%. These values do not differ significantly ($t=0.6$, $P=0.5$).

(b) *Noradrenaline* 1/8 $\mu\text{g./min.}$ —The initial infusion of this concentration of noradrenaline was associated with a reduction in flow which in 13 experiments averaged 29%. The reduction of flow in the test hands of these subjects after the hydrocortisone average 36% (Table 11, b). These means do not differ significantly ($t=1.1$, $P=0.3$). There was thus no consistent or significant change in the response to noradrenaline of hands previously infused with hydrocortisone.

Effect of intra-arterial hydrocortisone on the resting level of flow in the infused hand

An estimate of the net effect of hydrocortisone on the calibre of the arterioles in the infused hands was obtained by comparing the levels of flow before and immediately after each hydrocortisone infusion. In six experiments the mean flow in the test hands was 6.6 ml./100 ml./min. before and 5.9 ml. after hydrocortisone had been infused in a

TABLE I.—Effects of Intra-arterial Adrenaline (a) 1/16 $\mu\text{g./min.}$, and (b) 1/8 $\mu\text{g./min.}$, Before and After Hydrocortisone

Subject	Before Hydrocortisone							After Hydrocortisone						
	A	a	B	b	E	B-E	B-E/E%	A	a	B	b	E	B-E	B-E/E%
(a) Adrenaline 1/16 $\mu\text{g./min.}$														
E.P.	1.9	2.1	1.3	2.4	2.2	-0.9	-41	2.8	3.2	1.0	2.6	2.3	-1.3	-57
E.H.	15.8	17.4	6.1	11.7	10.6	-4.5	-42	11.7	7.9	8.6	9.1	13.5	-4.9	-36
C.H.	7.0	4.2	6.0	4.7	7.8	-1.8	-23	13.2	5.1	6.9	3.4	8.8	-1.9	-22
M.M.	15.6	19.7	14.2	19.7	15.6	-1.4	-9	11.9	11.3	9.5	19.5	20.5	-11.0	-54
J.B.	12.9	16.1	4.3	9.6	7.7	-3.4	-44	6.0	6.7	5.6	14.2	12.7	-7.1	-56
J.B.	9.1	12.0	5.1	10.7	8.1	-3.0	-37	6.8	7.7	3.5	10.6	9.4	-5.9	-63
I.D.	4.8	15.9	2.6	12.4	3.7	-1.1	-30	9.1	13.6	3.6	7.9	5.3	-1.7	-32
J.P.	8.8	15.9	7.1	16.0	8.8	-1.7	-13	3.3	5.2	1.9	5.8	3.7	-1.8	-49
U.C.	18.2	16.5	15.8	17.8	19.6	-3.8	-19	17.3	12.5	9.8	10.4	14.3	-4.5	-31
C.A.	14.3	15.9	12.0	15.6	14.0	-2.0	-14	19.2	20.8	12.1	18.7	17.3	-5.2	-30
M.M.	13.2	7.7	6.6	5.6	9.6	-3.0	-31	14.8	7.5	8.5	7.8	15.4	-6.9	-45
N.G.	7.2	6.7	3.8	4.4	4.7	-0.9	-19	6.3	6.3	4.4	4.7	4.7	-0.3	-6
J.C.	18.0	16.8	12.2	13.8	14.7	-2.5	-17	18.3	20.3	12.4	18.3	16.5	-4.1	-25
R.H.	5.6	7.1	3.4	6.9	5.4	-2.0	-37	3.9	6.8	1.5	6.8	3.9	-2.4	-62
T.P.	9.7	6.1	5.1	5.6	8.9	-3.8	-43	9.6	5.5	3.8	2.9	5.1	-1.3	-25
J.D.	16.7	8.3	9.0	7.2	14.5	-5.5	-38	18.8	12.4	4.7	8.5	12.9	-8.2	-64
Average							-29							-41
(b) Adrenaline 1/8 $\mu\text{g./min.}$														
C.A.	8.6	11.9	4.9	9.2	6.6	-1.7	-26	7.6	13.3	5.8	13.2	7.5	-1.7	-27
D.B.	7.6	9.9	6.6	9.1	7.0	-0.4	-6	7.1	16.2	4.7	10.9	6.1	-1.4	-23
B.B.	4.0	1.9	1.9	1.8	3.8	-1.9	-50	4.5	1.7	1.3	2.2	5.8	-4.5	-78
E.P.	2.2	1.6	1.1	1.6	2.2	-1.1	-50	2.9	3.7	1.3	5.1	4.0	-2.7	-68
T.F.	7.1	7.6	8.7	8.9	8.3	0.4	5	13.8	10.6	6.7	7.1	9.2	-2.5	-27
F.P.	7.3	5.2	2.2	6.4	9.0	-6.8	-76	8.4	9.3	2.5	13.7	12.4	-9.9	-80
G.M.	7.5	11.2	8.9	18.6	12.5	-3.6	-29	15.2	26.7	5.7	23.9	13.6	-7.9	-58
D.E.	44.9	31.0	41.5	31.2	45.2	-3.9	-9	36.7	21.6	23.2	21.3	36.2	-13.0	-36
J.C.	7.5	16.8	4.5	14.3	6.3	-1.8	-29	9.3	12.9	3.4	12.3	8.9	-5.5	-62
E.H.	9.1	11.5	5.3	11.9	9.4	-4.1	-44	15.7	15.1	11.0	20.1	20.9	-9.9	-47
M.M.	14.1	19.2	10.6	16.8	12.0	-1.4	-12	12.7	14.4	7.4	13.0	11.5	-4.1	-36
J.B.	9.6	14.2	4.2	16.3	11.0	-6.8	-61	9.2	11.7	3.5	15.3	12.0	-8.5	-71
J.C.	21.5	18.1	6.8	14.5	17.2	-10.4	-60	16.4	16.2	8.1	21.5	21.8	-13.7	-63
R.C.	23.5	20.6	12.5	22.6	25.8	-13.3	-52	23.8	23.5	13.6	25.4	25.7	-12.1	-47
Average							-36							-52

