Clinical and Cardiodynamic Effects of Adrenocortical Steroids in Congestive Heart Failure

By Murray A. Greene, M.D., Arthur Gordon, M.D., and Adolph J. Boltax, M.D.

The tendency of adrenocortical steroids and corticotrophin (ACTH) to promote salt and water retention, resulting in an increased extracellular fluid volume, has been established by many investigators. Studies have also demonstrated the importance of excessive activity of the adrenal cortex in the pathogenesis of fluid retention and edema formation in various disease states, including heart failure. In contrast, sodium and water diuresis and an improved response to mercurial diuretics, accompanied by clinical improvement, were reported in some patients with congestive heart failure during or following the administration of corticosteroids and ACTH. The need to elucidate the problem of the effects of these agents in heart failure and the role of adrenal corticoids in the formation of edema are the basis for the present study of the effects of corticosteroids upon the clinical status, electrolyte balances, and cardiovascular dynamics in a group of subjects having heart disease of varied etiologies and congestive failure of varying severity.

Materials and Methods

Nine patients, 4 female and 5 male, with congestive heart failure are the subjects of this study (table 1). Ages ranged from 17 to 65 years. The patients are further classified according to etiology, functional capacity (New York Heart Association), and clinical status in table 1. Varied etiologies and mild to severe degrees of heart failure are represented.

The patients were hospitalized throughout the study and were semi-ambulatory or restricted to bed rest according to their functional capacities.

A steady clinical state of at least 5 to 7 days' duration was required as a control period prior to physiologic testing. This state was maintained by the usual therapy for congestive heart failure and included a standard low-salt diet (less than 2 to 3 Gm. of sodium chloride daily). Mercurial diuretics were not given during this control period although most patients had received this therapy during the course of their previous illness.

During the control period, routine clinical observations were made and pertinent laboratory data obtained. Radiiodine uptakes by the thyroid gland were measured in order to evaluate the possibility that depression of thyroid function by steroids may secondarily affect clinical states and cardiodynamics. Phenolsulphonphthalein excretions and urea clearances were measured as gross indices of renal function. Fluid balances were evaluated according to daily fasting weights, daily urine outputs, and frequent determinations of serum and urine electrolytes.

Physiologic studies of cardiovascular hemodynamics by the standard technic of right heart catheterization were then performed (tables 2A and 2B). The patients were in the resting recumbent positions and were not given premedication. Duplicate measurements of cardiac output (direct Fick) were made. Pressures were obtained with the use of Statham strain-gage transducers (P23A) and recorded on a multichannel oscillographic photographic recorder. Mean pressures were obtained by electrical integration of the pressure pulses. Since flow and pressure measurements did not vary significantly during single studies, only average values are tabulated. Total pulmonary vascular, pulmonary "arteriolar," and total peripheral arterial resistances were calculated according to standard formulas (Poiseuille). Resistance is expressed as dynes sec. cm. -5 by the use of conversion factors.

Total plasma volumes were determined by the R31-labeled human serum albumin method and converted to total blood volumes by the use of peripheral hematocrit values. Predicted plasma volumes for either sex were based on the studies of Samet et al. These values are essentially similar to those reported by other investigators. Pul-

\[ \text{Conversion factor from mm. Hg to dynes cm.}^{-5} = 1,332 \]
Table 1

Clinical Status Prior to, during, and at Termination of Steroid Therapy

<table>
<thead>
<tr>
<th>Patients</th>
<th>Steroid therapy</th>
<th>Functional classification (N.Y. Heart Assn.)</th>
<th>Dyspnea?</th>
<th>Weight (lbs.)</th>
<th>Neck vein distention</th>
<th>Liver size (finger-breadths below costal margin)</th>
<th>Peri. edema?</th>
<th>Pulmonary vascular congestion (x-ray)</th>
<th>Mereurial diuretic therapy</th>
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<tr>
<td>1. F., age 43 yr. CHF, etiology unknown</td>
<td>Prednisone 40 mg. 4 days</td>
<td>I IV Severe 132</td>
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<td>2</td>
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<td>Moderate</td>
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<td>2. F., age 37 yr. RHD: AS, MI, AI, MS</td>
<td>Prednisone 15 mg. 6 days</td>
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<td>90°</td>
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<td>Moderate</td>
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<td>90°</td>
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<td>4. M., age 65 yr. IMH</td>
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<tr>
<td>8. M., age 42 yr. RHD: MS</td>
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<td>I III Moderate 134</td>
<td>90°</td>
<td>3</td>
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<td>Marked</td>
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<tr>
<td>9. M., age 59 yr. PF, E, CP</td>
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<td>3</td>
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</table>

For explanation of reference marks see opposite page.
monary vital capacities and maximum breathing capacities were obtained the day prior to catheterization with the modified Benedict-Roth spirometer.

