ADRENOCORTICOSTEROID THERAPY AND DOCUMENTED NURSING ASSESSMENT: A COMPARISON

A THESIS

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We hereby recommend that the thesis prepared under our supervision by Christine Seftchick Houde, R.N., B.S.N. entitled Adrenocorticosteroid Therapy and Documented Nursing Assessment: A Comparison

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CHAPTER I

INTRODUCTION

Over the past several years medical knowledge has grown at an astonishing rate. This rapid expansion of knowledge has imposed tremendous demands on all members of the health team to keep informed of new developments. This is essential in order to provide the consumer with the most medically current and sound care available.

The health care professionals have recognized the need to improve ways of absorbing and utilizing the vast amount of new knowledge. As one resolution to the problem, the health care professions have created specialists who function primarily in a concentrated area of expertise. The development of adequate educational programs in each discipline provides another possible answer to the problem at hand. Much concern and controversy exists in most of the health disciplines in regard to how its material should be updated, what should be deleted from the program and how long the program should be to thoroughly cover the material.

The ultimate responsibility for producing well prepared practitioners in each discipline lies primarily within that discipline. Practitioners of nursing as well as nurse educators must share the responsibility to plan

and develop nursing programs which will produce nurses who are well prepared to deliver nursing care within a dynamic health care delivery system. At the same time, the practitioner of nursing who has been out of school for a number of years must make a conscientious effort to keep informed of current advances in health care. The importance of attempting to stay current with medical trends becomes particularly evident when one realizes that it is the nurse who is with the patient on a twenty-four hour basis and is responsible for him. This factor in itself demands that the nurse be well informed of the various aspects of the patient's care. This knowledge is imperative if the nurse is to provide safe patient care while executing the broad spectrum of treatments, medication administration, and other nursing duties which she is expected to carry out with skill and understanding.

The advent of new drugs to the medical scene brings with it specific implications for practitioners of nursing as well as nurse educators. It is the nurse's responsibility to be aware of why a particular drug was ordered for a particular patient, the drug's basic pharmacological actions, side-effects, proper administration technique, and various nursing implications which result from the drug's overall effects on the patient. It is this background knowledge which

provides a basis from which the nurse can make necessary nursing assessments.

The adrenocorticosteroids are a group of drugs which is a relatively new addition to frequently prescribed medications. These drugs have extremely complicated physiological effects and are potentially dangerous. It is the nurse's responsibility to closely monitor the effects of these drugs and to therefore avoid any potentially dangerous side-effects. The question arises, exactly how knowledgeable is the nurse in adrenocorticosteroid physiology, side-effects, nursing implications and administration techniques, and, furthermore, is this level of knowledge reflected in the nursing assessments which are made on patients receiving an adrenocorticosteroid drug?

Statement of Problem

The problem of this study was to determine whether or not there is a relationship between nurses' knowledge in adrenocorticosteroid physiology, side-effects, nursing implications and administration techniques, and the documented nursing assessments which are made on patients receiving an adrenocorticosteroid drug.

Statement of Purposes

The purposes of this study were:

1. To determine what the nurses' level of knowledge was

in adrenocorticosteroid physiology, side-effects, nursing implications and administration techniques

2. To determine what kind of documented nursing assessments were made on patients receiving adrenocorticosteroid therapy
3. To determine whether or not there was a relationship between nurses' knowledge in adrenocorticosteroid physiology, side-effects, nursing implications and administration techniques, and the documented nursing assessments which were made on patients receiving an adrenocorticosteroid drug

Background and Significance

The adrenocorticosteroids are one of the most frequently prescribed drugs in the United States.[1] The adrenocorticosteroids consist of three groups of steroids known as the glucocorticoids, the mineralocorticoids, and the adrenal androgens. The primary action of the glucocorticoids is to regulate fat, carbohydrate, and protein metabolism, while the mineralocorticoids act to primarily regulate sodium-potassium metabolism.[2] The androgens are

[1]John E. Bethune, The Adrenal Cortex (Michigan: The Upjohn Company, 1974), p. 31.

[2]David P. Lauler, Gordon H. Williams, and George W. Thorn, "Disease of the Adrenal Cortex," in Harrison's Principles of Internal Medicine, 6th ed., ed. Maxwell M. Wintrobe et al. (New York: McGraw-Hill Company, 1970), p. 485.

sex hormones which are anabolic in their effects and have the primary action of stimulating protein synthesis.[1]

All three groups of the adrenocorticosteroids have been synthesized to yield a relatively pure form of the desired steroid group. However, many of the steroid drugs frequently used today have both glucocorticoid and mineralocorticoid effects with one of the two compounds having the dominant effect, depending on the formula of the particular drug being used.[2] The glucocorticoid group is the most frequently prescribed group of the three corticosteroids primarily because of its anti-inflammatory effect.[3] While attempting to achieve the anti-inflammatory effect of the glucocorticoids, numerous undesirable side-effects may also occur. Many body systems such as the musculoskeletal, gastrointestinal, neurological, endocrine, dermatologic,and others may be involved. The side-effects are quite diverse and range from sodium and fluid retention to impaired wound healing and other more severe effects.[4]

[1]David P. Lauler, Gordon H. Williams, and George W. Thorn, "Disease of the Adrenal Cortex," in Harrison's Principles of Internal Medicine, 6th ed., ed. Maxwell M. Wintrobe et al. (New York: McGraw-Hill Company, 1970), p. 485.

[2]John E. Bethune, The Adrenal Cortex (Michigan: The Upjohn Company, 1974), p. 39.

[3]Ibid., p. 31.

[4]Profiles of Three Corticosteroids (Pennsylvania: Division of Merck and Company, Incorporated, 1972), pp. 17-20.

Therefore, prior to initiating steroid therapy, careful consideration must be given to determine whether or not the beneficial effects of the drug will outweigh the undesirable side-effects.[1]

Implications for nurses are derived from the therapeutic effects and the many side-effects of adrenocorticosteroid therapy. It becomes the nurse's responsibility to be aware of what the pharmacological effects and side-effects are and how to therefore assess and monitor a patient who is receiving a corticosteroid drug.[2] Other important nursing duties are the proper administration of these drugs, patient education in regard to the drugs' effects, and prevention of complications.[3]

In discussing the many responsibilities of the nurse in relation to corticosteroid therapy, the question arises, does the nurse have an adequate level of knowledge to assume these responsibilities? Studies related to nursing and nursing education have been done as early

[1]John E. Bethune, The Adrenal Cortex (Michigan: The Upjohn Company, 1974), p. 39.

[2]Laura Govoni and Janice Hayes, Drugs and Nursing Implications, 2nd ed. (New York: Appleton-Century-Crofts, 1971), pp. 80-83.

[3]Lillian Sholtis Brunner et al., The Lippincott Manual of Nursing Practice (Philadelphia: J. B. Lippincott Company, 1974), pp. 666-672.

as 1923. In the many studies that have been conducted emphasis was placed on the need for more and better prepared nurses.[1] Many efforts have been directed to developing curricula which will meet these needs. The past trend in nursing education was to produce "rule-oriented" nurses, while today this trend is slowly being replaced by "knowledge-oriented" nurses.[2] Nurse educators are being challenged to teach students that knowledge utilization is part of nursing practice and is directly linked to the nursing process.[3] The emergence of knowledgeable, well prepared nurses is becoming an ever increasing concern of the nursing profession.

Hypothesis

The hypothesis that was tested in this study was that there would be no relationship between nurses' scores on the test portion of the questionnaire and the patient-assessment scores earned by the nurses on the chart checklist and the patient interview checklist.

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[1]Lucille Wood, "Proposal: A Career Plan for Nursing," American Journal of Nursing 73 (May 1973):832.

[2]Carrie B. Lenburg, "Nursing and Education," R. N. Magazine 39 (March 1976):22.

[3]Ibid.

Definition of Terms

The definition of terms that were used in this study are as follows:

1. Administration technique is the manner in which a drug is given
2. An adrenocorticosteroid is any compound which has either a glucocorticoid effect, a mineralocorticoid effect, or a combined effect of both a glucocorticoid and a mineralocorticoid
3. Documented information is that which is recorded on a patient's permanent chart
4. A nurse is anyone who is currently licensed to practice professional nursing
5. Nursing assessment is the process of analyzing collected data to identify nursing problems and needs[1]
6. Nursing implications are those responsibilities which are assumed by nurses as pertinent to patient care
7. The pharmacology of a drug is its effects on the human organism's normal functioning processes
8. A side-effect is an adverse effect which is produced by a drug

[1]Eileen Pearlman Becknell and Dorothy M. Smith, System of Nursing Practice (Philadelphia: F. A. Davis Company, 1975), p. 67.

Limitations

The limitations of this study were:

1. The willingness of the nurses to spend time answering the questionnaire
2. The nurses' individual motivation to stay current with new medical knowledge and drugs
3. The hospital's charting policies
4. The time available to nurses for charting

Delimitations

The delimitations of this study were as follows:

1. Nurses who are currently licensed to practice professional nursing
2. Nurses who are working at least forty hours a week on the unit being sampled
3. Patients who are receiving a form of an adrenocorticosteroid for at least the past twenty-four hours, but no longer than three weeks on a schedule of at least twice a day
4. Patients who are administered an adrenocorticosteroid drug by the oral, intramuscular, or intravenous routes
5. Patients who are eighteen years of age or older
6. Patients who are mentally and physically capable of answering questions

Assumptions

The assumptions that were applicable to this study are:

1. Once learning has taken place, the retained knowledge may be applied or used in a situation when indicated
2. An individual's knowledge which is pertinent to his profession will be reflected in his professional practice

Summary

The adrenocorticosteroids are one of the most frequently prescribed drugs in the United States. These drugs have extremely complicated physiological effects and are potentially dangerous. It is the nurse's responsibility to closely monitor the effects of these drugs and to therefore avoid any potentially dangerous side-effects. The problem under study was to determine whether or not a relationship existed between the nurses' knowledge in adrenocorticosteroid physiology, side-effects, nursing implications and administration techniques, and the documented nursing assessments which are made on patients receiving an adrenocorticosteroid drug.

Chapter II contains a review of literature. Glucocorticoid, mineralocorticoid, androgen, ACTH, and renin-angiotensin physiology is discussed. Adrenocorticosteroid

therapy and the nursing care of patients receiving an adrenocorticosteroid drug is also presented.

The procedure for collection and treatment of data is described in Chapter III. The data were collected by the utilization of two original tools. One tool that was used was a questionnaire on adrenocorticosteroid therapy which assessed the nurses' level of knowledge in adrenocorticosteroid physiology, side-effects, nursing implications and administration techniques. The second tool that was utilized was a checklist designed to audit the documented nursing assessments made on patients receiving an adrenocorticosteroid drug.

Chapter IV describes the analysis of data. Analysis was accomplished by calculating correlation and linear regression coefficients.

In Chapter V, a summary of findings and conclusions were drawn as a result of this study. Implications for nursing and recommendations for further studies are included.

CHAPTER II

REVIEW OF LITERATURE

Introduction

Today the nursing profession is assuming an interdependent relationship with other health professions and no longer relies solely upon the physician as a decision maker. Nurses make decisions based upon their scientific knowledge and clinical assessments. Nurses utilize their skills to identify patients' problems and to find ways to solve these problems.

The professional nurse assumes many responsibilities; drug administration is one of them. The nurse must be aware of the drugs' pharmacological effects, side-effects, proper administration techniques and nursing implications which accompany the use of the drug. The adrenocorticosteroids are no exception. These drugs have numerous and diverse side-effects which require the nurse to continually assess and monitor the patient who is receiving a corticosteroid drug.

In developing the many aspects of adrenocorticosteroid therapy, this review of literature discusses ACTH, renin-angiotensin, glucocorticoid, mineralocorticoid, and androgen physiology. Adrenocorticosteroid therapy and the

nursing care of patients receiving an adrenocorticosteroid drug are also presented.

ACTH Physiology

Cortisol synthesis and release by the zona fasciculata and the zona reticularis of the human adrenal cortex is regulated by adrenocorticotropin hormone or ACTH. ACTH is an unbranched pituitary polypeptide of thirty-nine amino acids whose structure varies from specie to specie.[1] ACTH is stored and released from the anterior pituitary gland specifically in the basophil cells. Approximately 50 units or 0.25 mg. of active peptide are actually stored in the anterior pituitary at any given time. The biologic half life of ACTH is less than ten minutes.[2]

The primary factor which stimulates the release of ACTH from the anterior pituitary is corticotropin-releasing factor or CRF. CRF is produced in the median eminence of the hypothalamus and is known to be a short-chain peptide. The release of CRF may be stimulated by high centers in the nervous system or by cortisol plasma

[1]Richard B. Fisher and George A. Christie, A Dictionary of Drugs (New York: Schocken Books, 1971), p. 114.

