The Suprarenal Function in Allergic Asthma

Determination of the Plasma and Urinary 17-OH-Corticoids and of the Urinary 17-Ketosteroids Before and After ACTH Zn.*

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Introduction

The connection between suprarenal function and allergic diseases has been the subject of some investigation. In 1922, Kenipow found that anaphylactic reactions were increased in guinea pigs whose suprarenal glands had been partially removed. Later Eriksson-Lihr et al. found that the daily excretion of the 17-ketosteroids was decreased in allergic diseases; these observations were subsequently confirmed by other authors. Rose and his colleagues have studied the daily excretion of the 17-OH-corticoids in patients during the dyspneic crisis and they have found it decreased. Siegel et al. have determined 17-OH-corticoids of the plasma in asthmatic patients and they have found increased values, particularly in patients with intense symptoms.

In previous works, we have studied the blood proteins and electrolytes in 40 patients with respiratory allergy, most of them asthmatic. We found a decrease of sodium and plasma chlorine and an increase of potassium and globular chlorine; these variations were highly significant from the point of view of statistics and they were similar to those observable in suprarenal insufficiency. Subsequently we have carried out with the same type of patients the determination of the urinary 17-ketosteroids and the eosinophil counts before and 24 hours after intramuscular injection of 80 units of ACTH gel. We found initially low values of 17-ketosteroids and a highly significant decrease (P=0.001) of the response to the ACTH. These findings have led us to a thorough study of the suprarenal function in allergic asthma.

Material and Methods

The intramuscular ACTH Zn test, according to the original method of Jenkins et al., was done on 22 adults, 11 women and 11 men, whose asthma was of definite allergic origin. A total of 40 units of slow-acting corticotrophine was administered.

Urine specimens were carefully collected 24 hours before and after the administration of ACTH and the plasma 17-OH-korticoids were determined according to the technique of Saier et al., the urinary 17-OH-corticoids by the method of Silber and Porter and the urinary 17-ketosteroids by the method of Cahen and Salter.

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The patients were previously studied from the allergic point of view by means of skin tests; most of them were shown to be sensitive to house dust and to fungi.

The patients were examined clinically before and after the administration of corticotrophine in order to appraise the intensity of symptoms and they were classed in three groups: (1) asymptomatic patients, seven cases; (2) patients with slight symptoms (nasal obstruction, faint sibilant rales), ten cases; (3) patients with intense symptoms (subjective dyspnea, abundant sibilant rales, and increased respiratory frequency), five cases.

In order to avoid all disturbing influence on the test, patients were chosen who had never taken corticoids.

In order to suppress hourly variations in the level of the plasma 17-OH-corticoids, all the blood counts were taken at the same time, that is at 8 a.m., the moment when the corticoids reach their highest level; this is the time at which most authors made determinations.

Results

Urinary 17-ketosteroids: The excretion of the 17-ketosteroids 24 hours before administration of ACTH showed decreased values. The average obtained in men was 9.6 standard deviation ±2.8 mg./24h. and in women, 7.1 standard deviation ± 2.8 mg./24h.; these are low values compared with those given by de Gennes,7 in whose Department the determinations were carried out, and who considers as normal values for a male adult from 8 to 22 mg. per day with an average of 14 mg./24h. and for women from 5 to 15 mg./24h. with an average of 9 mg./24h. On their part, Cahen and Salter,2 authors of the chemical method used, also gave figures

<table>
<thead>
<tr>
<th>TABLE 1—VALUES OF PLASMA AND URINARY CORTICOIDS OBTAINED IN MALE PATIENTS WITH RESPIRATORY ALLERGIES</th>
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<tbody>
<tr>
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<td></td>
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<tr>
<td>Men</td>
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<tr>
<td></td>
</tr>
<tr>
<td>F.A.</td>
</tr>
<tr>
<td>P.E.</td>
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<td>H. M.</td>
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<tr>
<td>CH. E.</td>
</tr>
<tr>
<td>C.R.</td>
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<tr>
<td>G.L.</td>
</tr>
<tr>
<td>M.R.</td>
</tr>
<tr>
<td>A.R.</td>
</tr>
<tr>
<td>L.M.</td>
</tr>
<tr>
<td>F.J.</td>
</tr>
<tr>
<td>S.A.</td>
</tr>
<tr>
<td>Average</td>
</tr>
<tr>
<td>Standard Deviation</td>
</tr>
<tr>
<td>Standard Error</td>
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</tbody>
</table>

References: W.S. = Without symptoms; S.S. = Slight symptoms; I.S. = Intense symptoms.
higher than those we found in asthmatic patients; they give from 13 to 
19 mg./24 h. for normal men and from 9 to 16 mg./24 h. for women.