A, a=mean blood flow in test and control hands, respectively, during saline infusion period; B, b=corresponding means during succeeding adrenaline infusion period; E=Ab/a; B-E/E%=mean percentage change in flow due to adrenaline.

TABLE II.—Effects of Intra-arterial Noradrenaline (a) 1/16 $\mu\text{g./min.}$ and (b) 1/8 $\mu\text{g./min.}$

Subject	Before Hydrocortisone							After Hydrocortisone						
	A	a	B	b	E	B-E	B-E/E%	A	a	B	b	E	B-E	B-E/E%
(a) Noradrenaline 1/16 $\mu\text{g./min.}$														
E.H.	17.4	18.2	15.1	17.6	16.8	-1.7	-10	13.8	12.3	5.9	12.5	14.0	-8.1	-58
C.H.	4.9	4.6	4.6	5.6	6.0	-1.4	-23	9.8	4.9	13.7	6.1	12.2	1.5	12
M.M.	15.9	21.2	15.6	20.9	15.6	0.1	01	17.9	18.6	11.1	15.2	14.6	-3.5	-24
J.B.	11.0	13.1	7.3	12.4	10.4	-3.1	-30	9.3	12.2	5.2	9.6	7.3	-2.1	-29
I.D.	4.9	16.6	4.4	12.1	3.6	0.8	22	7.6	7.9	6.1	6.4	6.2	-0.1	-02
J.P.	6.8	9.0	2.7	6.2	4.7	-2.0	-43	5.8	8.1	3.0	7.7	5.5	-2.5	-45
U.C.	13.0	8.1	11.2	8.5	13.6	-2.4	-18	12.6	9.1	12.1	10.0	13.8	-1.7	-12
C.A.	17.7	15.5	16.0	19.2	21.9	-5.9	-27	19.8	18.9	14.6	16.8	17.6	-3.0	-17
M.M.	14.0	6.6	13.9	9.0	19.1	-5.2	-27	17.2	11.5	11.8	6.5	9.7	-2.1	-22
N.G.	8.2	7.4	4.7	7.6	8.4	-3.7	-44	6.4	4.8	3.9	3.8	5.1	-1.2	-24
F.P.	8.2	8.7	5.1	9.6	9.0	-3.9	-43	9.2	7.4	4.5	7.1	8.8	-4.3	-49
R.C.	14.3	13.0	8.7	15.2	16.7	-8.0	-48	18.9	22.6	7.6	17.3	14.5	-6.9	-48
J.C.	16.3	22.0	11.6	17.4	12.9	-1.3	-10	19.9	11.3	11.0	11.0	19.4	-8.4	-43
T.P.	13.2	5.8	3.6	3.2	7.3	-3.7	-51	12.7	4.4	7.6	5.3	15.3	-7.7	-50
J.D.	16.9	6.6	11.3	7.2	18.4	-7.1	-39	13.7	8.0	12.1	9.6	16.4	-4.3	-26
Average							-26							-29
(b) Noradrenaline 1/8 $\mu\text{g./min.}$														
C.A.	9.1	8.7	4.9	7.7	8.1	-3.2	-40	5.6	6.9	4.1	7.6	6.2	-2.1	-34
D.B.	6.2	8.6	5.1	8.7	6.3	-1.2	-19	6.3	13.7	5.9	12.4	5.7	0.2	04
B.B.	4.0	1.9	1.4	2.2	4.6	-3.2	-70	4.1	2.5	1.8	3.9	6.4	-4.6	-72
E.P.	1.9	2.1	1.3	1.8	1.6	-0.3	-19	2.9	3.1	0.7	3.1	2.9	-2.2	-76
T.F.	10.2	7.4	10.1	8.6	11.9	-1.8	-15	12.7	11.6	8.4	9.8	10.7	-2.3	-21
F.P.	10.8	8.9	4.1	8.9	10.8	-6.7	-62	10.5	11.2	5.1	10.1	9.5	-4.4	-46