[2]David P. Lauler, Gordon H. Williams, and George W. Thorn, "Disease of the Adrenal Cortex," in Harrison's Principles of Internal Medicine, 6th ed., ed. Maxwell M. Wintrobe et al. New York: McGraw-Hill Company, 1970), p. 483.

levels. Once the hypothalamus has been stimulated to release CRF, the chemical mediator will travel via the pituitary-stalk portal bloodstream to the anterior pituitary gland where it will stimulate the release of ACTH.[1]

A negative feedback mechanism stimulates CRF to be released in such a manner that when free cortisol levels are decreased, CRF levels will be stimulated to increase; and conversely if plasma cortisol levels are increased, the release of CRF will be decreased.[2] (See Figure 1.) The negative feedback mechanism is of great importance to the body as it serves to maintain a constant blood level of cortisol. It has been known for years that plasma cortisol levels are much higher in the early morning than in the afternoon or evening. This diurnal or circadian rhythm was difficult to explain by using the concept of a pure negative feeback system; therefore, it was postulated that the central nervous system was responsible for altering the feedback receptor in a daily rhythmical manner.[3] Recent studies have shown that the smooth-line rhythm of cortisol

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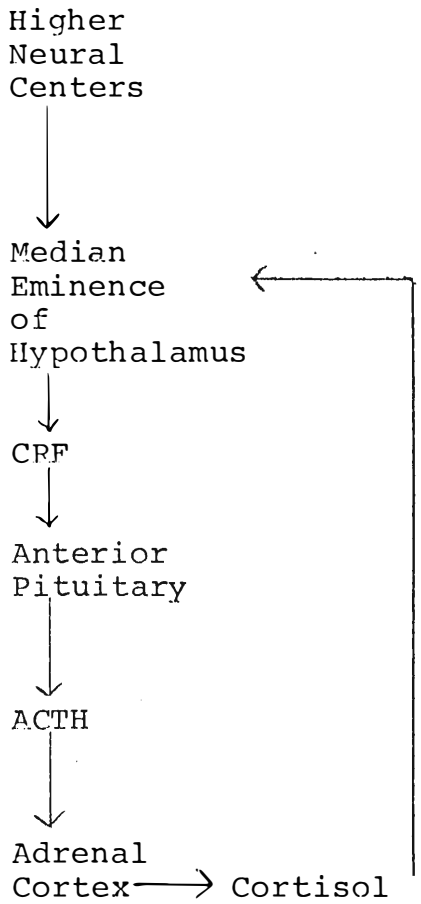
[1]Ibid.

[2]Ibid.

[3]John E. Bethune, The Adrenal Cortex (Michigan: The Upjohn Company, 1974), p. 30.

Figure 1. Normal ACTH - Cortisol Control Mechanisms

Control of cortisol secretion by ACTH is mediated by a negative feedback mechanism on the hypothalamic receptors and by higher neural centers.



SOURCE: John E. Bethune, The Adrenal Cortex (Michigan: The Upjohn Company, 1974):30, Figure 37.

levels is not entirely true, but that cortisol is secreted in "bursts" with high peaks and low valleys. The smooth diurnal pattern seen is a result of the fact that most of the ACTH-cortisol "bursts" occur in the early morning, peaking out between 4 a.m. and 8 a.m. (See Figure 2.)

The central nervous system exercises a complex control over CRF. This control may be seen in the regulation of the circadian rhythm and may also be noted when the organism is under stress. At these times, ACTH levels continue to remain high despite high plasma cortisol levels. In acute stress the adrenal production of steroids may increase from five to tenfold.[2] It also appears that cortisol has a direct feedback mechanism on the pituitary gland, higher brain centers, and perhaps even on the adrenal gland.[3]

ACTH has a rapid action on the adrenal gland as within minutes of its release there is an increase in the concentration of steroids in the blood. This produces a number of changes biochemically: there is an increase in the weight of the adrenal gland; there is a decrease in the

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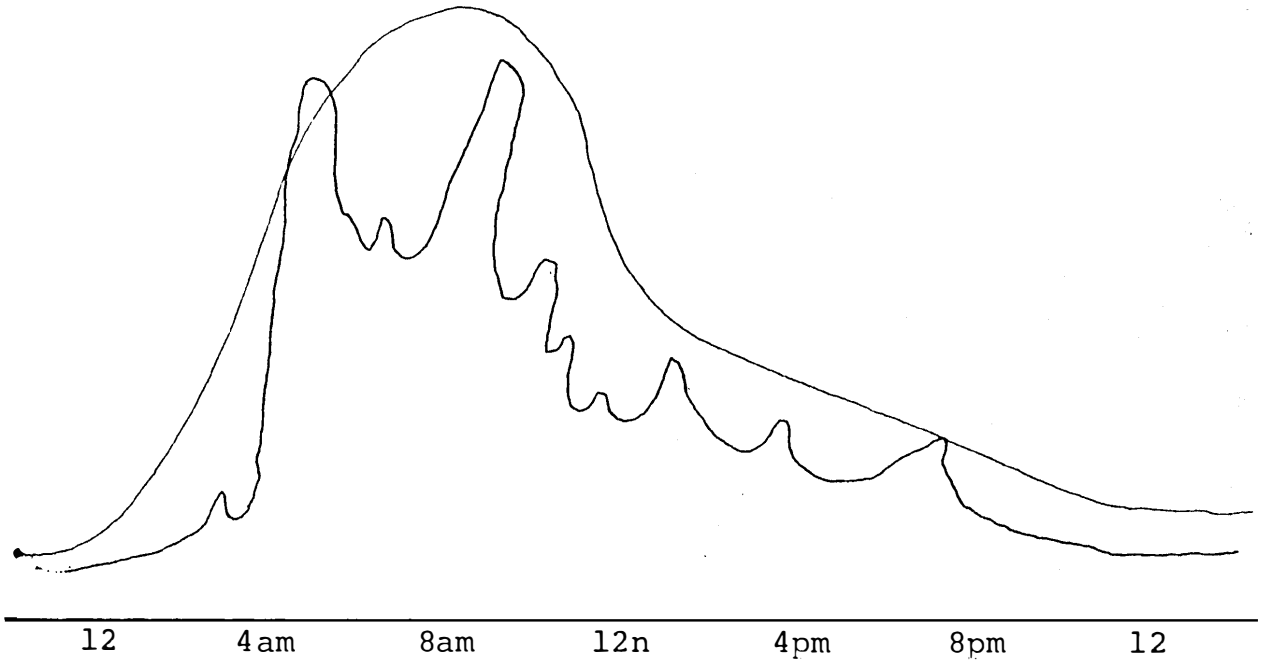
[1]Ibid., p. 31.

[2]Ibid., p. 29.

[3]David P. Lauler, Gordon H. Williams, and George W. Thorn, "Disease of the Adrenal Cortex," in Harrison's Principles of Internal Medicine, 6th ed., ed. Maxwell M. Wintrobe et al. (New York: McGraw-Hill Company, 1970), p. 483.

Figure 2. Diurnal Curve of Plasma Cortisol



"The diurnal curve of plasma cortisol is actually composed of irregular bursts of ACTH and cortisol secretion. The 'bursts' occur more frequently in the early morning and give rise to the 'circadian-rhythm' of cortisol shown by the smooth curve."

SOURCE: John E. Bethune, The Adrenal Cortex (Michigan: The Upjohn Company, 1974):31, Figure 38.

amount of adrenal lipids, cholesterol, and ascorbic acid; there is an increase in protein synthesis and oxidative phosphorylation; and there is an accelerated rate of glycolysis.[1]

The mechanism of ACTH is similar to the mechanism of action of many other non-steroid hormones. The hormone circulating in the plasma interacts with a receptor in the plasma membrane of the cell. This interaction leads to the activation of adenylate cyclase. This enzyme will then catalyze the formation of cyclic AMP from ATP. The cyclic AMP then triggers the hormone's recognized biological action.[2] In this reaction the hormone is considered to be the "first messenger" while the cyclic AMP is considered to be the "second messenger." Phosphodiesterase, which is a potent esterase, helps to regulate the intracellular level of cyclic AMP. Once the cyclic AMP has been formed, this nucleotide will activate a protein kinase which in turn activates the enzymes responsible for the expression of the hormone. The cyclic AMP is quickly destroyed by a specific phosphodiesterase.[3] (See Figure 3.)

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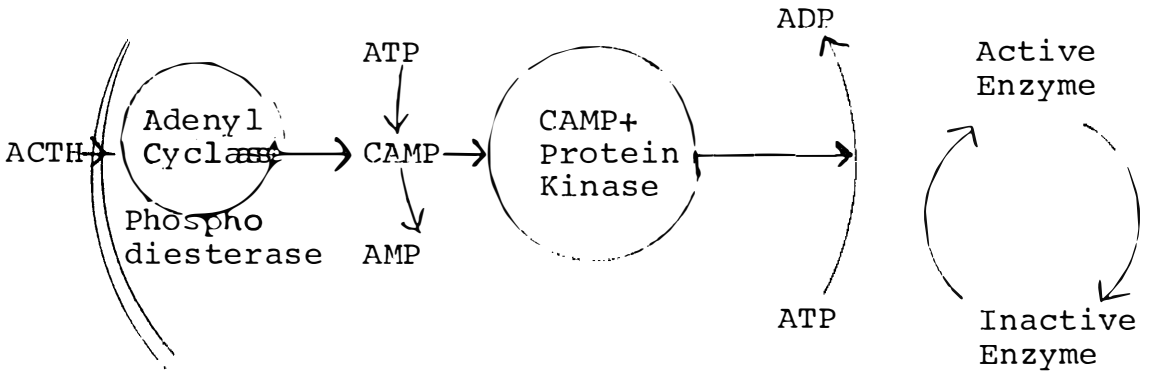
[1]Ibid.

[2]William A. Sodeman, Jr., and William A. Sodeman, Pathologic Physiology Mechanisms of Disease, 5th ed. (Philadelphia: W. B. Saunders Company, 1974), p. 867.

[3]Ibid., p. 868.

Figure 3. Cyclic AMP - Mediated Hormone Action

1. Circulating hormone interacts with receptor in cell membrane
2. Adenylate cyclase is activated
3. Adenylate cyclase then catalyzes the formation of cyclic AMP from ATP
4. Cyclic AMP then activates a protein kinase which causes the activation of enzymes responsible for the expression of the hormone
5. Cyclic AMP is then destroyed by a specific phosphodiesterase



SOURCE: William A. Sodeman, Jr. and William A. Sodeman, Pathologic Physiology Mechanisms of Disease, 5th ed. (Philadelphia: W. B. Saunders Company, 1974):868, Figure 32-1.

ACTH has other actions besides that of stimulating the formation and release of steroid hormones by the adrenal gland. It can stimulate the melanocytes of amphibians and can also increase adipokinetic activity in certain species.[1]

ACTH may not be the only substance which is capable of regulating adrenal cortical secretions. Recent work suggests that the adrenal androgen appears to be regulated by the level of pituitary luteinizing hormone (LH). Evidence to substantiate this claim comes from the observation that female patients with hirsutism, which is believed to be a result of an excess of circulating adrenal androgens, have often had their symptoms controlled by oral contraceptives which inhibit LH production.[2]

ACTH has successfully been synthesized with full biological activity. Synthetic ACTH is mediated through the final common pathways of increased steroid production from the adrenal cortex.[3]

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[1]David P. Lauler, Gordon H. Williams, and George W. Thorn, "Disease of the Adrenal Cortex," in Harrison's Principles of Internal Medicine, 6th ed., ed. Maxwell M. Wintrobe et al. (New York: McGraw-Hill Company, 1970), p. 483.

[2]Richard B. Fisher and George A. Christie, A Dictionary of Drugs (New York: Schocken Books, 1971), p. 127.

[3]Ibid., p. 114.

Renin-Angiotensin Physiology

The renin-angiotensin system is the prime regulator of aldosterone secretion from the zona glomerulosa. Renin is a proteolytic enzyme which is produced and stored in the granules of the juxtaglomerular cells surrounding the afferent arterioles of the cortical glomeruli and is also stored in arterial walls.[1] The juxtaglomerular apparatus includes the juxtaglomerular cells and the cells of the macula densa. Renin acts on the basic angiotensinogen, a circulating alpha$_2$-globulin made in the liver. The product of this interaction is angiotensin I, which is a vasoinactive substance.[2]

Angiotensin I is acted upon by a plasma enzyme and is converted to Angiotensin II. Angiotensin II is the most potent vasopressor produced in the body.[3] Its pressor effects are brought about by its direct effect on arteriolar

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[1]William A. Sodeman, Jr. and William A. Sodeman, Patholo ic Ph siolo Mechanisms of Disease, 5th ed. Philadelphia: W. B. Saunders Company, 1974), p. 188.

