

HYDROCORTISONE AS AN ADJUNCT IN THE TREATMENT OF POSTTRAUMATIC STIFFNESS OF THE HAND

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In the hand as well as in all other parts of the body, sufficiently severe injury will cause swelling. As Bunnell (5) puts it, the injured tissue will be bathed in sero-fibrinous exudate. As a result, fibrin will be deposited between the various tissue layers, around the joints, tendons, tendon sheaths, ligaments, and within and between the muscles. In this fibrin an ingrowth of blood vessels and cells will take place, as well as new-formation of fibrils and ground substance, in brief, connective tissue which again will shrink, and finally all these structures will be plastered together with firm adhesions.

The nature of the primary injury differs: Crushing of soft parts, fractures, distortions, infections, haematomas, or ordinary incised wounds or operative wounds leading to immobilization of the hand.

Immobilization is the dangerous factor. With immobilization, oedema and tissue fluids are not removed by the muscular pump, and cicatrization rapidly entails limitation of movement.

Ragan and his associates (12), studying the effect of cortisone on wounds healing in the rabbit, found inhibition of all new elements present in the connective tissue: a reduced number of blood vessels and fibroblasts and less ground substance. Cortisone, however, also affects existing fibrosis. This was shown by Baxter (3, 4) who used cortisone in the treatment of a patient with pronounced limitation of movement owing to excessive scar-formation 3½ months after an operation for Dupuytren's contracture. The range of motion improved considerably.

Owing to its predominantly topical effect 17-hydroxycorticosterone-21-acetate (hydrocortisone acetate, compound F-acetate) would appear

to afford a means of treating posttraumatic stiffness of the hand with doses so small that systemic effects are avoided. Our principle in this treatment was not to use hydrocortisone acetate until the condition had become stationary using ordinary physiotherapy.

The patients had one weekly injection¹. The injections were preceded by local anaesthesia to avoid the pain which may be caused by injection into the firm fibrous tissue of an injured hand. The dosage varied according to the size of the involved area, ranging from 10 to 25 mg in a suspension of 1-2 cc of physiological saline.

Table 1 gives a survey of the cases.

CASE REPORTS

Case 1. Housewife, 55 years of age, who had sustained a left-sided Colles' fracture on Oct. 27, 1952. The fracture line in the radius involved the joint. Treatment: Reduction and plaster cast. Oedema of the hand appeared, and the plaster cast was cut up. The fracture healed with the articular surface turning slightly dorsal. On Dec. 10 the plaster cast was removed and energetic physiotherapy instituted as the hand was very stiff.

On Apr. 15, 1953, after 4 months' physiotherapy, the condition was stationary. Movements of the wrist: dorsal 5°, volar 45°, greatly restricted mobility of the fingers, the tips of the four ulnar fingers being 6, 7, 7, and 6 cm short of reaching the palm. The patient was now given hydrocortisone acetate, 25 mg × 6, infiltrating the stiff fingers and the site of the fracture. The physiotherapy was continued.

On June 24, the wrist moved 35° towards the dorsal and 80° towards the volar aspect. The mobility of the fingers was perceptibly improved, the tips of the four ulnar fingers now being 3½, 4, 4, and 3½ cm short of reaching the palm. The patient also reported marked improvement of pain. After 2 months' follow-up the mobility was on the whole unchanged.

Case 2. Joiner, 53 years of age. On Jan. 13, 1953, the four ulnar fingers of his left hand had been caught in an electric cutter. Open fractures of the middle phalanges of all four fingers were treated with suture and plaster casts. Six weeks later all the fractures had healed, and physiotherapy was instituted. On May 20, despite 2 months' physiotherapy, movements were extremely limited. The patient had 25 mg of hydrocortisone acetate weekly for 3 weeks, injected into the fibrotic areas at the sites of the fractures. The treatment was ineffective, and the follow-up at the end of 2 months showed an unchanged range of motion.

Case 3. Foundry worker, 42 years of age. On Sept. 1, 1952, his left hand had been crushed by 65 kg of aluminium sheets. In addition to contusion wounds there were fractures of the third, fourth and fifth metacarpal bones and of the proximal phalanx of the long finger. Plaster cast for three months. The fractures healed, leaving posttraumatic dystrophy of the hand. On Apr. 23, 1953, the condition had become stationary. Despite more than 4 months' physiotherapy, the hand was bluish, cool, and painful with greatly restricted movement. There was halisteresis,

¹ "Hydrocortisate Leo" kindly supplied by Leo Pharmaceutical Products, Copenhagen.

TABLE 1
Survey of the cases treated, dosage of hydrocortisone acetate, and the results of treatment.