After the control clinical and physiologic studies were completed, adrenocortical steroids were administered for 13 to 16 days (table 1). Six patients received prednisone and 3 received triamcinolone. These agents were selected because of their diminished tendency to cause sodium and water retention, edema, and potassium depletion as compared to other agents. None of the other usual contraindications to steroid therapy was present.

Clinical status, routine laboratory data, and fluid balances were evaluated throughout the phase of steroid therapy. Radioiodine uptakes by the thyroid gland were measured prior to repeat cardiac catheterization. The previously stabilized cardiac regimen was continued during this period. Mercurial diuretics were given whenever warranted by the development of appreciable aggravation of clinical heart failure. Supplementary potassium therapy was not given.

Right heart catheterization and blood volume determinations were then performed on the last day of steroid administration; vital capacities and maximum breathing capacities were determined on the day prior to catheterization (tables 2A and 2B).

Clinical status and fluid balances were observed in some patients for several days following steroid withdrawal.

Changes in cardiovascular hemodynamics as a result of drug administration were evaluated in terms of statistical probabilities. In order to determine whether these changes may have been due to chance variation, or due to the action of an introduced variable (steroids in this study), 20 consecutive right heart catheterizations in this laboratory in adults with varied types of heart disease and with varying degrees of congestive failure were analyzed. In each of these studies at least 2 control “resting” cardiac output determinations (direct Fick), multiple pressure measurements, and resistance calculations were performed at intervals of 22 to 143 minutes (average 41 minutes), while the patients’ states were considered to be constant. Chance or expected variations in terms of 95 per cent confidence limits (2 standard deviations from the mean variation) were established on this basis for the various measures of cardiovascular hemodynamics. A wide range of abnormalities in cardiovascular hemodynamics was present in this control group, as would be expected in any series of patients with heart disease and congestive failure. It was therefore thought that greater reliability in an evaluation of the results would exist if these confidence limits were expressed in terms of percentage variations rather than changes in absolute values. Two exceptions are pulmonary “wedge” and right ventricular end-diastolic pressures. These parameters are expressed in terms of absolute changes because only minimal variations usually occur during a “resting” state and because small variations in their usually low numerical values frequently result in inordinately high percentage changes.

Changes in cardiovascular dynamics in the present series of 9 patients who received steroids were then evaluated in terms of statistical significance at the 5-per cent level. Changes that are considered to be statistically significant are appropriately indicated in tables 2A and 2B. It is appreciated that appraisals of the 2 sets of data in these subjects may not be strictly comparable with those of the control group because of the differences in time intervals. The possibility of greater variations in “resting” states may exist when studies are separated by greater time intervals. This factor does not appear to represent a major difficulty, however, since metabolic states could be studied and compared on the basis of oxygen consumptions and respiratory quotients.

Results

Three types of responses to steroid administration occurred in this series of patients:

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**The authors wish to thank The Squibb Institute for Medical Research, New Jersey, for their supply of triamcinolone, (Kenacort®).**

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*S. Time periods are denoted in this column. I, control observation period prior to initial physiologic studies; II, during the period of steroid administration (maximal changes and their time of occurrence are indicated); III, immediately prior to the repeat physiologic studies.

†Dyspnea. Severe if at rest, moderate if during mild effort, and slight if only during moderate effort.

‡Edema. Marked if involving more than lower extremities (presacral, abdominal wall), slight, if involving ankles.