G.M.	12.1	15.3	8.3	16.7	13.2	-4.9	-37	12.6	21.7	8.4	21.2	12.3	-3.9	-32
D.E.	37.9	26.3	35.2	24.4	35.2	0	0	30.9	23.9	23.4	16.3	21.1	2.3	11
J.C.	6.9	17.8	9.1	20.2	7.8	1.3	17	10.6	16.3	5.2	13.9	9.0	-3.8	-42
E.H.	15.8	15.9	8.3	14.9	14.8	-6.5	-44	11.7	11.2	10.8	18.3	19.1	-8.3	-43
R.C.	19.0	15.7	10.3	16.2	19.6	-9.3	-47	34.8	23.0	17.2	24.8	37.5	-20.3	-54
T.P.	15.7	15.0	12.1	15.3	16.0	-3.9	-24	16.6	12.0	7.1	10.7	14.8	-7.7	-52
J.E.	15.9	16.3	9.9	13.1	12.8	-2.9	-23	12.3	15.9	15.9	23.8	18.4	-2.5	-14
Average							-29							-36

A, a=mean blood flow in test and control hands, respectively, during saline infusion period; B, b=corresponding means during succeeding noradrenaline infusion period; E=Ab/a; B-E/E%=mean percentage change in flow due to noradrenaline.

concentration of 50 $\mu\text{g./min.}$ Similarly, using 100 $\mu\text{g./min.}$ hydrocortisone the mean blood flow levels were 12.4 ml. and 13.1 ml. respectively in 37 experiments, and with 200 $\mu\text{g./min.}$ the mean levels were 17.8 ml. and 16.6 ml. in 10 experiments. Thus, clearly, hydrocortisone itself (in concentrations between 50 and 200 $\mu\text{g./min.}$) does not cause any notable change in the level of flow through the arterioles into which it is infused.

Effect of hydrocortisone-free excipient on responses to adrenaline and noradrenaline

Ten experiments were performed, using a procedure identical to that adopted in the tests in which hydrocortisone was given at dose rates of 100 and 200 $\mu\text{g./min.}$, except that the excipient solution alone was used, the hydrocortisone itself being omitted. Adrenaline and noradrenaline infusions were given before and after the solvent had been infused into the brachial artery. The dose of each hormone was 1/16 $\mu\text{g./min.}$ In 23 paired tests with adrenaline the mean effects were a 28% reduction in flow before, and 25% after, the hydrocortisone-free solvent; these values clearly do not differ appreciably. In 10 tests with noradrenaline the corresponding values were 21% and 26%, which also do not differ significantly.