[2]Betty Bergerson and Elsie E. Krug, Pharmacology in Nursing, 11th ed. (Saint Louis: C. V. Mosby Company, 1969), p. 428.

[3]David P. Lauler, Gordon H. Williams, and George W. Thorn, "Disease of the Adrenal Cortex," in Harrison's Principles of Internal Medicine, 6th ed., ed. Maxwell M. Wintrobe et al. (New York: McGraw-Hill Company, 1970), p. 484.

smooth muscle. Angiotensin II also has the potent ability to directly stimulate the production of aldosterone by the adrenal cortex, specifically the zona glomerulosa.[1] Angiotensin II is destroyed by various peptidases which are known as angiotensinases. They are found in organ tissue, vessel walls and circulating plasma.[2] It is possible that angiotensin II may directly affect the renal tubules' ability to transport sodium. This remains unproven at present.

Renin can be released by two mechanisms, either extrarenal or intrarenal. The extrarenal mechanism may override the intrarenal mechanism on occasion; however, they usually act synergistically in series.

The extrarenal mechanism is stimulated by volume depletion. The juxtaglomerular cells are specialized myo-epithelial cells which are capable of acting like miniature pressor receptors. They sense any changes in renal perfusion pressures.[3] The changes in pressure are sensed by a difference in stretch on the arteriolar walls. For

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[1]Ibid.

[2]Ibid.

[3]David P. Lauler, Gordon H. Williams, and George W. Thorn, "Disease of the Adrenal Cortex," in Harrison's Principles of Internal Medicine, 6th ed., ed. Maxwell M. Wintrobe et al. (New York: McGraw-Hill Company, 1970), p. 484.

example, when the circulating volume is decreased, there will be a corresponding decrease in renal perfusion and therefore a decrease in arteriolar pressure. A decrease in stretch will be exerted on the afferent arteriolar walls, and thus the juxtaglomerular cells will be stimulated to release renin within the kidney circulation, which leads to the formation of angiotensin II.[1] Angiotensin II leaves the kidney by way of the lymphatic and venous flow. It directly stimulates the adrenal cortex to release aldosterone.

When aldosterone is released in sufficient quantities, sodium will be retained and consequently water retention will also occur, thereby expanding extracellular fluid volume. When the body's extracellular fluid has been restored to normal, stretch will be exerted on the juxtaglomerular cells in the afferent arterioles, and thus the renin will be turned off. (See figure 4.) Catecholamines and sympathetic nervous responses in the kidney are capable of enhancing the juxtaglomerular cell response to changes in perfusion.[2] If the angiotensin II is found in great

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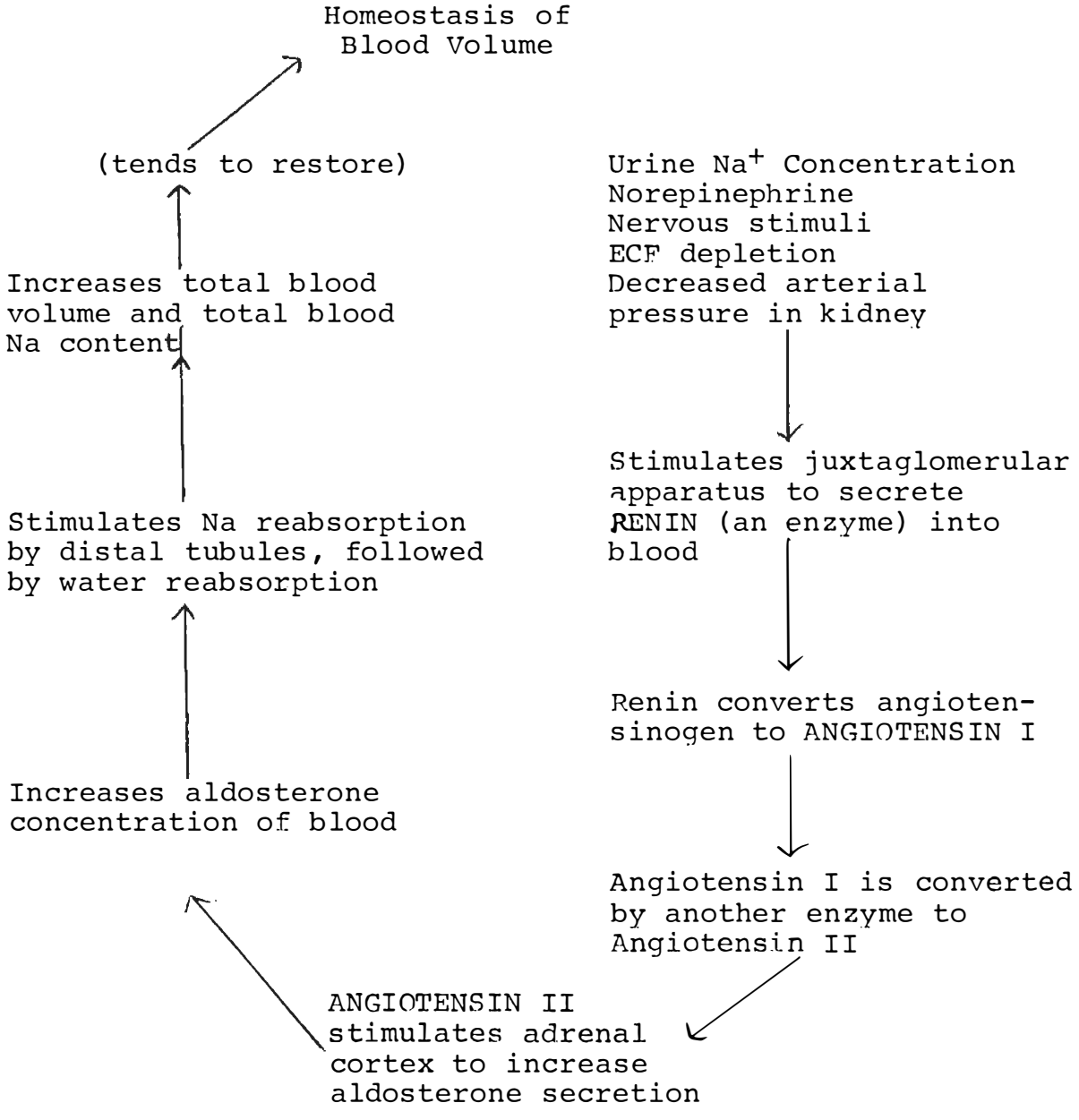
[1]Ibid.

[2]David P. Lauler, Gordon H. Williams, and George W. Thorn, "Disease of the Adrenal Cortex," in Harrison's Principles of Internal Medicine, 6th ed., ed. Maxwell M. Wintrobe et al. New York: McGraw-Hill Company, 1970), p. 484.

Figure 4. The postulated renin-angiotensin II mechanism for controlling aldosterone secretion and maintaining normal blood volume



SOURCE: Catherine Parker Anthony and Norma Jane Kolthoff, Textbook of Anatomy and Physiology, 8th ed. (Saint Louis: The C. V. Mosby Company, 1971):281, Figure 9-12.

enough quantities in the blood, it may act as a vasopressor itself.[1]

More important perhaps is the fact that angiotensin has major effects on sympathetic nervous activity via a direct stimulatory action on the vasomotor center. It enhances sympathetic activity peripherally by decreasing norepinephrine uptake, thereby allowing greater concentrations of the neurotransmitter to reach receptor sites. Angiotensin is finally a potent stimulus on the adrenal medulla to release catecholamines.[2]

The intrarenal control mechanism is based upon the theory that the macula densa cells function as chemoreceptors which monitor the sodium load in the distal tubule.[3] The macula densa is a group of special staining epithelial cells which are found in the distal convoluted tubules. This location is in direct opposition to the juxtaglomerular cells. Once the macula densa monitors the sodium level the information is fed directly back to the

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[1]Ibid.

[2]William A. Sodeman, and William A. Sodeman, Pathologic Physiology Mechanisms of Disease, 5th ed. (Philadelphia: W. B. Saunders Company, 974), p. 189.

[3]David P. Lauler, Gordon H. Williams, and George W. Thorn, "Disease of the Adrenal Cortex," in Harrison's Princi les of Internal Medicine, 6th ed., ed. Maxwell M. Wintrobe et al. New Yor : McGraw-Hill Company, 1970), p. 484.

juxtaglomerular cells where appropriate modification in renin release may take place.[1] It is thought that the intrarenal mechanism is capable of operating independently of changes in renal perfusion. Evidence for various hypotheses concerning the intrarenal mechanism's purpose and action is conflicting.

Other suggested mechanisms for the control of aldosterone release are potassium and ACTH. In experimental animals potassium has been found to have the ability to directly stimulate the zona glomerulosa to release aldosterone.[2] Since aldosterone increases renal potassium loss,it is not surprising that high cellular potassium has the ability to directly stimulate aldosterone release. ACTH seems to play a role in keeping aldosterone synthesis normal; however,it does not seem to play an important role in the regulation of body fluids through aldosterone.[3] There are apparently many unknown regulators of salt and water metabolism,since it is difficult to comprehend how a patient with Addison's disease can maintain perfect health and a normal body fluid regulation on a fixed intake of

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[1]Ibid.

[2]John E. Bethune, The Adrenal Cortex (Michigan: The Upjohn Company, 1974), p. 36.

[3]Ibid., p. 37.

glucocorticoid and mineralocorticoid despite a wide variation in salt and water intake.[1]

Aldosterone also follows a circadian variation similar to cortisol; however, it is less obvious than that for plasma cortisol. Postural changes greatly influence plasma renin and aldosterone levels. In an upright position blood pools in the legs away from the volume receptors in the kidneys, thereby stimulating the release of renin. A basal level will be reached in a supine position.[2]

Excess renin secretion has been found to be one of the important causes of hypertension, since excess renin is followed by excess angiotensin II formation.[3] (See figure 5.) It has been known since 1898 that a relationship existed between the kidney and hypertension. The subsequent research showed that a substance which was released from the kidney was converted by the blood stream to a vasoconstrictor. In 1940 reports began to appear in the United States and Argentina describing this vasopressor. This compound was not synthesized until 1957 and was named angiotensin.[4]

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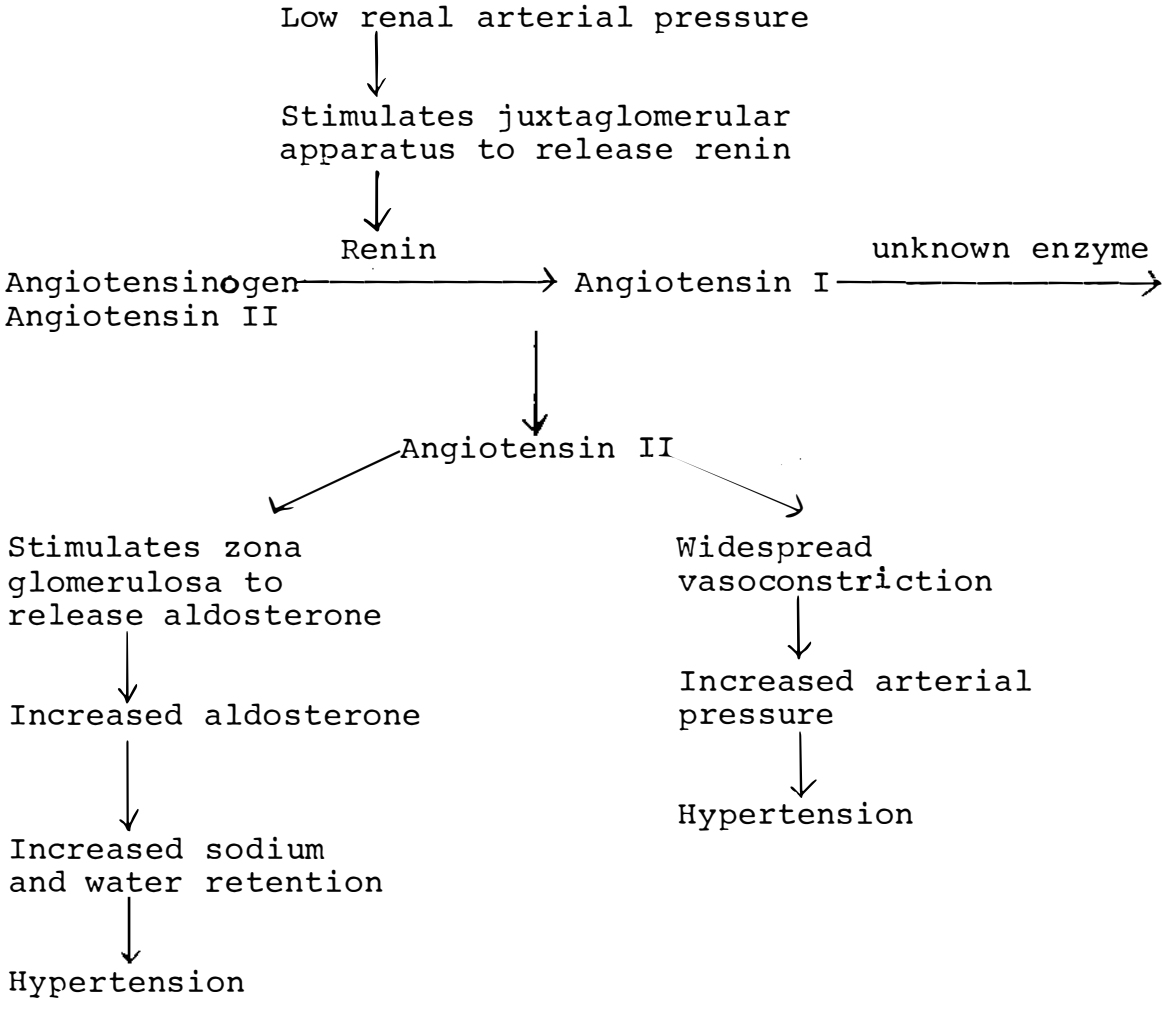
[1]Ibid.