AI, aortic insufficiency; AS, aortic stenosis; CHF, congestive heart failure; CP, cor pulmonale; E, emphysema; HHD, hypertensive heart disease; IMH, idiopathic myocardial hypertrophy; MI, mitral insufficiency; MS, mitral stenosis; PF, pulmonary fibrosis; RHD, rheumatic heart disease.
Table 2A
Cardiovascular and Pulmonary Dynamics before and at Termination of Steroid Therapy

<table>
<thead>
<tr>
<th>Patient</th>
<th>S*</th>
<th>Heart rate (beats/min.)</th>
<th>Stroke volume (ml./beat)</th>
<th>A-V O₂ difference (ml.)</th>
<th>O₂ consumption (ml./min./M²)</th>
<th>Respiratory quotient</th>
<th>Cardiac index (L./min./M²)</th>
<th>Systemic arterial O₂ (%)</th>
<th>Total blood volume (ml./M² BSA)</th>
<th>Total plasma volume (ml./M² BSA)</th>
<th>Predicted plasma volume (ml./M² BSA)</th>
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* S. Physiologic studies before (I) and at termination of steroid therapy (II). Results tabulated are those during resting states.
†Unsuccessful.
‡Cardiovascular dynamic changes that are statistically significant (≥2 standard deviations).
Table 2B
Cardiovascular and Pulmonary Dynamics before and at Termination of Steroid Therapy

<table>
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<tr>
<th>Patient</th>
<th>S*</th>
<th>Peripheral artery pressure (mm Hg)</th>
<th>Pulmonary artery pressure &quot;wedge&quot; (mm Hg)</th>
<th>Right ventricular end-diastolic pressure (mm Hg)</th>
<th>Resistance (Dynes sec. cm. -2)</th>
<th>Vital capacity (% predicted normal)</th>
<th>Maximum breathing capacity (% predicted normal)</th>
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*See legend, table 2A.
†Unsuccessful.
‡Cardiovascular dynamic changes that are statistically significant (>2 standard deviations).
§Right ventricular systolic pressure.
**Right atrial mean pressure.
Increases in congestive heart failure (5 patients), no significant changes in clinical states (3 patients), and improvement in clinical state and in cardiodynamics (1 patient).

**Increases in Congestive Heart Failure**

Clinical heart failure increased in severity in 5 (cases 1 to 5) of the 9 patients during steroid therapy (table 1). In 4 of these patients (cases 1 to 4) slight to moderate reductions in daily urine volumes occurred concomitant with increases in weight, indicating positive fluid balances. Laboratory data suggested worsening of hepatic function (increased bromsulfalein retention) and renal function (increased blood urea nitrogen and decreased urea clearance) in some patients of this group. All initially had normal 24-hour radioiodine uptakes (thyroid); no changes occurred during steroid therapy. Parenteral mercurial diuretics were given to 4 patients (cases 2 to 5) because it was considered hazardous to permit the deteriorated clinical conditions to persist. Responses to this therapy were poor.

Hyponatremia and hypochloremia existed in 2 patients in the control period, slight in one (case 1) and marked in the other (case 3). During steroid therapy slight depression of serum sodium occurred in 2 patients (cases 1 and 4) and of serum chloride in 2 (cases 1 and 3). In all 5 patients daily urine sodium and chloride excretions were markedly depressed in the control periods. During steroid therapy excretion of these ions did not change or decreased slightly in 4 patients, whereas an increase occurred in 1 (case 2).

At the time of the second physiologic studies, 1 patient (case 1) who did not receive mercurials had a weight gain of 6 pounds and considerable increase in heart failure, and another (case 2) was somewhat improved by mercurial therapy but was still worse than his control state as well as 2 pounds heavier. The others had resumed their control clinical states after mercurial therapy although 2 (cases 3 and 5) were 2 and 4 pounds lighter.

In 1 patient (case 3) the second catheterization was unsuccessful. In the others very few statistically significant changes in cardiodynamics occurred (tables 2A and 2B). This lack of change could be attributed to the appreciable neutralization of the clinically obvious detrimental effects of steroids by mercurial therapy, which was administered until clinical improvement was obtained.

Total plasma volumes were elevated initially in all 5 patients, but they did not change much after steroid therapy. Slight falls in plasma volumes in 3 patients may well have resulted from mercurial diuretics used to combat congestive failure. No significant changes in vital capacities and maximum breathing capacities occurred as a result of steroid administration. In 3 patients (cases 1 to 3) in whom fluid balances were measured for several days following steroid withdrawal, no significant changes occurred in weights, urine volumes, and urinary excretions of electrolytes.