Discussion

These experiments show (Fig. 2) that in the hands of healthy subjects the average constrictor effect of intra-arterial adrenaline was enhanced by infusing hydrocortisone

FIG. 2.—Mean constrictor effects in 22 subjects of adrenaline and of noradrenaline (1/16 and 1/8 $\mu\text{g./min.}$) before and after hydrocortisone intra-arterially.

into the brachial artery. The absence of any change in the control experiments, using the solvent present in ampoules issued commercially, indicates that the observed effects were not due to the material in which hydrocortisone is dissolved. It is therefore reasonable to attribute the increased response to the hydrocortisone itself.

The increased constrictor response was evident within a few minutes of completing the infusion of hydrocortisone. This effect must have been due to a purely peripheral action of the steroid, for the mode of administration and method of analysis excluded the influence of systemic factors and of changes in sympathetic activity. The arterioles have thus been "sensitized" to the direct action of adrenaline, and, since this "hypersensitivity" was not associated with any change in the resting level of hand flow, it is unlikely that some physical change such as swelling of the arteriolar walls was responsible. This interpretation is supported by the fact that the hydrocortisone was not usually followed by an increase in the constrictor response of these same vessels to noradrenaline.

The pattern of response to the adrenergic hormones after intra-arterial hydrocortisone, when there is a more powerful constriction with adrenaline than with noradrenaline, contrasts with that usually found in healthy subjects. But in untreated hypertensive patients studied under identical

experimental conditions it was found previously that the constrictor responses of the hands to adrenaline tended on the whole to be greater than normal (Duff, 1957a). No such striking difference with regard to noradrenaline was revealed as between subjects with normal and raised blood pressures (Duff, 1955b, 1957b). Thus the selective enhancement of the arteriolar response to adrenaline after the introduction of hydrocortisone into the brachial artery of healthy subjects closely resembles that observed in the hands of patients with untreated essential hypertension.

Although hydrocortisone has been identified as a normal product of the human adrenal cortex, and although there is good evidence that the output of this hormone may be raised after the administration of corticotrophin (Nelson *et al.*, 1951; Sweat, 1955) and in Cushing's syndrome (Eik-Nes *et al.*, 1955; Sweat, 1955) it is within normal limits in essential hypertension (Eik-Nes *et al.*, 1955). There is indeed no direct evidence that hydrocortisone itself is involved in hypertension. However, the possibility that in hypertensive subjects there may be an alteration in vascular function similar to that observed after hydrocortisone in the present study, consequent upon some imbalance of adrenal cortical secretion, perhaps deserves further investigation.

Although the present investigation revealed no consistent enhancement of the response to noradrenaline in vessels of the hand (Fig. 2), pressor responses to this hormone are definitely potentiated by systemic cortisone in both animals and men (Kurland and Freedberg, 1951; Ramey *et al.*, 1951; Howland *et al.*, 1956). The latter effect must therefore be attributed to actions on vascular beds other than those of the skin, and it is interesting that topical applications of cortical extracts increase the constriction produced by noradrenaline in the meso-appendix of the adrenalectomized rat (Fritz and Levine, 1951). Further investigation might therefore be directed to an examination of the influence of hydrocortisone and other adrenal corticoids on the actions of both adrenaline and noradrenaline in vascular beds apart from those of the skin.

Summary

The effects of intra-arterial hydrocortisone on the constrictor responses of vessels of the hand to intra-arterial adrenaline and to noradrenaline have been measured in 22 healthy adults by a plethysmographic method.

Hydrocortisone itself, in amounts totalling between 0.6 and 1.2 mg., caused no appreciable change in the level of blood flow in the hand.

After infusing hydrocortisone there was usually an increase in the vasoconstriction produced by given amounts (0.25 and 0.5 $\mu\text{g.}$) of adrenaline; but the response to noradrenaline was not greatly altered.