Case	Diagnosis	Complication	Hydrocortisone	Result	Follow-up period
1	Colles' fracture	Oedema of the hand	25 mg × 6	Good	2 months
2	Multiple phalangeal fractures	Fibroses	25 mg × 3	Unchanged limitation of movements	3 months
3	Multiple metacarpal and phalangeal fractures	Posttraumatic dystrophy, fibroses	25 mg × 7	Dystrophy disappeared. Unchanged limitation of movements	4 months
4	Lacerated wound on the dorsum of the hand	Fibrosis	10 + 25 mg	Good	1 month
5	Lacerated wound on the palm	Infection of the tendon sheaths	25 mg × 4	Good	3 months
6	Open phalangeal fracture	Fibrosis	25 mg × 3	Good	2 months
7	Open phalangeal fracture	Fibrosis	25 mg × 4	Good	3 months
8	Multiple phalangeal fractures	Posttraumatic dystrophy, fibroses	10 mg × 5 into each finger	Dystrophy disappeared. Improved mobility	4 months
9	Haemangioma of the hand	Postoperative fibroses	25 mg × 4	Good	6 months

osteoarthritis of the metacarpo-phalangeal joint of the long finger, hyperextension of the proximal joint of the ring finger, firm fibrosis in the palm. The patient refused capsulectomy of the ring finger. After 7 injections of hydrocortisone acetate, 25 mg, into the fibrosed areas, the dystrophic symptoms subsided, the fibroses disappeared, and there was an improvement in passive movement, whereas the range of active movement remained unchanged. The patient started working on July 30. At the follow-up on Sept. 8 the trophic condition of the hand was normal, but the mobility remained unchanged.

Case 4. Spinner, 50 years of age. On Jan. 17, 1953, his left hand had been caught in a roller. There was a large lacerated wound on the dorsum of the hand, extending from the ulnar part of the wrist to the first interstice and as far as the middle



Fig. 1.

Case 5. Maximum flexion of the fingers 2½ months after the injury. Condition stationary despite physiotherapy (May 8, 1953).

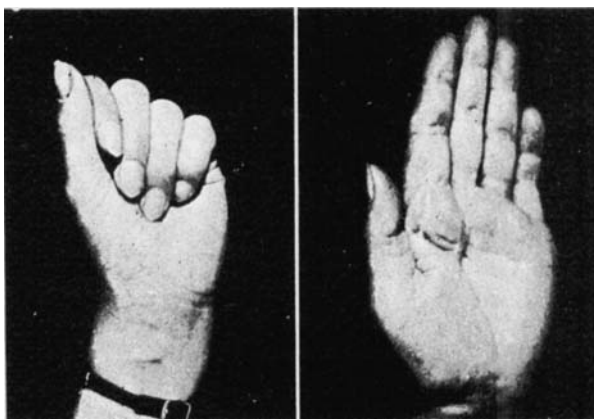


Fig. 2.

Case 5. Left: Maximum flexion of the fingers after treatment with hydrocortisone acetate, 4 × 25 mg. (June 24, 1953). Right: Full extension after the treatment. In the palm the remains of the cicatricial shrinkage and the fibroses (June 24, 1953).

joint of the index finger. After prolonged treatment with aureomycin dressing, the wound healed, leaving only a cicatricial, dermal contraction over the extensor tendon to the index finger which was 2 cm short of reaching the palm. This condition remained stationary until July 10. On this day 10 mg and on July 17 25 mg of hydrocortisone acetate were injected into the cicatricial tissue on the dorsum of the hand. As early as one week after this first injection, the tissue had softened, and on July 31, the patient could clench his hand without any tightening and the tip of the index finger now reached the palm.

Case 5. Housewife, 51 years of age. On Jan. 17, 1953 her left hand was caught in a wringer. A large lacerated wound in the distal and radial part of the palm was excised, sutured and treated with penicillin. Healing was slow, and on March 25 infection in the tendon sheaths of the index finger developed. This was treated with incisions into the palm and aureomycin dressing. The infection subsided and the wounds healed, leaving pronounced fibrosis. Energetic physiotherapy was instituted, but at the end of one month, on May 8, there was a marked limitation of all movements and the hand was painful (Fig. 1).

Four injections of hydrocortisone acetate, 25 mg, were now made into the cicatricial shrinkage of the skin as well as into the firm subcutaneous fibrosis and around the tendons. The physiotherapy was continued in order to utilize the resulting softening and suppleness of the fibrous tissue. During this treatment, the pain disappeared and the mobility improved, almost to normal (Fig. 2).

The improvement of the index finger may be seen from Table 2.