[2]Ibid.

[3]Catherine Parker Anthony and Norma Jane Kolthoff, Textbook of Anatomy and Physiology, 8th ed. (Saint Louis: The C. V. Mosby Company, 1971), p. 280.

[4]Betty Bergerson and Elsie E. Krug, Pharmacology in Nursing, 11th ed. (Saint Louis: The C. V. Mosby Company, 1969), p. 428.

Figure 5. Hypertension as Caused by Increased Angiotensin II Production



SOURCE: Catherine Parker Anthony and Norma Jane Kolthoff, Textbook of Anatomy and Physiology, 8th ed. (Saint Louis: The C. V. Mosby Company, 1971):282, Figure 9-13.

Angiotensin amide is a controversial drug whose therapeutic role has not yet been clearly identified. It has been used for hypotension when other vasopressors have been ineffective.[1]

Glucocorticoid Physiology

Although the adrenal steroids have been divided into glucocorticoids and mineralocorticoids it should be noted that most glucocorticoids have some mineralocorticoid properties while most mineralocorticoids have some glucocorticoid properties. The term glucocorticoid is given to the group of adrenal steroids which have their primary action on intermediary metabolism. Cortisol is the major glucocorticoid.

The action of the glucocorticoids is mainly on intermediary metabolism which includes the regulation of protein, carbohydrate, lipid, and nucleic acid metabolism.[2] The mechanism of action of both cortisol and aldosterone, the major mineralocorticoid, seems to be similar to the action of several other steroid hormones. The suggested mode of action is that each steroid hormone combines with

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[1] Ibid.

[2] David P. Lauler, Gordon H. Williams, and George W. Thorn, "Disease of the Adrenal Cortex," in Harrison's Principles of Internal Medicine, 6th ed., ed. Maxwell M. Wintrobe et al. New York: McGraw-Hill Company, 1970), p. 484.

a specific receptor protein in the cytoplasm; this steroid-receptor complex then moves into the cell's nucleus where it combines with a specific acceptor in the chromatin. The steroid-receptor-acceptor complex in turn causes the nuclear genetic material to produce messenger RNA, which in turn stimulates the formation of the enzyme,which is more directly responsible for the biological expression of the hormone's activity.[1] There is approximately a two to four hour delay before the steroid's effects are seen.[2]

Glucocorticoid action seems to be mainly catabolic in certain tissues other than the liver, in as much as an increase in protein breakdown and nitrogen excretion occurs. This results in a negative nitrogen balance in the presence of high concentrations of glucocorticoids.[3] Glucocorticoids increase hepatic glycogen content by increasing hepatic glucose synthesis. This process in combination with the glucocorticoid's ability to inhibit the insulin induced glucose movement into the muscle and adipose cell results

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[1]William A. Sodeman, Jr. and William A. Sodeman, Pathologic Physiology Mechanisms of Disease, 5th ed. (Philadelphia: W. B. Saunders Company, 1974), p. 869.

[2]John E. Bethune, The Adrenal Cortex (Michigan: The Upjohn Company, 1974), p. 31.

[3]Ibid.

in hyperglycemia.[1] These are two mechanisms whereby the glucocorticoids promote the conservation of glucose as an energy source. Hepatic glucose synthesis occurs by increasing the quantity of intermediary metabolites, especially those derived from glycogenic amino acids. Hepatic trapping and de-aminization of amino acids derived from peripheral supporting structures such as bone, skin, muscle, and connective tissue seem to be essential to the process.[2] The increase in protein mobilization results in increased plasma levels of amino acids and, therefore, an increase in amino acid and uric acid excretion.

Cortisol enhances the release of free fatty acids from adipose tissue during fasting or adrenergic stimulation. Cortisol affects structural protein and adipose tissue in a different manner depending on the part of the body in which the tissue is located. An example of this occurrence is that the protein matrix of the vertebral column may be severely depleted of protein while the protein

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[1]David Rabinowitz and Turner Bledsoe, "The Adrenal Glands," in Principles and Practice of Medicine, 18th ed., ed. A. McGehee Harvey et al. (New York: Appleton-Century-Crofts, 1972), p. 857.

[2]David P. Lauler, Gordon H. Williams, and George W. Thorn, "Disease of the Adrenal Cortex," in Harrison's Principles of Internal Medicine, 6th ed., ed. Maxwell M. Wintrobe et al. (New York: McGraw-Hill Company, 1970), p. 485.

matrix of the long bone may be affected only slightly, with an end result of only mild osteoporosis.[1] Adipose tissue is similarly affected in as much as peripheral adipose tissue may diminish while abdominal and interscapular fat may accumulate.[2] Fatty acid mobilization is another mechanism whereby the glucocorticoids attempt to conserve glucose as an energy source as it encourages muscle tissue to use fatty acid metabolism instead of glucose to derive its energy.[3] Cortisol also seems to play a permissive part in allowing thyroxine and epinephrine to carry out their fat mobilizing effects.[4]

The glucocorticoids are also responsible for directly increasing the levels of specific hepatic enzymes. The increased levels of these hepatic enzymes can influence the balance of serum amino acids which, in the long run,

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[1]David P. Lauler, Gordon H. Williams, and George W. Thorn, "Disease of the Adrenal Cortex," in Harrison's Principles of Internal Medicine, 6th ed. ed. Maxwell M. Wintrobe et al. (New York: McGraw-Hill Company, 1970), p. 485.

[2]Ibid.

[3]John E. Bethune, The Adrenal Cortex (Michigan: The Upjohn Company, 1974), p. 31.

[4]David Rabinowitz and Turner Bledsoe, "The Adrenal Glands," in Principles and Practice of Medicine, 18th ed., ed. A. McGehee Harvey et al. (New York: Appleton-Century-Crofts, 1972), p. 857.

can be responsible for decreasing protein metabolism with subsequent cytolysis of lymphoid and muscle tissue and a cessation of growth.[1] Prolonged muscle cytolysis results in muscle weakness with atrophy, while the lymph tissue cytolysis results in a decreased production of lymphocytes with an overall decrease in immune response, as well as a decrease in the body's ability to establish an inflammatory response.[2]

The glucocorticoids are known and used most frequently for their anti-inflammatory effects. These effects are thought to occur not only due to a production of lymphoid tissue lysis but are also due to the glucocorticoid's ability to decrease the number of circulating eosinophils. Inhibition of exudation, cell proliferation, capillary dilation, production of histamine, and antibody formation are other contributing factors in producing an anti-inflammatory effect.[3] The glucocorticoids also have the ability to stabilize lysosomal membranes. This particular action

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[1]David P. Lauler, Gordon H. Williams, and George W. Thorn, "Disease of the Adrenal Cortex," in Harrison's Principles of Internal Medicine, 6th ed., ed. Maxwell M. Wintrobe et al. (New York: McGraw-Hill Company, 1970), p. 485.

[2]John E. Bethune, The Adrenal Cortex (Michigan: The Upjohn Company, 1974), p. 32.

[3]Windsor C. Cutting, Handbook of Pharmacology, 5th ed. (New York: Appleton-Century-Crofts, 1972), p. 338.

suppresses the release of acid hydrolases which are stored in the lysosomes, thereby helping to lessen an inflammatory response and maintain cell integrity.[1]

Cortisol affects body water distribution and excretion. It helps to maintain the extracellular fluid volume by decreasing the movement of water into the cell.[2] Cortisol affects fluid excretion by increasing glomerular filtration rate and also by directly stimulating the renal tubule. The result of this action is to increase solute free water clearance. The end result is tubular reabsorption of sodium and excretion of potassium.[3]

It is thought that cortisol is necessary for normal vascular reactivity. It helps the vascular smooth muscle respond to circulating vasoconstrictive factors, but prevents excessive vasoconstriction which may lead to ischemia.[4] This is a particularly important action during times of stress.

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[1]David P. Lauler, Gordon H. Williams, and George W. Thorn, "Disease of the Adrenal Cortex," in Harrison's Principles of Internal Medicine, 6th ed., ed. Maxwell M. Wintrobe et al. (New York: McGraw-Hill Company, 1970), p. 485.

[2]Ibid.

[3]Ibid.

[4]Ibid.

Personality is definitely affected by cortisol levels. Emotional disturbances are common with either excess or deficit of cortisol; however, the exact mechanism of action on the central nervous system is not known.[1]

Some of the other effects of the glucocorticoids are on glandular tissue, blood clotting, and the anterior pituitary gland. The glucocorticoids exert a stimulatory effect on glandular tissue whereby saliva, sweat, gastric acid, and sebum production is increased.[2] Peptic ulcers and acne are the two most commonly seen side-effects in this category. There is an increased blood clotting ability seen in conditions with high concentrations of glucocorticoids. A complication of this effect is thrombo-emboli formation.[3] A final effect of the glucocorticoids is their ability to suppress ACTH release from the anterior pituitary gland.[4]

Despite many years of investigation, the exact mechanism of action of the glucocorticoids has not clearly been established. A widely accepted concept is

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[1]David P. Lauler, Gordon H. Williams, and George W. Thorn, "Disease of the Adrenal Cortex," in Harrison's Principles of Internal Medicine, 6th ed., ed. Maxwell M. Wintrobe et al. (New York: McGraw-Hill Company, 1970), p. 485.

[2]Windsor C. Cutting, Handbook of Pharmacology, 5th ed. (New York: Appleton-Century-Crofts, 1972), p. 337.

[3]Ibid.

[4]Ibid.

that cortisol exerts a "permissive" action on metabolic and enzyme function throughout cellular tissue thereby allowing a variety of other agents to exert their effects.[1]

Mineralocorticoid Physiology

The three major mineralocorticoids are 11-deoxycorticosterone (DOC), corticosterone, and aldosterone. DOC and corticosterone are the precursors of aldosterone. The production of DOC and corticosterone is directly regulated by ACTH levels.[2]

The major effects of the mineralocorticoids is on sodium and potassium metabolism. The effects of each of the various mineralocorticoids are very similar and indistinguishable. Aldosterone is the most potent of the mineralocorticoids and thus is considered to be the primary mineralocorticoid.[3]

Aldosterone has two major physiological functions; it is the primary regulator of extracellular fluid volume

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[1]John E. Bethune, The Adrenal Cortex (Michigan: The Upjohn Company, 1974), p. 31.

[2]David P. Lauler, Gordon H. Williams, and George W. Thorn, "Disease of the Adrenal Cortex," in Harrison's Principles of Internal Medicine, 6th ed., ed. Maxwell M. Wintrobe et al. (New York: McGraw-Hill Company, 1970), p. 485.

[3]Ibid.

and of potassium metabolism. Aldosterone regulates fluid volume through its effects on renal tubular reabsorption of sodium ions. It acts mainly in the renal distal convoluted tubules where it accelerates the exchange of sodium ions for secreted potassium and/or hydrogen ions.[1] Aldosterone plays a major part in making the sodium-potassium exchange more efficient in the distal tubules. When the sodium ions are reabsorbed they are transported out of the tubular epithelial cells into the interstial fluid volume of the kidney and from there into the renal capillary circulation.[2] Since water passively follows the sodium ion, it may be said that aldosterone is, therefore, the prime regulator of the extracellular fluid volume, though it actually regulates the sodium ion load.[3]

Under conditions of extracellular fluid volume depletion aldosterone will influence the iso-osmotic reabsorption of the proximal tubular sodium; conversely, when the extracellular fluid volume is increased, aldosterone will not be released and thus sodium will not be reabsorbed.[4]

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[1]David P. Lauler, Gordon H. Williams, and George W. Thorn, "Disease of the Adrenal Cortex," in Harrison's Princi les of Internal Medicine, 6th ed., ed. Maxwell M. Wintro e et al. New York: McGraw-Hill Company, 1970), p. 485.