**No Significant Changes in Clinical States**

In 3 patients (cases 6 to 8) no changes in clinical heart failure occurred during steroid administration (table 1). A weight loss of 5 pounds was noted in 1 patient (case 8). Marked anorexia and diminished food intake as well as a decline in urine output and in urinary sodium and chloride excretions suggested that this weight loss was due to a decrease in tissue mass rather than to a diuretic response. No changes occurred in laboratory data or in 24-hour radioiodine uptake by the thyroid gland. In 2 patients (cases 6 and 7) control 24-hour urine sodium and chloride excretions were within normal limits; in the other patient excretions of these ions were diminished. Moderate reductions in daily sodium and chloride excretions occurred in these 3 patients during steroid administration.

No significant changes in cardiodynamics occurred as a result of corticosteroid administration except for a decrease in right ventricular end-diastolic pressure and pulmonary "arteriolar" resistance in 1 patient (case 7) (tables 2A and 2B).

Total plasma volumes were elevated initially in all 3 patients. In 2 (cases 6 and 7)
slight to moderate decreases occurred during steroid therapy; in 1 (case 8) a moderate increase occurred.

No significant changes in vital capacities and maximum breathing capacities occurred as a result of steroid therapy. In 2 patients (cases 6 and 8) in whom fluid balances were studied for several days following steroid withdrawal, no changes occurred in body weights, urine volumes, and urine electrolyte excretions.

Improvement in Clinical State and in Cardiodynamics

In the patient (case 9) with pulmonary fibrosis and emphysema and cor pulmonale as the basic disease, there was an improvement in dyspnea during steroid therapy (table 1). However, a weight gain of 4 to 5 pounds and a slight decrease in daily urine output occurred concomitantly. There were no changes in routine laboratory data or in radioiodine uptake by the thyroid gland. A slight fall in serum chloride occurred.

Improvement in cardiodynamics resulted from administration of corticoids (tables 2A and 2B). There were a significant increase in cardiac output and lowered pulmonary artery systolic and mean pressures and right ventricular end-diastolic pressure. Although these changes in pressures may have been due to corresponding changes in intrathoracic pressure (which was not measured), the similarity of pulmonary "wedge" pressures in both studies suggests that the decreases in pulmonary artery and right ventricular pressures were due to decreases in effective intravascular pressures. Pulmonary vascular and pulmonary "arteriolar" resistances also decreased significantly. Oxygen consumption was higher during the second study and may have indicated some change in metabolic state. Nevertheless, the data indicate improvement in dynamics. Total plasma volume increased slightly with steroid therapy. Minimal, if any, improvement in vital capacity and maximum breathing capacity occurred.

No changes in fluid balance occurred following steroid withdrawal.

Discussion

The effects of ACTH and adrenal cortical steroids upon renal and cardiovascular systems in man and experimental animals have been far from uniform, so that generalization about the specific activities of these hormones is most difficult. The tendency of these hormones to promote salt and water retention and, consequently, an increase in extracellular fluid volume has been observed by many investigators.1-5, 26 Corticoids that have prominent actions of this type include desoxy-corticosterone, cortisone, hydrocortisone, and aldosterone. Newer synthetic derivatives have distinctly less tendency to cause salt and water retention although positive fluid balance has been observed, especially with higher dosages.21-25 In contrast, some studies demonstrate that these hormones may possess diuretic properties. Davis and Howell27 observed that both ACTH and cortisone produced an initial loss of salt and water in the intact dog. Other authors have reported natriuresis and chloruresis during the administration of ACTH in man28 and in the rat.29 Gaunt, Birnie, and Eversole30 cited experimental data indicating that cortical hormones may produce diuresis in animals with normal water balance, in overhydrated ones, and even in mildly dehydrated ones.

In view of this diversity of steroid actions, it is not difficult to understand the uncertainties regarding clinical applicabilities of these hormones in patients with diseases characterized by positive fluid balances, particularly congestive heart failure. This difficulty is further compounded by studies that have demonstrated increased activity of endogenous salt-retaining adrenal cortical hormone, aldosterone, in these diseases.7, 9-11, 31-33 In general corticoids have been used with caution in these conditions because of their tendency to promote salt and water retention. Various investigators, however, have reported favorable results following the administration of these hormones to patients with congestive heart failure.12-19 Results, however, were not uniform in all studies. Some patients had
clinical and hemodynamic deterioration probably due to renal retention of salt and water.\textsuperscript{12} In another study\textsuperscript{18} a small number of patients demonstrated decreased urinary sodium excretion, increase in weight, and possibly increase in dyspnea.