TABLE 2

Case 5. Improvement in the mobility of the index finger during treatment with hydrocortisone acetate. This patient received 4 injections of 25 mg (May 8, 22, and 29 and June 24). The improvement is pronounced as early as 14 days after the first injection, first in the proximal joint, then in the middle and eventually—after the injections were stopped—in the distal joint.

Date	Proximal joint	Middle joint	Distal joint
8/5	15°	15°	5°
22/5	60°	30°	10°
29/5	60°	50°	10°
24/6	65°	90°	25°
27/7	70°	90°	45°

This patient was followed up for 3 months after the treatment was finished, and the mobility has improved a little more.

Case 6. Labourer, 30 years of age. On March 10, his right index finger was crushed between two iron rails. An open fracture of the proximal phalanx with a wound, 1 cm in length, on the dorsal aspect was treated with reduction and plaster cast. On May 20 the fracture had healed and physiotherapy was instituted. On July 20 the condition was stationary, the range of movement in the proximal joint being 85°, in the middle joint 20° and in the distal joint 15°. The tip was 5 cm short of reaching the palm, and on the dorsal aspect of the finger there was a large area of fibrosis (Fig. 3).

The physiotherapy was now combined with three injections of hydrocortisone acetate, 25 mg. On Sept. 21 the range of movement in the proximal joint was 110°, in the middle joint 70°, and in the distal joint 30°. The finger tip-palm distance was now only 1½ cm (Fig. 4). The follow-up two months later showed the same range of movement.

Case 7. Joiner, 25 years of age. On Dec. 29, 1952 he cut his left long finger in a band saw. There was an incised wound on the volar aspect of the middle phalanx with a severed tendon and fracture of the middle phalanx. A plaster cast was ap-



Fig. 3.

Case 6. Left: Maximum flexion of the index finger 4½ months after open phalangeal fracture. Right: Maximum extension of the index finger. Condition stationary despite physiotherapy (Aug. 1, 1953).



Fig. 4.

Case 6. Range of mobility in the index finger after treatment with hydrocortisone acetate, 3 × 25 mg. Left: Maximum flexion. Right: Maximum extension (Sept. 21, 1953).

plied. One month later callus formation was satisfactory, and physiotherapy was instituted.

On Apr. 22 the condition was stationary, the range of movement in the proximal joint of the long finger being 70°, in the middle joint 40°, and in the distal joint 0°. The tip was 5½ cm short of reaching the palm. The patient asked for amputation of the finger which interfered with his work (Fig. 5).

Four injections of hydrocortisone acetate, 10 mg, were given into the fibrotic area on the volar aspect of the finger. On June 2, the mobility in the proximal joint was 100°, in the middle joint 100°, and in the distal joint 0°. The tip now reached the palm (Fig. 6). Four months later the range of movement was un-

changed. It is worth mentioning that after the treatment passive movement in the distal joint had increased from 0 to 35°.

Case 8. Female factory worker, 45 years of age. On Oct. 10, 1952 her right hand was crushed in a machine. There were large contusion wounds on the fingers and fractures of the proximal phalanx of the long finger and the middle phalanges of the three ulnar fingers. A plaster cast was applied until Dec. 5, when physiotherapy

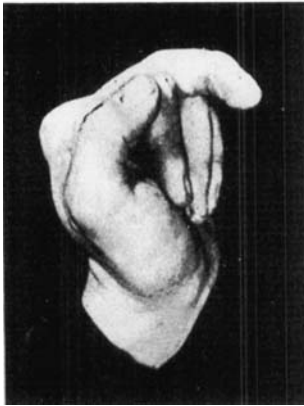


Fig. 5.

Case 7. Maximum flexion of the long finger 4 months after open phalangeal fracture. Condition stationary despite physiotherapy (Apr. 22, 1953).



Fig. 6.

Case 7. Maximum flexion of the long finger after treatment with hydrocortisone acetate, 4 × 25 mg (June 2, 1953).

was instituted. Posttraumatic dystrophy developed, and on Apr. 21 the condition was stationary, showing haliteresis, shiny, cyanosed, pointed fingers, and pain upon movement which was greatly limited (Fig. 7).

Hydrocortisone acetate was now started, 10 mg × 5 into each finger, infiltrating the firm fibrous tissue. This resulted in marked improvement, exemplified by the index finger: proximal joint 45°, middle joint 10°, distal joint 0° on Apr. 21 to 80°, 40°, and 10° respectively on July 22. During this treatment the dystrophic symptoms and haliteresis subsided and the cyanosis and pain disappeared (Fig. 8).

At the follow-up four months later the condition was unchanged from July 22.