[2] Ibid.

[3]Ibid.

[4]Ibid.

It has not clearly been demonstrated whether or not aldosterone affects the ascending loop of Henle's transport of sodium. Aldosterone is known to act directly on the epithelium of the salivary ducts and sweat glands, and also on the mucosa cells of the gastro-intestinal tract to cause a direct reabsorption of sodium in exchange for potassium.[1] It has been difficult to prove whether or not a direct cellular action occurs, as that which takes place on muscle cells. It has been shown that in the absence of aldosterone, sodium tends to leave the extracellular fluid by migrating into the cells. Increased sodium wasting by the kidneys occurs concurrently with the collection of sodium on tendon and bone surfaces.

At the present time three control mechanisms for the release of aldosterone have been identified. It is thought that many other control mechanisms exist; however, they have not been discovered as yet. The three known control systems are the renin-angiotensin system, potassium, and ACTH. A fourth postulated mechanism for aldosterone regulation is that a receptor in the diencephalon near the

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[1]David P. Lauler, Gordon H. Williams, and George W. Thorn, "Disease of the Adrenal Cortex," in Harrison's Principles of Internal Medicine, 6th ed., ed. Maxwell M. Wintrobe et al. (New York: McGraw-Hill Company, 1970), p. 485.

[2]Ibid.

pineal body elaborates a lipid hormonal substance in response to decreased fluid volume or a low sodium concentration.[1] The renin-angiotensin is the main control mechanism, as volume regulation in the absence of the renin-angiotensin system is extremely difficult and is characterized by marked oscillations of blood pressure.[2]

The potassium control mechanism for the release of aldosterone operates independently of the renin-angiotensin system. It has been found that if a solution of potassium is injected into the adrenal artery, an immediate increase in adrenal venous aldosterone levels can be measured.[3] This finding indicates that potassium has a direct stimulatory effect on adrenocortical production of aldosterone. The mechanism is thought to operate on a transmembrane principle, whereby the adrenocortical cells act as a monitor of the flux of potassium across their membranes.[4] When there is an increase in the number of potassium ions across the membranes, there is an increased production of aldosterone,

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[1]Windsor C. Cutting, Handbook of Pharmacology, 5th ed. (New York: Appleton-Century-Crofts, 1972), p. 336.

[2]David P. Lauler, Gordon H. Williams, and George W. Thorn, "Disease of the Adrenal Cortex," in Harrison's Principles of Internal Medicine, 6th ed., ed. Maxwell M. Wintrobe et al. (New York: McGraw-Hill Company, 1970), p. 485.

[3]Ibid.

[4]Ibid.

while a decrease in aldosterone production will result when the number of potassium ions which cross the adrenocortical cell membranes are decreased.[1] The potassium control mechanism works synergistically with the renin-angiotensin mechanism; if the renin-angiotensin system becomes impaired, either through acute or chronic renal failure, the potassium-aldosterone control system can take over its control system with comparative potency.[2] The potassium-aldosterone control mechanism acts as a protective system against potassium intoxication.

ACTH plays a small role in the control of aldosterone release. Hypophysectomized patients, or patients with pituitary insufficiency, have a suboptimal aldosterone response to sodium restriction. In a normal person with ACTH administration there is an acute rise in aldosterone secretion to levels of three to four times above base line.[3] With continued administration of ACTH, however, the aldosterone production rate returns to control levels, providing suggestive evidence that ACTH is not an important control mechanism

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[1]David P. Lauler, Gordon H. Williams, and George W. Thorn, "Disease of the Adrenal Cortex," in Harrison's Principles of Internal Medicine, 6th ed., ed. Maxwell M. Wintrobe et al. (New York: McGraw-Hill Company, 1970), p. 486.

[2]Ibid.

[3]Ibid.

in day to day physiological conditions.[1] This mechanism, however, is of great value in times of stress.

It has recently been suggested that pituitary factors other than ACTH may exist which are necessary for a maximum adrenal-aldosterone response. When a normal individual is given a long term course of aldosterone, an initial period of sodium retention is followed by natriuresis, and sodium balance is re-established. As a result clinical edema does not develop. Even though the patient continues to be given a potent sodium retaining hormone, the patient achieves sodium homeostasis without exhibiting sodium retention symptoms. This phenomenon is known as the "escape phenomenon." This means the renal tubules are not affected by the sodium retaining action of the chronically administered aldosterone.[3] The mechanism for this phenomenon is unknown, although it seems that it is dependent on normal renal hemodynamics; however, it may also be due to the inhibition of proximal tubular sodium resorption by a "third factor," which at one time was called

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[1]David P. Lauler, Gordon H. Williams, and George W. Thorn, "Disease of the Adrenal Cortex," in Harrison's Principles of Internal Medicine, 6th ed., ed. Maxwell M. Wintrobe et al. (New York: McGraw-Hill Company, 1970), p. 486.

[2]Ibid.

[3]Ibid.

the "salt wasting" adrenal hormone.[1] This "escape" phenomenon is exhibited by patients with essential hypertension but is absent in patients with congestive heart failure, nephrosis, and ascites.[2]

Androgen Physiology

The primary adrenal androgens are dehydroepiandrosterone (DHEA), androstenedione, and 11-hydroxyandrostenedione. These androgens are biologically weaker than testosterone; however, they are capable of being converted to testosterone by many tissues of the body.[3] Adrenal androgens are stimulated to be released by ACTH and not by gonadotropins. When ACTH release is stimulated, there is a definite increase in the 17 ketosteroid and 17 hydroxycorticoids in the urine. About two-thirds of the urinary 17 ketosteroids in the male are derived from the adrenal androgens, while the remaining third is from the breakdown of testicular secretions. All of the female's 17 ketosteroids are derived from the adrenal androgen secretion.[4] Adrenal androgens are suppressed by exogenous administration

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[1]John E. Bethune, The Adrenal Cortex (Michigan: The Upjohn Company, 1974), p. 38.

[2]Ibid.

[3]Ibid., p. 24.

[4]Ibid.

of glucocorticoids. Other specific control mechanisms for adrenal androgen release may exist independent of the ACTH system.[1]

Major effects of the androgens are on protein synthesis and stimulation of secondary sex characteristics. Muscle mass and strength are increased by the androgens' ability to stimulate protein synthesis. Linear growth is accelerated prior to epiphyseal closure. Secondary sex characteristics are affected as feminine qualities are suppressed and male characteristics are accentuated.[2] Androgens also stimulate the production of sebum by sebaceaus glands; therefore, acne may become a problem. Nitrogen is retained, and inorganic phosphorus, sulphate, sodium, and potassium storage is also affected.[3]

Symptoms of over production of adrenal androgens in the female are hirsutism and virilization with amenorrhea; atrophy of the breasts and uterus; enlargement of the clitoris; deepening of the voice; acne; increase in muscle

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[1]David P. Lauler, Gordon H. Williams, and George W. Thorn, "Disease of the Adrenal Cortex," in Harrison's Principles of Internal Medicine, 6th ed., ed. Maxwell M. Wintrobe et al. (New York: McGraw-Hill Company, 1970), p. 486.

[2]Ibid.

[3]Betty Bergerson and Elsie E. Krug, Pharmacology in Nursing, 11th ed. (Saint Louis: C. V. Mosby Company, 1969), p. 561.

mass; heterosexual drives; and a receding hairline. Symptoms in the male are increased body and sex hair with enlargement of sexual organs.[1]

The cellular action of androgens are not certain. The androgen compound is apparently converted into a closely related compound. It is then bound to nuclei in cells of sensitive organs where it increases the biosynthesis of nucleic acids, thus indirectly increasing the biosynthesis of protein.[2]

Adrenocorticosteroid Therapy

The adrenal glands were first given attention in 1849 when Thomas Addison wrote a description of a clinical syndrome which resulted when the adrenal glands were destroyed.[3] Continued work resulted in demonstrating that the adrenal cortex, not the adrenal medulla, produced a life sustaining hormone. Methods for preparing adrenocortical

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[1]David P. Lauler, Gordon H. Williams, and George W. Thorn, "Disease of the Adrenal Cortex," in Harrison's Principles of Internal Medicine, 6th ed., ed. Maxwell M. Wintrobe et al. New York: McGraw-Hill Company, 1970), p. 486.

[2]Richard B. Fisher and George A. Christie, A Dictionary of Drugs (New York: Schocken Books, 1971), p. 229.

[3]David P. Lauler, Gordon H. Williams, and George W. Thorn, "Disease of the Adrenal Cortex," in Harrison's Principles of Internal Medicine, 6th ed., ed. Maxwell M. Wintrobe et al. New York: McGraw-Hill Company, 1970), p. 477.

extracts were begun between 1927 and 1930. It was not until 1935 that the first group of crystalline substances was isolated from the adrenal cortex. Cortisone was one of those isolated compounds. It was first used in medicine in 1948 in the treatment of rheumatoid arthritis.[1]

Since that time hundreds of synthetic corticosteroid compounds have been synthesized and biologically tested in an attempt to improve the therapeutic effectiveness of cortisol.[2] Great energy has been expended in an effort to enhance the drug's anti-inflammatory properties while concurrently negating its adverse effects, especially that of its sodium retaining properties. To date, this goal has not been attained.

Pure glucocorticoid and mineralocorticoid drugs have been produced; however, it should be noted that many of the naturally occurring hormones as well as the synthetic derivatives possess characteristics of both categories. A corticosteroid compound is therefore placed in either of

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[1]Betty Bergerson and Elsie E. Krug, Pharmacology in Nursing, 11th ed. (Saint Louis: C. V. Mosby Company, 1969), p. 17.

[2]John E. Bethune, The Adrenal Cortex (Michigan: The Upjohn Company, 1974), p. 39.

these categories as a matter of convenience.[1] This is the reason that the adrenal steroid compounds should be thought of as existing on a continuum ranging from a pure glucocorticoid to a pure mineralocorticoid.[2] Individual preparations have different potencies with a varying amount of glucocorticoid versus mineralocorticoid effect. See Table 1 for the comparative potencies of some of the more commonly used corticosteroid preparations.

Despite the numerous available corticosteroid compounds, relatively few are currently being used in the practice of medicine. Corticosteroids are used in clinical medicine in three ways:

1. As physiologic replacement therapy to alleviate any existing adrenocortical hypofunctioning
2. As a diagnostic aid to determine whether or not the endocrine system is functioning adequately
3. As a pharmacologic agent which possesses either an exaggeration of its physiologic effect or an entirely new effect which is not observed when used in physiologic doses[3]

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[1] Mary Evans Hamdi, "Nursing Interventions for Patients Receiving Corticosteroid Therapy," in Advanced Concepts in Clinical Nursing, ed. Kay Corman Kintzel (Philadelphia: J. B. Lippincott Company, 1971), p. 236.

[2] Ibid.

[3] Ibid., p. 237.

TABLE 1

Adrenal Preparations
Comparative Potencies of Some Corticoids

Commonly Used Names	Estimated Potencies*	
	Glucocorticoid	Mineralocorticoid
Hydrocortisone (Cortisol)	1	1
Cortisone (Cortone)	0.8	0.8
DOC (Percorten)	0	15
Aldosterone (Electrocortin)	0.3	400
Prednisolone (Meticortelone)	4	0.25
Prednisone (Meticorten)	4	0.25
Methylprednisolone (Medrol)	5	*
Triamcinolone (Aristocort)	5	*
Dexamethasone (Decadron)	30 - 40	*
Fluorohydrocortisone (Florinef)	10	300

*Relative milligram comparisons to cortisol, setting the glucocorticoid and mineralocorticoid properties of cortisol as 1.

SOURCE: David P. Lauler, Gordon H. Williams, and George W. Thorn, "Diseases of the Adrenal Cortes," in Harrison's Principles of Internal Medicine, 6th ed., ed. Maxwell M. Wintrobe et al. (New York: McGraw-Hill Company, 1970): 512, Table 92-12.

The corticosteroids are one of the most frequently prescribed drugs in the United States.[1] Corticosteroid therapy is used mainly as a pharmacologic agent in the treatment of a wide variety of pathological conditions ranging from endocrine disorders, such as adrenal insufficiency, to collagen diseases such as systemic lupus erythematosus.

The following are the major beneficial pharmacological effects for which the glucocorticoids are prescribed.

1. Anti-inflammatory effects: These effects are a result of a decrease in vascular permeability, an increased capillary resistance, and the potentiation of norepinephrine's vasoconstricting effects. The glucocorticoids may also suppress the release or activity of histamine while the migration of inflammatory cells from capillaries are depressed. Cellular integrity to water is maintained while edema fluid formation is reduced.[2] These effects are desired in the treatment of rheumatoid arthritis.
2. Lymphoid tissue involution: The suppression of lymphoid tissue activity and formation results in the decrease of eosinophils with an increase in polymorphonucleocytes. The latter effect is probably due to a

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[1]John E. Bethune, The Adrenal Cortex (Michigan: The Upjohn Company, 1974), p. 31.