More important than the low elimination of the 17-ketosteroids was the absence of response to the administration of ACTH. Indeed, the average values for men were the same after the administration of ACTH; 9.6 standard deviation ± 3.2 mg./24 h.; and for women, the rise was very slight: from 7.11 standard deviation ± 2.8 mg./24 h. to 8.0 standard deviation ± 2.4 mg./24 h., that is to say an average of 12 per cent. De Gennes' considers that ACTH raises regularly the proportion of these androgens, twice their value (100 per cent), and that the Thorn test is not normal if the 17-ketosteroids do not reach at least 50 per cent (an average of 5 mg.). Jenkins et al.,' authors of the method, have observed an average increase of 6 mg. in normal subjects with the intramuscular injection of ACTH gel.

17-Hydroxycorticoids. The urinary elimination of the 17-OH-corticoids before administration of ACTH was an average of 5.1 standard deviation ± 1.7 mg./24 h. in men sufferers from respiratory allergies. (Values in normal men: 4 to 8 mg./24 h.).

In women the average excretion was from 3.4 standard deviation ± 1.4 mg./24 h. (normal values: 2 to 5 mg./24 h.). Thus it is clear that the static elimination of 17-OH-corticoids was maintained within normal limits.

The increase induced by the injection of ACTH in men was from 5.1 standard deviation ± 1.7 to 11.4 standard deviation ± 7.7 mg./24 h., that is to say of 123 per cent. In women the increase was of 167 per cent, the figures increased from 3.4 standard deviation ± 1.4 to 8.0 standard deviation ± 3.3 mg./24 h. De Gennes' considers that in normal circumstances the 17-OH-corticoids increase 300 per cent.

One can observe that the basal excretion was normal but that the increase by the action of ACTH was diminished.

### TABLE 2—VALUES OF PLASMA AND URINARY CORTICOIDs

<table>
<thead>
<tr>
<th>Woman</th>
<th>Age</th>
<th>17-CT Urinary mg./24h.</th>
<th>17-OH Urinary mg./24h.</th>
<th>17-OH Plasma Urinary mg./24h.</th>
<th>Symptomaticity</th>
<th>17-CT Urinary mg./24h.</th>
<th>17-OH Urinary mg./24h.</th>
<th>17-OH Plasma Urinary mg./24h.</th>
<th>17-OH Plasma 24th. h.</th>
<th>Symptomaticity</th>
</tr>
</thead>
<tbody>
<tr>
<td>L.M.</td>
<td>63</td>
<td>5.8</td>
<td>1.0</td>
<td>9</td>
<td>S.S.</td>
<td>8.2</td>
<td>8.0</td>
<td>11</td>
<td>S.S.</td>
<td></td>
</tr>
<tr>
<td>S.M.</td>
<td>41</td>
<td>8.5</td>
<td>3.7</td>
<td>17</td>
<td>S.S.</td>
<td>8.8</td>
<td>11.4</td>
<td>25</td>
<td>W.S.</td>
<td></td>
</tr>
<tr>
<td>G.O.</td>
<td>64</td>
<td>4.7</td>
<td>4.3</td>
<td>I.S.</td>
<td>6.6</td>
<td>10.4</td>
<td>I.S.</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>S.Q.</td>
<td>38</td>
<td>8.1</td>
<td>4.2</td>
<td>27</td>
<td>W.S.</td>
<td>9.1</td>
<td>12.7</td>
<td>38</td>
<td>W.S.</td>
<td></td>
</tr>
<tr>
<td>H.M.</td>
<td>58</td>
<td>5.2</td>
<td>2.8</td>
<td>41</td>
<td>W.S.</td>
<td>4.3</td>
<td>6.2</td>
<td>31</td>
<td>W.S.</td>
<td></td>
</tr>
<tr>
<td>A.M.</td>
<td>54</td>
<td>4.4</td>
<td>3.4</td>
<td>26</td>
<td>S.S.</td>
<td>5.2</td>
<td>4.5</td>
<td>28</td>
<td>S.S.</td>
<td></td>
</tr>
<tr>
<td>U.I.</td>
<td>35</td>
<td>11.2</td>
<td>4.9</td>
<td>38</td>
<td>W.S.</td>
<td>12.2</td>
<td>12.1</td>
<td>25</td>
<td>W.S.</td>
<td></td>
</tr>
<tr>
<td>P.S.</td>
<td>43</td>
<td>10.1</td>
<td>4.1</td>
<td></td>
<td>W.S.</td>
<td>8.7</td>
<td>5.2</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>L.R.</td>
<td>53</td>
<td>3.9</td>
<td>1.5</td>
<td>S.S.</td>
<td>6.2</td>
<td>6.7</td>
<td>I.S.</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>D.J.</td>
<td>31</td>
<td>8.1</td>
<td>5.3</td>
<td>24</td>
<td>S.S.</td>
<td>10.4</td>
<td>3.5</td>
<td>17</td>
<td>W.S.</td>
<td></td>
</tr>
<tr>
<td>A.L.</td>
<td>40</td>
<td>8.2</td>
<td>3.1</td>
<td>27</td>
<td>I.S.</td>
<td>6.2</td>
<td>6.7</td>
<td>I.S.</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