Case 9. Clerk, 56 years of age. A very large haemangioma of the right hand was removed on Jan. 28, 1953. Large incisions were required on the dorsum as well as on the ulnar edge of the hand. The haemangioma was removed radically. Post-operative haematoma of the wound and marginal necrosis occurred. After 2 months' physiotherapy the condition was stationary, showing great limitation of movement, particularly in the three ulnar fingers, and fibrous thickening, particularly of the 4th interstice (Figs. 9 and 10).

Hydrocortisone acetate was now injected into the firm fibroses in the palm and on the dorsum, 25 mg weekly, four times in all. Follow-up on Apr. 25:—ring finger 4 cm long and index fingers 3 cm short of reaching the palm. The proximal joints lacked 30° of full extension. Follow-up Sept. 9:—the hand can now be clenched normally except that the two ulnar fingers touch the palm 2 cm proximal



Fig. 7.

Case 8. Maximum flexion of the fingers 6 months after injury involving multiple phalangeal fractures. Posttraumatic dystrophy with shiny, cyanosed, pointed fingers (Apr. 21, 1953).



Fig. 8.

Case 8. Maximum flexion of the fingers after treatment with hydrocortisone acetate, 5×10 mg into each finger. Dystrophy has disappeared (July 22, 1953).

to the normal site. Extension defect of only 15° in the proximal joints, middle and distal joints freely movable. All fibroses had disappeared, and the colour and trophic conditions were normal (Figs. 11 and 12).

DISCUSSION

Without a doubt, the effect of hydrocortisone acetate is due to its inhibitory effect on connective tissue. As mentioned above, the stiffness is caused by sero-fibrinous exudate, deposition of fibrin, formation of connective tissue, and cicatrization. The mechanism of the inhibitory effect of cortisone on the connective tissue formation has not yet been fully elucidated.

Ragan (12) has called attention to an anti-anabolic effect of hyperadrenalism on the protein metabolism which may be responsible for the reduced reaction from mesenchymal tissue.

According to Asboe-Hansen (1-2) the mast cells are probably the secretors of hyaluronic acid which is an important constituent of the connective tissue ground substance. He also showed that under the influence of cortisone, these cells decrease in number and become degranulated.

In tissue cultures, Cornman (6) has shown the inhibitory effect of cortisone on fibroblasts which according to Gersh and Catchpole (8)



Fig. 9.

Case 9. Maximum extension of the fingers 2 months after operation for haemangioma. Condition stationary despite physiotherapy (March 28, 1953).



Fig. 10.

Case 9. Maximum flexion of the fingers before treatment (March 28, 1953).



Fig. 11.

Case 9. Maximum extension of the fingers after treatment with hydrocortisone acetate, 4×25 mg (Sept. 9, 1953).



Fig. 12.

Case 9. Maximum flexion of the fingers after treatment (Sept. 9, 1953).

form the ground substance. In other words, a reduced number of fibroblasts give a reduced amount of ground substance.

In addition, it has been demonstrated that hydrocortisone affects the lymphocytes (7) and that cortisone reduces the number of plasma cells (13).

Menkin (10) found that hydrocortisone inhibits the effect of in-

flammatory reaction by reducing the effect of leukotaxine, thus diminishing the increased capillary permeability caused by the injury and reducing the deposition of fibrin. This suggests that perhaps an even better effect on injured tissue may be obtained by immediate administration of hydrocortisone in cases which empirically develop stiffness. This is, however, contra-indicated by the risk of infection in open wounds and, e.g. in cases of fractures and tendon injuries, by the delay in union caused by hydrocortisone.

Our patients were not started on hydrocortisone until the condition had become stationary while using ordinary physiotherapy. Consequently, the effect of the drug stands out clearly. At the same time, we avoid the risks involved by starting the treatment before all wounds have healed, especially the risk of infection, as cortisone or hydrocortisone acetate prevent the natural defence reactions.

Harrison (9) has reported a case of crush injury of the hand in which cortisone was started too early. Infection occurred; it could not be controlled, and the patient lost 4 fingers.

It may perhaps give rise to some surprise that the hormone affects fibrous scar tissue which has already become firm. It must be borne in mind, however, that any tissue is constantly being broken down and built up. Such tissue also contains more active areas which perhaps are responsible for the continued production of connective tissue, and it is probably the action of cortisone on these islets which softens the tissue.

S U M M A R Y

Following adequate, but ineffective physiotherapy 9 patients with posttraumatic stiffness of the hand were treated by topical injections of hydrocortisone acetate, 10-25 mg weekly. In 7 cases the mobility improved perceptibly and in 2 of these associated dystrophic symptoms disappeared. In the remaining two cases the limitation of active movement remained unchanged.