[2]Ibid., p. 32.

decrease destruction and egress of mature leukocytes from the capillaries.[1] The treatment of acute and chronic lymphocytic leukemia is based on these effects.

3. Anti-allergy effects: These effects are a primary result of suppression of the anti-inflammatory response that results from antigen-antibody induced injury. In man, steroids do not interfere with the antigen-antibody interaction in therapeutic doses. If, however, high doses are maintained for a prolonged period of time, new antibody formation is depressed. This is a result of lymphocytolysis and suppressed gamma globulin formation. A week of high dose therapy is necessary before this desired effect can occur. The glucocorticoids probably do not suppress histamine release, but rather oppose its tissue effects. Steroids must be present at the actual site of inflammation so that this action may be realized.[2]
4. Lysosomal stabilization effects: The glucocorticoids have the ability to stabilize lysosomal membranes. This is an important effect in as much as it prevents the lysosomes from spilling their hydrolytic enzymes

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[1]John E. Bethune, The Adrenal Cortex (Michigan: The Upjohn Company, 1974), p. 32.

[2]Ibid.

into the cell thereby preventing the destruction of intracellular contents. Endotoxins, x-rays and ischemia are three known factors which induce the disruption of lysosomal membranes; therefore, corticosteroids are beneficial in the treatment of these conditions.[1]

5. Anti-stress effects: During times of stress, such as a surgical procedure or an auto accident, the adrenal cortex responds naturally by increasing its secretion of corticosteroids to help the body meet the demands of the stress. There is, however, a limit to the adrenals' ability to respond in a stressful situation. When the demands of the stressors supercede the adrenals' capacity to produce corticosteroids, pharmacologic agents must be given to meet the deficit. Gram negative septicemia and septic shock are two such overwhelming stressors.[2] The body's response to overwhelming stress is vascular collapse. The corticosteroids are used to help reduce this response, although no definite theory on their mode of action has been established. Suggested modes of action are that the glucocorticoids enhance the vasoconstrictive action of norepinephrine, but that they prevent excessive

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[1]John E. Bethune, The Adrenal Cortex (Michigan: The Upjohn Company, 1974), p. 32.

[2]Ibid., p. 33.

vasoconstriction which leads to tissue ischemia. This effect is thought to occur by a direct action on vascular smooth muscle and by directly stimulating the myocardium.[1] Corticosteroids may also exert metabolic effects by preventing lactate accumulation, suppressing histamine release, inducing key oxidative enzyme formation, directly antagonizing toxins and kinins, and directly stabilizing lysosomal membranes.[2]

The mineralocorticoids are used mainly in conjunction with replacement therapy in cases of a hypofunctioning adrenal cortex.

Despite the diverse conditions for which corticosteroid therapy is indicated, and the therapeutic effects of these drugs, several conditions exist in which therapy is either absolutely or relatively contraindicated. Absolute contraindications are systemic fungal infections, ocular herpes simplex, acute psychoses, and herpes simplex keratitis.[3] Table 2 is a comprehensive list of indications and contraindications for the use of corticosteroid therapy.

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[1]John E. Bethune, The Adrenal Cortex (Michigan: The Upjohn Company, 1974), p. 33.

[2]Ibid.

[3]Profiles of Three Corticosteroids (Pennsylvania: Division of Merck and Company, Inc., 1972), pp. 17-13.

TABLE 2

Indications and Contraindications for Use of Adrenocorticosteroids

Indications	Contraindications
Effective:	Systemic fungal infections
1. Endocrine disorders	Ocular herpes simplex
Primary or secondary adrenocortical insufficiency	Acute psychoses
	Herpes simplex keratitis
Acute adrenocortical insufficiency	Use with caution in:
	Active or latent peptic ulcer
Preoperatively and postoperatively, and in severe trauma of illness in patients with adrenocortical insufficiency	Chickenpox
	Congestive heart failure
	Diabetes Mellitus
	Diverticulitis
	Fresh intestinal anastomoses
Shock which is unresponsive to conventional therapy if adrenocortical insufficiency is suspected	Hypertension
	Myasthenia gravis
	Osteoporosis
	Pregnancy
Congenital adrenal hyperplasia	Psychotic tendencies
	Renal insufficiency
Nonsuppurative thyroiditis	Thromboembolic tendencies
	Vaccinia
2. Rheumatic disorders	Cushing's syndrome
For use in acute episode in:	Fungal diseases
	Tuberculosis
Post-traumatic osteoarthritis	
Synovitis of osteoarthritis	
Rheumatoid arthritis	
Acute and subacute bursitis	
Epicondylitis	
Acute tenosynovitis	
Acute gouty arthritis	
Psoriatic arthritis	
Ankylosing spondylitis	
Juvenile rheumatoid arthritis	

TABLE 2 (continued)

Indications	Contraindications
3. Collagen diseases During an exacerbation or as maintenance in cases of: Systemic lupus erythematosus Acute rheumatic carditis Systemic dermatomyositis Periarteritis nodosa	
4. Dermatologic diseases Pemphigus Severe erythema multiforme Exfoliative dermatitis Bullous dermatitis herpetiformis Severe seborrheic dermatitis	
5. Allergic states Bronchial asthma Contact dermatitis Atropic dermatitis Serum sickness Allergic rhinitis Drug hypersensitivity reactions Transfusion reactions Laryngeal edema	
6. Opthalamic diseases Allergic conjunctivitis Keratitis Herpes zoster Iritis Chorioretinitis Optic neurtitis	
7. Gastrointestinal diseases Ulcerative colitis Regional enteritis	

TABLE 2 (continued)

Indications	Contraindications
8. Respiratory diseases Berylliosis Aspiration pneumonia	
9. Hematologic diseases Hemolytic anemia Thrombocytopenic purpura Erythroblastopenia Hypoplastic anemia	
10. Neoplastic diseases Hypercalcemia associated with cancer Palliation in leukemias and lymphomas	
11. Edematous state To induce diuresis in nephrotic syndrome Ascites To prevent and treat cerebral edema	
12. Immunosuppressant agents Transplant procedures	
13. Neurologic disorders Multiple sclerosis	
14. Probably and possibly effective uses are being determined Acute shock in overwhelming infections	

SOURCES: Profiles of Three Corticosteroids (Pennsylvania: Division of Merck and Company, Inc., 1972):7-13.

Windsor C. Cutting, Handbook of Pharmacology, 5th ed. (New York: Appleton-Century-Crofts, 1972):339.

Mary Evans Hamdi, "Nursing Intervention for Patients Receiving Corticosteroid Therapy," in Advanced Concepts in Clinical Nursing, ed. Kay Corman Kintzel (Philadelphia: J. B. Lippincott Company, 1971):237.

The choice of a particular corticosteroid preparation to be used in a given situation depends primarily on whether or not mineralocorticoid effects are desired. For example, in the treatment of Addison's disease both glucocorticoid and mineralocorticoid effects are necessary to act as replacement substances for a hypofunctioning adrenal cortex, while only glucocorticoid properties are needed in the treatment of rheumatoid arthritis.[1] The cost of the medication is a second consideration in the selection of a preparation. Some of the newer synthetic corticosteroids are more expensive than the original preparations and are basically comparable in their effects.[2] If the drug is needed for a long period of time, it is desirable to use the least expensive preparation possible. A final consideration in selecting a corticosteroid compound is the route by which the drug is to be administered. This is a most important consideration in as much as all preparations are not equally absorbed by all routes.[3]

The dosages used in corticosteroid therapy are highly individualized. The proper dose for each patient is determined by closely observing for the desired therapeutic

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[1]Mary Evans Hamdi, "Nursing Interventions for Patients Receiving Corticosteroid Therapy," in Advanced Concepts in Clinical Nursing, ed. Kay Corman Kintzel. (Philadelphia: J. B. Lippincott Company, 1971), p. 239.

[2]Ibid.

[3]Ibid.

effects while also observing for any side effects. The dose is then adjusted according to these observations.[1]

Corticosteroids may be administered by numerous routes. The route of administration is determined mainly by whether a systemic or a local effect is desired. Whenever possible, corticosteroids are administered locally to minimize the absorption and therefore the systemic complications which may occur.[2] Corticosteroids may be administered by the oral, intramuscular, intravenous, subcutaneous, sub-lingual, topical, rectal, inhalation, intraocular, intrasynovial, and intra-articular routes. Each route of administration has advantages and disadvantages which should be considered prior to initiating therapy. For example, a topical preparation may be necessary and convenient to use; however, if it is being used on a relatively large area with an occlusive dressing, plasma cortisol levels may decrease indicating that the hypothalamic-adrenal system has been suppressed.[3] Also, despite the convenience in the use of oral preparations, a potentially dangerous side effect of gastro-intestinal ulcers may result.

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[1]Mary Evans Hamdi, "Nursing Interventions for Patients Receiving Corticosteroid Therapy," in Advanced Concepts in Clinical Nursing, ed. Kay Corman Kintzel. Philadelphia: J. B. Lippincott Company, 1971), p. 239.

[2]Ibid.

[3]Ibid.

The schedule for the administration of corticosteroid drugs depends greatly on whether a constant blood level or a normal circadian rhythm is desired. To maintain a constant blood level the drug may be given in divided doses over a twenty-four hour period, whereas higher doses are ordered in the morning tapering to lower doses in the evening in an attempt to mimic the normal circadian rhythm.[1] In mimicing the circadian rhythm, the last dose is usually given no later than 6 p.m. in order to prevent insomnia. When suppression of the pituitary-adrenal system is desired, the last dose is usually given as close to midnight as is possible. Corticosteroid therapy may be ordered on an intermittent schedule of every other day. The rationale for such a regime is based on the knowledge that the therapeutic effects of corticosteroids last longer than the metabolic effects.[2] The advantages of this routine are there is a lower incidence of side-effects and a better preservation of the pituitary-adrenal system while the withdrawal of corticosteroid therapy is facilitated.[3] The

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[1]Mary Evans Hamdi, "Nursing Interventions for Patients Receiving Corticosteroid Therapy," in Advanced Concepts in Clinical Nursing, ed. Kay Corman Kintzel. (Philadelphia: J. B. Lippincott Company, 1971), p. 240.

[2]Ibid.

[3]Ibid.

withdrawal of corticosteroid therapy should be accomplished gradually. Time must be allowed for the normal pituitary-adrenal system to restore itself to a full functional capacity. Corticosteroid therapy should never be abruptly discontinued as cardiovascular collapse may result if the adrenal cortex is not able to produce sufficient quantities of corticosteroids. This is a direct result of adrenal suppression by the administration of exogenous corticosteroids.[1]

Although there are numerous beneficial effects of corticosteroid therapy, caution must be exercised in the utilization of these drugs as many side-effects accompany the therapeutic effects. Side-effects may range in severity from acne to death. Table 3 provides a comprehensive list of the numerous side-effects of corticosteroid therapy.

Nursing Care of the Patient Receiving Adrenocorticosteroid Therapy

When the nursing profession came into existence, its practitioners were bound by numerous rules and regulations. Frequently these rules were established by people

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[1]Mary Evans Hamdi, "Nursing Interventions for Patients Receiving Corticosteroid Therapy," in Advanced Concepts in Clinical Nursing, ed. Kay Corman Kintzel. (Philadelphia: J. B. Lippincott Company, 1971), p. 240.

TABLE 3

Side Effects of Adrenocorticosteroid Therapy

1. Fluid and electrolyte disturbances:
   - Sodium retention
   - Fluid retention
   - Congestive heart failure in susceptible patients
   - Potassium loss
   - Hypokalemic alkalosis
   - Hypertension
   - Hypotension or shock-like reaction
   - Calcium and phosphorus loss

2. Musculoskeletal:
   - Muscle weakness
   - Steroid myopathy
   - Loss of muscle mass
   - Osteoporosis
   - Vertebral compression fractures
   - Aseptic necrosis of femoral and humeral heads
   - Pathological fractures of long bones
   - Negative nitrogen balance

3. Gastrointestinal:
   - Peptic ulcer with possible perforation and hemorrhage
   - Pancreatitis
   - Abdominal distention
   - Ulcerative esophagitis

4. Dermatologic:
   - Impaired wound healing
   - Thin fragile skin
   - Petechiae and ecchymoses
   - Erythema
   - Increased sweating
   - Burning or tingling especially in perineal area after IV injection

5. Neurological:
   - Convulsions
   - Increased intracranial pressure with papilledema after treatment
   - Vertigo
   - Headache
   - Insomnia
   - Psychoses

TABLE 3 (continued)

6. Endocrine:
   - Menstrual irregularities
   - Development of Cushing syndrome
   - Suppression of growth in children
   - Secondary adrenocortical and pituitary unresponsiveness, partially in times of stress such as trauma, surgery, or illness
   - Decreased carbohydrate tolerance
   - Signs of latent diabetes mellitus
   - Increased requirements for insulin or oral hypoglycemic agents in diabetes
   - Hypothyroidism

7. Opthalmic:
   - Posterior subcapsular cataracts
   - Increased intraocular pressure
   - Glaucoma
   - Exopthalmos

8. Other Suppressed Immune Mechanisms:
   - Hypersensitivity
   - Thromboembolism
   - Mask signs of infection
   - The following are due to parenteral therapy:
     - Hyperpigmentation or hypopigmentation
     - Subcutaneous atrophy
     - Sterile abscess
     - Formation of crystals

SOURCES: Profiles of Three Corticosteroids (Pennsylvania: Division of Merck and Company, Inc., 1972):17-20.