Average 46 7.1 3.4 26.1 8.0 8.0 25
Standard
Deviation ±2.8 ±1.4 ±9.7 ±2.4 ±3.3 ±9.0
Standard Error ±0.9 ±0.4 ±3.4 ±0.7 ±1.0 ±3.7

References: W.S. = without symptoms; S.S. = slight symptoms; I.S. = intense symptoms.
**Plasma 17-hydroxycorticoids.** The values of the plasma 17-OH-corticoids found in asthmatic patients at 8 a.m., before injection of ACTH, gave increased values, the average being from 20.7 standard deviation ± 10.4 μg. per cent in men and from 26.1 standard deviation ± 9.7 μg. per cent in women.

Silber and Porter" found an average of 13.3 standard deviation ± 6.2 μg. per cent in six normal adults. Eik-Nes et al." noted in normal subjects before ACTH, an average of 10 standard deviation ± 3 μg. per cent; two hours after the plasma level rose to 27 sd ± 7 μg. per cent; four hours later to 35 sd ± 10 μg. per cent and after six hours to 40 μg. ± 12 per cent.

On their part, Christy et al." found in 11 normal subjects an average of 13 μg. per cent before ACTH and of 44 μg. per cent after ACTH. All these results are noticeably lower than those we have found in our patients.

With regard to the action of ACTH Zn on the plasma levels, we have observed slight variation in the values 24 hours after the intramuscular injection, since a slight increase of 20.7 standard deviation ± 10.4 μg. per cent to 23.2 standard deviation ± 10.1 μg. per cent was noted in men, and in women a slight decrease of 26 standard deviation ± 9.7 μg. per cent to 25 standard deviation ± 9.0 μg. per cent.

We have also carried out on three patients blood counts eight hours after the administration of corticotrophine; in two cases we observed an increase of 17 and 15 μg. per cent to 25 and 25 μg. per cent respectively; in the third case the rise was slight, from 6 μg. per cent to 7 μg. per cent before and after the ACTH.

Even in the two cases where a more marked increase of the plasma 17-OH-corticoids was observed, this increase was lower than that established by Geller et al." who found rises of from 15 μg. per cent to 35 μg. per cent, on average, eight hours after the intramuscular injection of ACTH. The same authors have carried out determinations of plasma 17-OH-corticoids every two hours, after intramuscular injection of 40 units of ACTH, in the two forms, Zn and gel.

From the comparative study of the two preparations of corticotrophine they conclude that the highest point is reached two hours after injection of corticotrophine in both forms, while in the gel form it decreases rapidly. On the contrary, with ACTH Zn (which was used in our work) the high level is maintained for up to eight hours, subsequently decreasing progressively. After 24 hours the levels of the plasma corticosteroids are observed to be close to the level before the ACTH.