This selective change in vascular response produced by hydrocortisone is shown to resemble the vascular hypersensitivity found in some untreated hypertensive patients.

Some implications of these findings are discussed.

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METHOD OF REDUCING FRACTURE-DISLOCATION OF HIP-JOINT WITH FRACTURED FEMUR ON SAME SIDE

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A fracture of the shaft of the femur complicating a dislocation of the hip on the same side makes the task of reducing the dislocation extremely difficult. Two such cases are described of severe multiple injuries to a lower limb each complicated by a posterior dislocation of the hip in which the dislocation was reduced by a method based on that described by Ingram and Turner (1954).

The method consists in transfixing the greater trochanter of the femur percutaneously in an antero-posterior direction with a stout Steinmann pin. On to each projecting end of the pin is clamped a Mole self-grip wrench of the type used by mechanics and commonly known as the "third hand." This wrench grips the pin more efficiently than any surgical instrument, and, once clamped tight, it does not slip or rotate. With one assistant controlling the fractured femur and another steadying the pelvis, the operator holds the wrench in either hand and exerts traction on the proximal fragments of the femur in a position of flexion and abduction until reduction is accomplished. Strong traction may have to be sustained for a few minutes. After reduction has been performed the pin is withdrawn.

In the first case described here the method was employed after exposing the hip-joint, while in the second case a closed reduction was used successfully.

Case 1

A man of 25 sustained severe multiple injuries to his left leg in a motor-cycle accident. The foot and ankle were so badly damaged that a below-knee amputation was carried out after initial resuscitation with blood transfusions. Radiographs showed a comminuted supracondylar fracture of the left femur. The hip region was not radiographed initially. When attempts to reduce the supracondylar fracture failed to align the fragments a posterior dislocation of the hip-joint was suspected. The proximal fragment of the femur was fixed in flexion, abduction, and internal rotation. The dislocation defied all attempts at reduction and manipulation. The patient's condition at this stage did not warrant an open reduction being performed, and he was returned to bed with the

limb on a Thomas splint with skeletal traction through the tibial tubercle. Radiographs taken next day showed a posterior dislocation of the left hip.

For the next few days his condition gave rise to anxiety in spite of further blood transfusions, but at the end of a week he had recovered sufficiently for an open reduction of the dislocation to be attempted.

Operation.—The left hip was exposed by a postero-lateral incision, and the head of the femur was found lying on the back of the ilium with no sign of an investing capsule. The posterior margin of the acetabulum had been split off into two separate fragments. Attempts were made to reduce the dislocation, but in spite of using all the available instruments the head could not be made to budge. A pair of self-grip wrenches were borrowed from the hospital engineer, cleansed, and sterilized. The greater trochanter was transfixed with a Steinmann pin and the wrenches were clamped on to the pin on each side of the bone. By grasping the wrenches the femur could then be lifted up and forward until the dislocation was reduced with surprising ease. The posterior acetabular margin was replaced and held in position with two screws. The fractured femur was then realigned and placed on the Thomas splint.

Twelve weeks after this operation the fractured femur had united and the patient was discharged from hospital, walking with crutches. He had regained an excellent range of movement of both hip- and knee-joints. Three months later he was walking about with his artificial limb.

Photograph of model showing the application of the self-grip wrenches.

Case 2

A youth of 18 was admitted to hospital in a shocked state with severe multiple injuries to his left lower limb. There was a compound fracture of the big toe, a double fracture of the tibia, and a supracondylar fracture of the femur. In addition to these injuries there was a posterior dislocation of the hip-joint which on this occasion was recognized initially when attempts made to straighten the femur had failed to correct the angulation. Closed manipulation of the dislocation was unsuccessful and a Steinmann pin was then inserted through the skin, transfixing the greater trochanter. The self-grip wrenches were again borrowed and brought into use as previously described. With this method the dislocation was reduced with an audible snap. The pin was withdrawn and the other injuries to the left leg were then treated. Two Steinmann pins were driven across the upper and lower ends of the