Windsor C. Cutting, Handbook of Pharmacology, 5th ed. (New York: Appleton-Century-Crofts, 1972):338.

outside of nursing. For many years nursing adhered to old traditions and customs, one of them being that the nurse was totally dependent upon the physician to make decisions. Today the nursing profession is assuming an interdependent relationship with other health professions and no longer is solely dependent upon the physician as a decision maker. Nurses now make decisions based upon their scientific knowledge and clinical assessments. Nurses utilize their skills to identify patients' problems and needs and to find ways to solve these problems or satisfy these needs.[1] Nurses are now attempting to evolve from the once flourishing "rule-oriented culture" to a "knowledge-oriented culture."[2] The rationale for such a transition is based on the premise that knowledge is the foundation upon which nursing practice is built. The practice of nursing is structured around the nursing process, while knowledge utilization is the foundation of the nursing process.

The nursing process is a logical sequence of steps which involves the collection of data, the analysis of data to identify patient needs, the development of a plan to meet those needs, implementing the plan, and evaluating

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[1]Carrie B. Lenburg, "Nursing and Education," R. N. Magazine 39 (March 1976):21.

[2]Ibid., p. 22.

the outcome of the plan.[1] A background in the biological, psychological, and sociological sciences is essential in order to effectively utilize the nursing process. This preparation also allows the nurse practitioner to carry out the many functions of a professional nurse which, according to McManus, are

> 1) the identification or diagnosis of the nursing problem and the recognition of its interrelated aspects, and 2) the deciding upon a course of nursing action to be followed for the solution of the problem in light of immediate and long term objectives of nursing, with regard to prevention of illness, direct care, rehabilitation and promotion of the highest standards of health possible for the individual.[2]

The administration of medications is one responsibility of the nurse. In performing this duty the nurse must be aware of the drugs' pharmacologic effects, side-effects, administration techniques, and other nursing implications which accompany the use of the drug. The adrenocorticosteroids are no exception. Their complicated pharmacologic effects and subsequent side-effects demand that the nurse use the nursing process concurrent with her knowledge of adrenocorticosteroid pharmacology to continually evaluate the patient's response to therapy. The nurse's assessment

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[1]Eileen Pearlman Becknell and Dorothy M. Smith, System of Nursing Practice (Philadelphia: F. A. Davis Company, 1975), p. 121.

[2]Virginia C. Conley, Curriculum and Instruction in Nursing (Boston: Little, Brown and Company, 1973), p. 127.

skills are the most vital component in the care of these patients. Assessments must be made continually to lessen the occurrance of any dangerous side-effects and to plan nursing interventions which are indicated by the patient's condition.

There are primarily five roles which the nurse assumes in the care of the patient receiving corticosteroid therapy. These roles are the administrator of the drug, the monitor of the drug's effects, the preventor of complications, the assessor and planner of care and, finally, the patient educator.[1] The successful fulfillment of each role is dependent upon the nurse's knowledge of corticosteroid pharmacology.

Prior to initiating corticosteroid therapy, the physician may make several tests which will help him to rule out any existing conditions which may complicate or be contraindicated for a patient receiving corticosteroid therapy. A complete history and physical exam should be done to establish a base line for each patient. The remainder of the preliminary work-up should consist of a tuberculin skin test to determine the need for anti-tuberculin drugs, a glucose tolerance test to determine

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[1]Mary Evans Hamdi, "Nursing Interventions for Patients Receiving Corticosteroid Therapy," in Advanced Concepts in Clinical Nursing, ed. Kay Corman Kintzel. (Philadelphia: J. B. Lippincott Company, 1971), p. 244.

the existence of diabetes mellitus, and an upper gastrointestinal x-ray series to rule out the presence of peptic ulcers.[1] Frequently, the patient's level of adrenocorticosteroids is evaluated before long term corticosteroid therapy is initiated. Throughout the preliminary testing period, the nurse should provide explanations of why the tests must be done, what the test procedures involve, and answer any questions the patient may have. She should continually provide emotional support and encouragement for these patients.

Once a patient has been thoroughly evaluated by the physician and the decision to begin corticosteroid therapy has been made, the physician will order the preparation, the dosage, the route, and the time of administration. The nurse's role of administrator of the drug now comes into play. The proper administration of a corticosteroid drug requires the nurse to be aware of the following:

1. Which preparation, dose, route of administration, and time of administration is desired by the physician
2. What is expected of the therapy or the purpose for which the drug was prescribed

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[1]Mary Evans Hamdi, "Nursing Interventions for Patients Receiving Corticosteroid Therapy," in Advanced Concepts in Clinical Nursing, ed. Kay Corman Kintzel. (Philadelphia: J. B. Lippincott Company, 1971), p. 237, 239.

3. The pharmacologic effects of the preparation being used
4. What the advantages and disadvantages are for the selected route of administration (See Table 4.)
5. What the proper administration techniques are, i.e., intravenous administration requires knowledge of how rapidly the drug may be infused and an awareness that corticosteroids are incompatible in solution with numerous other drugs (See Table 5.)
6. Impinging stressors on the patient, especially during the time when corticosteroids are being gradually withdrawn
7. The signs and symptoms of adrenal insufficiency during the time of corticosteroid therapy withdrawal, i.e., tiredness, muscular weakness, lethargy, hypotension, nausea, vomiting, diarrhea, headache
8. The patient's need for reassurance during the administration of a therapeutic agent
9. Her roles of assessor, monitor, preventor of complications, and patient educator

The nurses' roles of monitor of the drugs' effects, preventor of complications, and patient educator are a result of the corticosteroids' pharmacologic effects and their subsequent complications. The side-effects of corticosteroid therapy may be classified according to their

TABLE 4

Advantages and Disadvantages for Six Routes of Administration of Adrenocorticosteroid Drugs

Oral	Intramuscular	Intravenous
Advantages 1. Convenient Disadvantages 1. Peptic ulcers may occur	Advantages 1. Rapid onset with prolonged effect Disadvantages 1. Must be given deep in gluteal muscle not in the deltoid muscle 2. Sterile abscess, subcutaneous atrophy and hyper/hypopigmentation may occur	Advantages 1. Rapid onset of action Disadvantages 1. An indwelling catheter is needed 2. Close monitoring is required to deliver precise amount of medication 3. Corticosteroids are incompatible in solution with many other drugs.
Sublingual	**Topical**	**Intraocular**
Advantages 1. There is a more rapid arrival of the medication in the pulmonary circulation than is obtained from the oral route.	Advatages 1. The medication may be applied directly to the desired area.	Advantages 1. The medication may be applied directly to the desired area.

TABLE 4 (continued)

Sublingual	Topical	Intraocular
2. A smaller dose is needed. 3. There is a longer lasting effect.		
Disadvantages 1. Patient may not be capable of holding medication sublingually.	Disadvantages 1. When used on large areas with an occlusive dressing there is a systemic absorption with a subsequent adrenopituitary suppression.	Disadvantages 1. Corneal ulceration may occur. 2. Increased intraocular pressure may occur. 3. Herpetic infections of the eye may occur.

SOURCE: Mary Evans Hamdi, "Nursing Interventions for Patients Receiving Corticosteroid Therapy," in Advanced Concepts in Clinical Nursing, ed. Kay Corman Kintzel. (Philadelphia: J. B. Lippincott Company, 1971):239-240.

TABLE 5

I.V. Admixture and Compatibility of Two Commonly Given Adrenocorticosteroids

Drug Can Be Mixed With	Cannot Be Mixed With
Hydrocortisone sodium succinate (Solu-Cortef)	
Common I.V. solutions	Amobarbital sodium
Aminophyllin	Chloramphenicol sodium succinate
Calcium gluceptate	Dimenhydrinate (dramamine)
Carbenicillin disodium	Ephedrine sulphate
Corticotropin	Heparin sodium
Erythromycin	Insulin
Fluorouracil	Kanamycin
Penicillin G	Metaraminol bitartrate
Potassium chloride	Methicillin sodium
Sodium iodide	Methylprednisolone sodium succinate
	Multiple vitamins
	Novobiocin sodium
	Oxytetracycline HCL
	Pentobarbital sodium
	Phytonadione
	Phenobarbital sodium
	Prochlorperazine (compazine)
	Tetracycline HCL
	Vancomycin
	Vitamins B and C
	Chlorpromazine HCL
	Colistimethate sodium
	Hyaluronidase
	Hydroxyzine HCL (atarax)
	Meperidine HCL
	Promazine HCL (sparine)
	Promethazine HCL (phenergan)
	Protein hydrolysate

TABLE 5 (continued)

Drug Can Be Mixed With	Cannot Be Mixed With
Methylpredisolone sodium succinate (Solu-Medrol)	
Calcium gluceptate	Aminophyllin
Carbenicillin disodium	Chloramphenicol sodium succinate
Corticotropin	Heparin sodium
D5S	Hydrocortisone sodium succinate
D5W	Menadione sodium bisulfite
Erythromycin	Phytonadion
Mannitol	Promethazine HCL (phenergan)
Penicillin G	Tetracycline HCL
Potassium HCL	Vitamins B and C
Sodium chloride	Chlorpromazine HCL
Sodium iodide	Digitoxin
	Diphenhydramine HCL (benadryl)
	Meperidine HCL (demerol)
	Metaraminal bitartrate (aramine)

SOURCES: A Guide to I.V. Admixture Compatibility (New Jersey: Medical Economics Company, 1973):27, 35.
Betty L. Gahart, Intravenous Medications (Saint Louis: C. V. Mosby Company, 1973):72, 104.

effects on body systems. To facilitate assimilation of the copious amount of material concerning the nursing care of the patient receiving corticosteroid therapy, a table has been compiled. The table contains the side-effects of corticosteroid therapy which have been classified according to body system. The nursing implications which are derived from the corticosteroids' side-effects, have been divided into the nurse's roles of monitor, preventor of complications and patient educator. (See Appendix A.)

Summary

The nurse plays a major part in helping to maximize the therapeutic effects of corticosteroid therapy while minimizing their potentially dangerous side-effects. Effective use of the nursing process, coupled with a good background knowledge of adrenocorticosteroid pharmacology, provides the basis from which the nurse may assume her roles of monitor of the drugs' effects, the preventor of complications, the assessor and planner of care and patient educator.

The procedure for collection and treatment of data is described in Chapter III. The data were collected by the utilization of two original tools. Chapter IV describes the analysis of data. Analysis was accomplished by calculating correlation and linear regression coefficients. In

Chapter V, a summary of findings and conclusions were drawn as a result of this study. Implications for nursing and recommendations for further studies are included.

CHAPTER III

PROCEDURE FOR COLLECTION AND TREATMENT OF DATA

A nonexperimental explanatory research design[1] was used in this study. A causal relationship was being sought between the nurses' knowledge in adrenocorticosteroid physiology, side-effects, nursing implications, and administration techniques, and the documented nursing assessments which were made on patients receiving an adrenocorticosteroid drug.

Setting

The setting for this study was an 800 bed teaching hospital. This institution is a county, acute-care facility located in the Southwest. Written permission was obtained to use this institution as the setting for the study. (See Appendix B.)

Population and Sampling Technique

Through the use of the convenience sampling technique,[2] seven general adult non-intensive care medical-surgical units were sampled. During the twenty-four hour

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[1] Faye G. Abdellah and Eugene Levine, Better Patient Care Through Nursing Research (New York: The MacMillan Company, 1965), p. 212.

[2] Ibid., p. 310.

period the audit tool was used to collect data, the nurse who cared for the patient on each of the three shifts and who worked on the sample unit at least forty hours per week was asked to complete a questionnaire.