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**TABLE 3—COMPARATIVE TABLE OF THE 17-KETOSTEROIDS AND THE 17-OH-CORTICOIDS ACCORDING TO THE SYMPTOMATOLOGY OF PATIENTS**

<table>
<thead>
<tr>
<th>Patients without Symptoms (7 cases) Average</th>
<th>Before ACTH (7-CT Urinary)</th>
<th>Before ACTH (17-OH Urinary)</th>
<th>Before ACTH (17-OH Plasma)</th>
<th>After ACTH (7-CT Urinary)</th>
<th>After ACTH (17-OH Urinary)</th>
<th>After ACTH (17-OH Plasma)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Average</td>
<td>8.1</td>
<td>4.8</td>
<td>31</td>
<td>8.3</td>
<td>8.4</td>
<td>25</td>
</tr>
<tr>
<td>Patients with Slight Symptoms (10 cases) Average</td>
<td>8.9</td>
<td>4.0</td>
<td>19</td>
<td>9.3</td>
<td>9.6</td>
<td>21</td>
</tr>
<tr>
<td>Average</td>
<td>7.9</td>
<td>5.4</td>
<td>17</td>
<td>8.0</td>
<td>12.1</td>
<td>23</td>
</tr>
</tbody>
</table>
These authors have shown, in normal subjects, an increase of from two to three times the percentage of the plasma 17-OH-corticoids, after injection of 40 units of ACTH Zn. They conclude that these values eight hours after the administration of slow-acting corticotrophine are the same as those found by Bayliss' and Elk-Nes' who used the intravenous method, and that the effect produced by 40 units of ACTH Zn is similar to 20 or 25 units of intravenous ACTH.

With regard to the connection between the plasma levels and the intensity of the symptoms, we have observed that asymptomatic patients showed higher plasma levels: 31 μg. per cent on average, and patients with slight and intense symptoms, 19 and 17 μg. per cent, respectively.

**Discussion**

We shall now consider the state of the suprarenal function in patients suffering from respiratory allergies, with reference to three functions of the suprarenal cortex, the androgenic function, the glycemic function and the mineral function.

The androgenic function was studied by determining the urinary 17-ketosteroids before and after administration of corticotrophine, showing values lower than normal in both sexes. We have arrived at the same results in a previous work9 carried out on 33 sufferers from respiratory allergy.

The glycemic function was studied by determining the urinary 17-OH-corticoids and by the study of the plasma levels of the glycocorticoids.

The exact figures of the 17-OH-corticoids were found within normal limits, but the increases after ACTH were lower than 300 per cent; in asthmatic men the increase was 123 per cent and in the women, 167 per cent.

Recently Haydar et al. have published seven cases of suprarenal inadequacy with a rise in these, under the action of ACTH, lower than that observed in normal subjects. In these patients, the static 17-ketosteroids were normal, but no rise was produced by the action of the ACTH. The authors consider these as cases of a partial or compensated suprarenal inadequacy. The adrenal function is sufficient for normal needs, but incapable of increasing the secretion of corticosteroids in case of stress or by stimulation with corticotrophine.

Pedersen and Sondergaard have also published a similar case which they called partial Addison's disease with normal excretion of 17-OH-corticoids and absence of response to ACTH by the 17-ketosteroids. De Gennes et al. have observed ten similar cases and they have called them subclinical or formes frustes.

Judging from the conclusions of the works quoted above which coincide with the results we have obtained with our patients, it would seem that the androgenic function represented by the 17-ketosteroids is more easily disturbed in cases of subclinical insufficiency than the glycemic function, which maintains the values of 17-OH-corticoids within normal limits.

The high levels of the plasma 17-hydroxy corticoids found in our patients could be explained by the need for hydrocortisone and cortisone to attenuate the allergic reaction present in this type of patient. The antiallergic action of the glycocorticoids is manifested by different mechanisms, by its inhibitory action of the formation of antibodies and probably also by its stimulating action on the cholinesterase activity, which we have proved in a previous work. and in a symptomatic way by its anti-inflammatory and anti-edema action. However, the glycocorticoids do not exert a direct antihistaminic action. This action explains, in some measure, why patients who show higher plasma levels are precisely those who have no symptoms; on the other hand, in allergic patients studied with both slight and intense symptoms, the plasma levels of the 17-OH-corticoids are lower, thus permitting the appearance of symptoms.

As for the effect of ACTH Zn on the proportion of plasma glyco corticoids 24 hours after intramuscular injection, it has not been perceptibly modified because, as Geller et al. have shown, the increase induced by the corticotrophine Zn becomes progressively less pronounced after the eight hours until it reaches levels approximately equal to the initial levels 24 hours after the injection.