The present study was undertaken to elucidate the effects of adrenal corticoids in congestive heart failure. In the 5 of the 9 patients in this series subjective and objective clinical manifestations and laboratory data indicated positive fluid balances with increased fluid accumulation and increased cardiac decompensation during the phase of steroid therapy. Responses to mercurial diuretics were poor. Heightened responsiveness during steroid therapy was not observed in any patient in this study. An evaluation of cardiodynamic changes is more difficult because of the effects of additional diuretic measures given to 4 patients during steroid therapy. These agents were given because of the development of precarious clinical conditions. It is therefore not surprising that no significant changes in cardiodynamics and some decreases in plasma volumes were noted in those patients given mercurial diuretics until clinical improvement occurred. In 1 patient (case 2) who was clinically worse and had an increase in plasma volume (in spite of 2 mercurial injections) at the time of the second cardiac catheterization significant increases occurred in pulmonary artery and right ventricular end-diastolic pressures.

In 3 of the remaining 4 patients no changes in clinical status occurred during steroid therapy. Some diminution in urinary sodium and chloride excretions occurred in these patients although the depression present in the first group was not observed. One patient (case 8) had a reduction in circulating volume and a decline in right ventricular end-diastolic pressure. A minimal increase in daily urinary volume in this patient would not appear to account completely for the diminution in circulating volume although it could have contributed to it. Another possibility may be that of internal fluid shifts.

In 1 patient (case 9) clinical symptoms improved during steroid therapy. This was the only subject who had pulmonary fibrosis and emphysema with cor pulmonale. This patient had improvement in cardiodynamics despite a positive fluid balance. It would be difficult in this case to define the primary mode of action of the steroid from the limited amount of data available concerning primary pulmonary functions. Possibilities include improvement in pulmonary inflammation, bronchomotor tone, and vascular resistance, causing a decline in right heart work load and, consequently, improvement in right heart function. Other workers have described favorable clinical results and improvements in pulmonary function following corticosteroid therapy in patients with chronic pulmonary disease.\textsuperscript{34} This case is of interest in that clinical and hemodynamic improvement occurred only in that patient who did not have uncomplicated heart failure. Factors were probably present that are known to be favorably affected by corticosteroids, e.g., inflammation, and so forth. It is also probable that the additional fluid retained was distributed throughout the body compartments and any increase in fluid that might have occurred in the right heart was more than compensated by the favorable primary effect on the cardiopulmonary systems.

It is difficult to delineate those factors that may favor fluid retention during steroid therapy in some patients and not in others. The patients whose heart failure increased were those who generally seemed to have greater degrees of decompensation characterized by severe subjective manifestations, considerable fluid accumulation, and marked depressions in sodium and chloride excretions. Comparison of cardiovascular dynamics between the first 2 groups reveals much overlapping in many of the measurements. However, the generally higher pulmonary "wedge" and right ventricular end-diastolic pressures and total plasma volumes in those patients worsened by steroids suggest that deterioration is more likely to be produced in those patients who already have considerable fluid accumulation. The addition of agents that can retain
salt and water simply increases the severity of the existing positive fluid balance.

It is difficult to explain the differences in results in the present study from those reported by others, \textsuperscript{12-19} in which diuresis and clinical improvement were noted in many patients with congestive heart failure. Evaluation of the effects of prolonged administration of various drugs in heart failure may be hazardous in view of the spontaneous variations that may occur in this state. Strict selection of patient material is also important. Subjects who are in a steady clinical state are best studied; those who have had acute deterioration in cardiac function prior to drug administration may not be satisfactory subjects for this way suggest variable extraneous adverse factors which by their presence or remittance may influence results. Concomitant therapy that may be detrimental to subjects with heart failure should be avoided. One group of investigators maintained high levels of water intake before and during steroid administration.\textsuperscript{15-16} Leiter\textsuperscript{35} has reviewed data which indicate that subjects with congestive failure may retain water excessively (resembling the effects of excessive antidiuretic hormone), leading to a worsening of clinical status. Steroids may have prevented overhydration in these patients\textsuperscript{15, 16} as it does in water intoxication and as an antagonist to antidiuretic hormone\textsuperscript{30, 36, 37} and may not have affected any of the originally existing abnormal cardiovascular-renal hemodynamics.