The mechanism of the onset of the stiffness and the effect of hydrocortisone are discussed.

The treatment should be carried out under local anaesthesia, as it is painful. Contra-indications are open wounds because of the risk of infection and unhealed fractures or tendon injuries as hydrocortisone would delay their union.

R E S U M E

Après physiothérapie adéquate mais inefficace, 9 malades souffrant de raideur post-traumatique de la main ont été traités par des in-

jections topiques d'acétate d'hydrocortisone, 10 à 25 mg par semaine. Dans 7 cas la mobilité s'est améliorée d'une façon perceptible et dans deux de ces cas des symptômes dystrophiques associés ont disparu. Dans les deux autres cas, la limitation des mouvements actifs est restée inchangée.

Le traitement doit être administré sous anesthésie locale, car il est très douloureux. Les contre-indications sont les plaies ouvertes parce qu'elles entraînent le risque d'infection et les fractures non guéries ou les lésions des tendons, l'hydrocortisone retardant alors leur jonction.

ZUSAMMENFASSUNG

9 Patienten bei denen eine angemessene aber unwirksame physikalische Behandlung wegen posttraumatischer Versteifung der Hand vorgenommen worden war, wurden mit örtlichen Einspritzungen von Hydrocortison Azetat, 10–25 mg wöchentlich, behandelt. In 7 Fällen besserte sich die Beweglichkeit deutlich und in 2 dieser Fälle verschwanden auch die dystrophischen Symptome. In den übrigen 2 Fällen hielt sich die Einschränkung der aktiven Beweglichkeit unverändert.

Die Behandlung sollte unter örtlicher Betäubung ausgeführt werden, da sie schmerzvoll ist. Gegenanzeigen sind: Offene Wunden wegen der erhöhten Infektionsgefahr und nicht geheilte Brüche oder Sehnen-schäden, da Hydrocortison die Heilung verzögern würde.

REFERENCES

1. *Asboe-Hansen, G.*: The Origin of Synovial Mucin, *Ann. Rheumat. Dis.*, 9: 149, 1950.
2. – The Mast Cell an Object of Cortisone Action on Connective Tissue, *Proc. Soc. Exp. Biol. & Med.*, 80: 677–679, 1952.
3. *Baxter, H., Johnson, L., Mader, V. & Schiller, C.*: Cortisone as an adjunct in the treatment of postoperative stiffness of the hand. *Canad. M.A.J.* 63: 540–543, 1950.
4. *Baxter, H., Schiller, C., Johnson, L., Whiteside, I. H. & Randall, R. E.*: Cortisone Therapy in Dupuytren's Contracture. *Plastic and Reconstr. Surg.*, 9: 261, 1952.
5. *Bunnell, S.*: *Surgery of the Hand*. I. B. Lippincotts & Co., Philadelphia 1944, p. 199.
6. *Cornman, I.*: Selective Damage to Fibroblasts by Desoxycorticosterone in Cultures of Mixed Tissue. *Science* 113: 37–39, 1951.
7. *Frank, I. A. & Dougherty, T. F.*: Cytoplasmic Budding of Human Lymphocytes Produced by Cortisone and Hydrocortisone in in vitro Preparations. *Proc. Soc. Exp. Biol. & Med.* 82: 17–19, 1953.

8. *Gersh, I. & Catchpole, H. R.*: The Organization of Ground Substance and Basement Membrane and its Significance in Tissue Injury, Disease and Growth. *Am. J. Anat.*, 85: 457-522, 1949.
9. *Harrison, S. H.*: Crush injury of the hand treated by cortisone. *Brit. J. of Plastic Surgery*, 5: 181-186, 1952.
10. *Menkin, V.*: Mechanism of suppression of inflammation by Compound F. *Fed. Proc.* 12: 318, 1953.
11. *Ragan, Ch., Howes, E. L., Plotz, C. M., Meyer, K. & Blunt, Y. W.*: Effect of Cortisone on Production of Granulation Tissue in the Rabbit. *Proc. Soc. Exp. Biol. & Med.*, 72: 718, 1949.
12. *Ragan, Ch., Howes, E. L., Plotz, Ch. M., Meyer, K., Blunt, J. W. & Lattes, R.*: The effect of ACTH and cortisone on connective tissue. *Bull. of the New York Akad. of Med.*, 26: 251-254, 1950.
13. *Teilum, G., Engbæk, H. C. & Simonsen, M.*: Effect of cortisone on plasma cells and reticulo-endothelial system in hyperimmunized rabbits. *Acta endocrinol.*, 5: 181, 1950.