In the three cases where determinations of the plasma glyco corticoids were done eight hours after the injection, an absence of response to ACTH was shown in one case, and in the two others a response lower than that found by other authors in normal subjects.

With regard to the efficacy of slow-acting ACTH given intramuscularly to stimulate the suprarenal, clinicians have shown that the action of the corticotrophine, both intravenous and intramuscular, produces comparable effects. Geller et al. have compared the plasma levels of the 17-OH-corticoids eight hours after stimulation with 40 units of ACTH Zn and they have found them comparable to the plasma levels obtained with 20 to 25 units given intravenously.
The mineral function was indirectly studied in a previous work\textsuperscript{16,17} carried out on 40 sufferers from respiratory allergies and we noted a highly significant decrease of sodium and plasma chlorine and an equally significant increase of potassium and globular chlorine, with a decrease of the ratio Na/K as was observed in the suprarenal hypofunction.

These abnormalities could be explained in the following way: the suprarenal of the asthmatic patient, obliged to maintain a high plasma level of glyocorticoids in order to brake the allergic reaction, prefers to produce these hormones to the detriment of the androgenic and mineral function.

Moreover, it is interesting to observe that in allergic patients without symptoms, at the time of the blood determination, we have found the proportion of the plasma 17-hydroxycorticoids comparatively higher than what we have observed in allergic patients with slight and pronounced symptoms. This leads us to think that the stress, in the form of asthma, succeeds not in increasing the plasma glyocorticoids, but on the contrary in decreasing them, probably because the gland has reached the limit of its possibilities of supporting the stress.

Another difficult problem to solve is to know whether the suprarenal hypofunction is primary or secondary to allergy; that is to say, whether the abnormal tendency of certain subjects to acquire diverse sensitizations is due to a congenital dysfunction of the suprarenal gland or if on the contrary the anomalies observed in its function are secondary to the permanent stress represented by the allergic reaction which leads to a progressive weakening of the gland, especially in the androgenic function and probably in the mineral function.

SUMMARY AND CONCLUSIONS

In 22 adult asthmatic patients of definite allergic origin who had never received corticosteroid treatment, the adrenal function was studied by means of the 24-hour test with ACTH Zn given by intramuscular injection. The urinary 17-ketosteroids, the urinary 17-hydroxycorticoids, and the plasma 17-hydroxycorticoids were studied.

Elimination of 17-ketosteroids was found to be diminished before the administration of ACTH. After the ACTH, the increase of the 17-ketosteroids was practically non-existent (+ 6 per cent).

The basal elimination of the 17-hydroxycorticoids was maintained within normal limits. However, the increase after stimulation was less (+ 145 per cent), than that which occurs with normal subjects (+300 per cent).

The proportion of plasma 17-hydroxycorticoids was found higher than the normal proportion. This manifested in inverse ratio to the intensity of symptoms.

The increase of the plasma corticoids eight hours after stimulation with ACTH was found to be diminished.

From these facts it is inferred that in the case of allergic asthma, there is a disturbance which can be classed among the subclinical adrenal insufficiencies, also called partial or compensated adrenal insufficiencies.

ACKNOWLEDGEMENTS: To Prof. L. de Gennes for having permitted the carrying out of this study in his department; to Profs. J. Turlaf and B. Halber and Dr. J. Schaffer who gave us the necessary clinical material; to Dr. M. H. Laudat and to Mme. D. Laurent for their technical collaboration.

RESUMEN

Se estudió la función suprarrenal en 22 adultos asmáticos de origen claramente alérgico que nunca habían recibido tratamiento por cortico-esteroide, usando la prueba de las 24 horas con ACTH Zn por vía intramuscular. Fueron estudiados los 17-cetosteroides, urinarios, los 17-hidroxicorticoides urinarios y los 17-hidroxicorticoides plasmáticos.

Se encontró que la eliminación de 17 cetosteroides estaba disminuida antes de la administración del ACTH. Después del ACTH el aumento de los 17 cetosteroides prácticamente no existía (+ 6 por ciento).

La eliminación basal de los 17 hidroxicorticoides se mantuvo dentro de límites normales. Sin embargo, el aumento después de la estimulación fue menor (+145 por ciento) que el que ocurre en los sujetos normales (+300 por ciento).

La proporción en el plasma de 17-hidroxicorticoides se encontró más alta que lo normal. Esto se manifestó en razón inversa de la intensidad de los síntomas.