Patients were selected for the present study who were in a steady clinical state and who had heart disease of varied etiologies and of varying severity and no other condition that could be favorably affected by steroids, especially infectious diseases and overt inflammatory processes. The one exception was the patient with pulmonary fibrosis and emphysema. The results in this patient demonstrate the need for care in patient selection. The phase of drug administration was restricted to 13 to 16 days to minimize the possibility of spontaneous change in disease states.

Further work with agents that more specifically antagonize endogenous fluid-retaining factors may prove fruitful in patients with congestive heart failure. The prominent role of endogenous adrenocortical hormones (especially aldosterone) in retaining fluid and in promoting formation of edema in patients with heart failure has recently been emphasized. Drugs that may antagonize these hormones would be most important.\textsuperscript{38}

**Summary**

This study is concerned with the effects of adrenocortical steroids in patients with congestive heart failure.

Nine adults with heart failure were studied during a steady clinical state (5 to 7 days), during administration for 13 to 16 days of prednisone (6 patients) or triamcinolone (3 patients) and in some subjects following steroid withdrawal. Daily determinations of fluid balances were made. Standard right heart catheterizations and measurements of blood volumes were performed prior to and at the termination of therapy.

Three types of responses to steroids occurred. In 5 patients increases in subjective and objective manifestations of heart failure and increased fluid retention occurred. Four of them developed precarious clinical conditions during steroid therapy, requiring mercurial diuretics, but with poor results. In general, cardiodynamic status at the termination of steroid therapy correlated well with clinical status. In 3 patients the clinical status did not change. Some depression in urinary sodium and chloride excretions were noted during therapy. Cardiodynamics were unaltered, except for an unexplained decrease in right ventricular end-diastolic pressure in 1 patient. In 1 patient clinical and cardiodynamic state improved, despite positive fluid balance. This was the only subject with primary lung disease (emphysema) and cor pulmonale.

These studies suggest that corticosteroids are generally detrimental in uncomplicated congestive heart failure. Greater deterioration appeared in subjects having the more severe degrees of decompensation, suggesting
that exogenous fluid-retaining influences were added to endogenous fluid-retaining forces, resulting in further accumulation of fluid in a circulatory system already burdened by hypervolemia.

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**Summario in Interlingua**

Iste studio es concernite con le effectus de steroids adrenocortical in patientes con congestive disfallimento cardine.

Novem adultos con disfallimento cardine esseva studiate in un stabile statu clinico (pro periodos de 5 a 7 dies), durante 13 a 16 dies de administracion de prednisona (6 patientes) o triameinolona (3 patientes), e in cete casos post le fin del curso de medication steroide. Eszeva effectuate determinaciones diuriae del balancias de liquido. Catheterismo dextere standard e mesuration del volumine de sanguine eseva effectuate ante le initiation del therapia e post su termination.

Occurreva tres typos de responsa al administration del steroids. In 5 patientes il habeva subjective e objective augmentos del manifestationes de disfallimento cardine e augmentos del retention de liquido. Quatro del cinque disveloppava precari conditiones clinice durante le therapia steroide. Illes requirove diureticos mercurial, sed le resultatos non esseva bon. A generalmente parlar, le stato cardiodynamic al termination del therapia steroide se mostrava ben correlationate con le stato clinico. In 3 patientes le stato clinico non se alterava. Un cete depression del excretion urinari de natrium e chlorouro esseva notate durante le therapia. Le cardiodynamic remaneva inalterate, con le exception del occurrentia d'un inexplicite reduction del tension dextero-ventricular termino-diastolice in 1 patiente. In 1 patiente le stato clinico e cardiodynamic se meliorava in despecto d'un positive balancia de liquido. Iste patiente esseva le sol con primari morbo pulmonar (emphysema) e corde pulmonal.

Le hie reportate studios suggere que corticosteroides es generalmente detrimentose in non complicate cases de congestive disfallimento cardine. In subjectos con plus sever grades de discompensation le deterioration esseva plus marcate. Isto pareva indicar que exogene influentias liquido-retentori esseva aditte, con le resultato d'un accumulation additional de liquido in un sistema circulatori jam suffrante de hypervolemia.

**References**


STEROIDS IN CONGESTIVE FAILURE


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