El aumento de los corticoides plasmáticos ocho horas después del estimulo con ACTH, se encontró disminuido.

Se infiere de estos hechos que en el asma alérgico hay un trastorno que puede clasificarse entre las insuficiencias suprarrenales subclínicas llamadas también insuficiencias parciales o compensadas de la suprarrenal.

ZUSAMMENFASSUNG

Be 22 erwachsenen Kranken mit Asthma nachgewiesen allergischen Ursprungs, die zuvor niemals mit Corticoiden behandelt worden waren, wurde die Nebennierenfunktion mittels des 24-Stundentestes mit intramuskulär gegebenem ACTH
geprüft. Untersucht wurden die Urinausscheidungen von 17-Ketosteroiden und 17-
Hydroxycorticoiden sowie die Plasmawerte von 17-Hydroxycorticoiden.

Es erwies sich die Ausscheidung von 17-Ketosteroiden herabgesetzt vor der Verab-
folgung von ACTH. Nach ACTH war der Anstieg der 17-Ketosteroid praktisch
unerheblich (+ 6%).

Die Basis-Ausscheidung der 17-Hydroxycorticoiden blieb innerhalb normaler Grenzen
aufrechterhalten. Jedoch betrug die Zunahme nach der Reizung weniger (+145%) als
die bei normalen Personen auftretende (+300%).

Der Anteil der 17-Hydroxycorticoiden im Plasma lag höher als im Normalfall. Darin
kam ein umgekehrtes Verhältnis zur Intensität der Symptome zum Ausdruck. Weiter
zeigte sich die Vermehrung der Plasmascorticoid 8 Stunden nach der Reizung mit
ACTH herabgesetzt.

Aus diesen Beobachtungen wird der Schluß gezogen, daß im Falle einer allergischen
Asthmas eine Störung vorliegt, die eingereiht werden kann unter die klinisch unter-
schwelligen Nebenniereninsuffizienzen, auch partielle oder kompensierte Nebennieren-
insuffizienzen genannt.

REFERENCES
1 Bayliss, R. I., and Steinbeck, A. W.: “The Adrenal Response to Corticotrophine
2 Cahen, R. L., and Salter, W. T.: “Urinary 17-Ketosteroids in Metabolism: Stand-
3 Christy, M. P., Wallace, E. Z., and Jaller, J. W.: “The Effect of Intravenously Ad-
ministered ACTH on Plasma 17-21-dihydroxy-20-ketosteroids in Normal Individu-
als and in Patients with Disorders of the Adrenal Cortex,” J. Clin. Invest., 34:899,
1955.
“Changes in Plasma Levels of 17-hydroxycorticoids During the Intravenous Ad-
1957.
7 Gennes, L. de, Bricaire, H., and Moreau, L.: “Les Insuffisances Surrénales Intra-
8 Haydar, N. A., St. Marc, J. R., Reddy, W. J., Laidlaw, J. C., and Thorn, G. W.:
“Adrenocortical Insufficiency with Normal Basal Levels of Urinary 17-hydroxycor-
9 Jenkins, D., Forsham, P. H., Laidlaw, J. C., Reddy, W. J., and Thorn, G. W.: “Use of
11 Rose, B., Fyles, T. W., and Verning, E. H.: “Corticotrophin, Cortisone and Hydro-
cortisone in Diseases of Hypersensitivity,” J. Allergy, 26:1, 1955.
for the Simultaneous Determination of 17-ketosteroids Dehydroepiandrosterone
J. Allergy, 8:94, 1956.
14 Silber, R. H., and Porter, C. C.: “The Determination of 17-21-dihydroxy-20-keto-
16 Vaccarezza, J. R., Bonacossa Torrent, R. J., and Wilson, J. A.: “Estudio de los
Allergy, 18:961, 1960.
18 Vaccarezza, J. R., and Peltz, L.: “Action de la Corticotrophine sur l’activité Coli-
nestérasique Sanguine Chez les Sujets Normaux et Chez les Malades Allergiques
con Gel de Corticotrofina en los Enfermos Alérgicos Respiratorios,” Prensa Médica
20 Vaccarezza, J. R.: La Fonction Surrénale dans L’asthme Allergique Mémoire pour
le Titre D’Assistant Étranger, Faculté de Médecine de Paris, 8 July, 1960.