The patients on the selected units were chosen according to the following criteria:

1. Patients who were receiving a form of an adrenocorticosteroid for at least the past twenty-four hours, but no longer than three weeks on a schedule of at least twice a day
2. Patients who were administered an adrenocorticosteroid drug by the oral, intramuscular, or intravenous routes
3. Patients who were eighteen years of age or older
4. Patients who signed the Texas Woman's University's Form B, the Consent to Act as a Subject for Research and Investigation after a brief explanation of the study was verbally given
5. Patients who were mentally and physically capable of answering questions

Tools

Two original tools were used in this study. The first tool was a checklist to audit the documented nursing assessments, with a separate checklist

to interview the patient. It was composed after taking a course in nursing audit in undergraduate school and also after completing an extensive review of literature on adrenocorticosteroid physiology, pharmacology, side-effects, nursing implications and administration techniques. (See Appendix C.) The checklists' contents were validated by having an endocrinologist review them. The endocrinologist commented that the checklists definitely reflected the major anticipated physiologic alterations in a patient receiving corticosteroid therapy; however, he felt that the patient interview checklist was the most valuable portion of the checklist. The endocrinologist stated that direct patient input into what he was perceiving as physiological changes was an important indicator of what side-effects may actually be occurring. The tool was then tested for the adequacy of its construction by auditing ten patients' charts and by also interviewing the same ten patients. The results of this procedure showed that there was a need for minor alterations in the wording of some of the criteria. These changes were made and the final validated checklist emerged. The validated checklist was then used to gather information to evaluate what type of documented nursing assessments were made in relation to the particular problems of patients who are receiving adrenocorticosteroid therapy.

A questionnaire on adrenocorticosteroid therapy comprised the second tool used in this study. (See Appendix D.) This questionnaire had a demographic information section and a test section. The test portion of the questionnaire was composed as a result of an extensive review of literature on adrenocorticosteroid physiology, pharmacology, side-effects, nursing implications and administration techniques, and the information gained from a graduate class on tests and measurement. Its content was validated by having a physiologist review it, while its technical construction was reviewed by an expert in test construction. Revisions were suggested by both experts. The physiologist made the following suggested alterations to help clarify specific questions:

1. On question #2 alter "primary action" to "primary effects"
2. On question #7 add to the stem that renin is released in a "normal person" under certain circumstances as it was found experimentally that renin can be stimulated to be released in people who have high renal pressures rather than low
3. On question #8b alter the word "metabolism" to "balance"

The overall comment was that the test was good. The expert in test construction suggested that all the test questions be given 5 choices instead of 4 to reduce the chance of correctly guessing the answer.

All suggested revisions were made, and the test was then pilot tested by five volunteers from a graduate medical-surgical nursing program. The five volunteers made the following comments and scores:

> Comments: The test read clearly; however, the content was difficult. They seemed to think that it was necessary to take some classes on the given subject since the test was so difficult to them. No comments were offered on any specific way in which to improve the test.
>
> Scores on the Pilot Test:
>
> | # Correct | % Based on Score of 100 |
> |---|---|
> | 7 | 48.0% |
> | | 41.5% |
> | | 41.5% |
> | 4 | 28.5% |
> | 4 | 28.5% |
>
> Average difficulty 37.6%.

This group's comments and scores were considered; however, no further revisions were made at that time. The test was then given to fifteen volunteer graduate nursing students in a medical-surgical program, to ensure that the test portion of the questionnaire had between a forty percent and a fifty percent difficulty. The overall difficulty of the test as calculated by the test scores of all the volunteers (twenty) who took the test was forty-two percent. The item analysis, item success, and discrimination results are contained in Appendix E. Analysis of the test tool can be summarized as follows:

> TEST DIFFICULTY
> Wanted a fifty percent difficulty average for test. Actually had a forty-two percent difficulty average on test

TEST DISCRIMINATION

Wanted at least a 2 point discrimination per item. Actually got a 2 point discrimination or better for all items except #4, 5, 11, and 12

A LOOK AT THE ITEMS AND THEIR DISTRACTORS

Most of the distractors got some votes. With only twenty volunteers to take the test, it would not be indicated at this time to re-do any of the distractors until they were tested further

TEST RELIABILITY

Reliability of test is .1 at this time

Data Collection

The data collection process was begun on July 23, 1976, and ended on August 7, 1976. The general adult non-intensive care medical-surgical units were scouted at least three times per week for patients who met the criteria. A total of thirteen units were searched for patients; however, only seven units actually had patients who were eligible to be audited.

The patients were selected by initially looking at the Kardex on the floor to ascertain whether or not they were receiving a corticosteroid drug. If a patient was being given a corticosteroid drug, the Kardex was checked for the route of administration, the length of time the drug had been given, the schedule the drug was being administered on, and lastly, the patient's age. The doctor's orders on the chart were then checked to insure that the information on the Kardex was correct.

If the patient under consideration met all the criteria to this point, the charge nurse was asked whether or not the patient was able to answer questions. If the charge nurse responded "yes," then the chart audit was done.

The audit procedure was accomplished by using the chart checklist. The patient's chart was checked for the desired information during the twenty-four hour period immediately preceding the time of audit. Once this procedure was completed, the charge nurse was asked which registered nurse was responsible for that patient's care on each shift during the twenty-four hour period the audit was done. The names of these nurses were then recorded on the chart checklist. The patient's name and hospital unit were also recorded on the chart checklist.

The patient was then asked to be a participant in the study. He was given a brief description of the study and was also informed that he could withdraw at any time.

If the patient gave permission to participate in the study and signed the consent form (see Appendix F), the Patient Interview Checklist was used. After interviewing the patient to find out what particular signs and/or symptoms he was noticing, the nurses' notes were checked again to see if any note was made regarding the symptoms the patient was having. The audit process was done until twenty patients' charts were audited.

After completing audits on twenty patients, the validated questionnaire was given to the registered nurses who had cared for a patient who was audited. The selected nurses were given the questionnaire sometime during their shift. At that time each of the nurses was assigned a number by which she could be identified throughout the study. A list of the assigned numbers with the corresponding nurses' names, as well as a list of patients' names and corresponding assigned numbers, was kept until the data collecting procedure was completed. Throughout the data collection, care was taken to recognize and maintain the identity of the specific nurse-patient relationships on which the interest was centered. This list was kept strictly confidential at all times. The nurses were instructed to read the introduction on the questionnaire carefully and were then given the opportunity to ask any questions. The nurses were then instructed to complete their questionnaires and return them the following day to a folder which was left with the ward clerk. A total number of twenty-four nurses were asked to fill out questionnaires.

Treatment of Data

The test portion of the questionnaire was scored by dividing the number of correct responses by the total number of questions on the test. The two checklists were

scored in the following manner. The sum of scores on the two checklists was named the patient-assessment score. Items considered not applicable were ignored. Those items or criteria on the chart checklist which were to be satisfied once a shift were marked not applicable as they occurred. A nurse received one point for each applicable item or criterion which was required to be satisfied once each shift, if that nurse satisfied that criterion. Zero points were given for each criterion the nurse failed to satisfy. An item or criterion which was required to be satisfied once on a daily basis was not considered applicable for a given nurse if one of the other nurses assigned to that patient during the twenty-four hour period satisfied the criterion and that nurse did not. For each applicable criterion which must be satisfied daily, a nurse received one point for each criterion she satisfied. Zero points were given to all the nurses assigned to that patient if that criterion was not satisfied by at least one nurse. Items on the patient interview checklist were scored in the same manner as described above. If a patient responded "No" to having a symptom, that item was considered not applicable for all the nurses assigned to that patient. If a patient responded "Yes" to having a symptom, then each nurse who recorded the presence of that symptom in that patient received one point. Zero points were given to

all the nurses if the symptom was not recorded by at least one nurse. If a nurse failed to record the existence of a symptom, but one of the nurses caring for that patient during that twenty-four hour period did record the presence of that symptom, the item was scored not applicable for the nurse who failed to record the presence of that symptom.

Using the identity of the nurse-patient relationships which had been recognized during the data collection, each patient-assessment score was paired with the test score of the nurse involved in each particular relationship. Note that several paired nurses' test and patient-assessment score data points could correspond to each nurse since each nurse cared for several patients. Each such nurse-patient relationship could result in a paired data point.

A nurse's knowledge was measured by the test portion of the questionnaire on adrenocorticosteroid therapy. Documented nursing assessments were measured by a chart checklist and patient interview checklist. A correlation coefficient and linear regression coefficients were computed. This was accomplished by using the results from the test portion of the questionnaire and the data compiled from the checklists.

A product-moment correlation coefficient was calculated to determine whether or not there was a relationship between nurses' knowledge in adrenocorticosteroid

physiology, side-effects, nursing implications and administration techniques, and the documented nursing assessments which were made on patients receiving an adrenocorticosteroid drug. The formula for the product-moment correlation coefficient is

$$r = \frac{n\sum_{i=1}^{n} x_i y_i - \left(\sum_{i=1}^{n} x_i\right)\left(\sum_{i=1}^{n} y_i\right)}{\sqrt{\left[n\sum_{i=1}^{n} x_i^2 - \left(\sum_{i=1}^{n} x_i\right)^2\right]\left[n\sum_{i=1}^{n} y_i^2 - \left(\sum_{i=1}^{n} y_i\right)^2\right]}}$$

where x_i is the nurse's test score from the i^{th} nurse-patient relationship and y_i is the patient-assessment score from the i^{th} nurse-patient relationship.

The coefficient of linear correlation is a statistical indicator of the degree of closeness to a linear relationship between the nurses' test scores and the patient-assessment scores. Two properties of the correlation coefficient manifested are that it is a pure number, independent of the scales in which the two scores were measured and that it ranges between -1.0 and +1.0. The greater absolute values would indicate the stronger linear correlations.

> Perfect correlation (r=1.0) rarely occurs in biological data, though values as high as 0.99 are not unheard of. Each field of investigation has its own range of coefficients. Inherited characteristics such as

height ordinarily have correlations between 0.35 and 0.55.[1]

It was of interest to determine whether there was any statistical interaction between shifts of nurses. An elementary way to do this was to segregate the nurse-patient relationship data pairs by shift and to calculate the equivalent correlation coefficient for each of the three shifts of nurses.

If the coefficient of correlation shows a significant level of linearity between the test scores and the patient-assessment scores, then an attempt will be made to fit a linear regression model to the data. An appropriate model would be of the form $y_i = a + bx_i$ where y_i is the patient-assessment score of the i^{th} observed nurse-patient relationship, x_i is the nurse's test score from the i^{th} observed nurse-patient relationship, b is the slope of the regression line, and a is the y-intercept of the regression line. The formulas for the coefficients are:

$$b = \frac{n\sum_{i=1}^{n} x_i y_i - \left(\sum_{i=1}^{n} x_i\right)\left(\sum_{i=1}^{n} y_i\right)}{n\sum_{i=1}^{n} x_i^2 - \left(\sum_{i=1}^{n} x_i\right)^2} \quad \text{and}$$

$a = y - b\,x$ where x_i and are as defined above and x and y are the respective average test scores and patient-assessment scores.

[1]George W. Snedecor and William G. Cochran, Statistical Methods, 6th ed. (Iowa: The Iowa State University Press, 1967), p. 175.

Following the analysis of the paired test scores and patient-assessment scores, the test scores were examined without regard to the patient-assessment scores and the patient-assessment scores were examined without specific regard to the test scores. The knowledge test was described from an educational testing point of view. Then such routine statistics as the average, the median, the mode, and the standard deviation of test scores were calculated to statistically quantify the univariate test scores. Subsequently, the nurses' test scores were segregated by various demographic variables. An analysis of variance was used to determine from the variation of the data whether the segregating variable is responsible for apparent differences between the groups or if the apparent differences are due to random variation of the data. The Duncan's Multiple Range Test was used to determine between which specific groups there was a significant difference.[1]

The Chart Checklist and the Patient Interview Checklist were evaluated from a testing and measurement vantage point. Then routine descriptive statistics (the mean, the median, the mode, the standard deviation) were enumerated for the patient-assessment scores derived from these checklists. A histogram of the patient-assessment

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[1]R. Lowell Wine, Statistics for Scientists and Engineers (New Jersey: Prentice Hall, Inc., 1964), p. 359.

scores was constructed to depict the distribution of patient-assessment scores in general.

To determine the influence of the three Basic Nursing Programs on the patient-assessment scores, an analysis of variance for nested classifications with unequal sample sizes was performed. The model under consideration is

$$y_{ijk} = \mu + a_i + b_{j(i)} + e_{ijk} \ , \ i=1,2,3; \ j=1,2,\ldots, n_i; \ k=1,2,\ldots, m_{j(i)}$$

where μ is an overall mean, a_i is a fixed effect due to the i^{th} nursing program, $b_{j(i)}$ is a random effect due to nesting nurses within each of the i categories, e_{ijk} are normally distributed random errors with zero mean and standard deviation one, n_i is the number of nurses in the i^{th} nursing program and $m_{j(i)}$ is the number of patient-assessment scores for the j-th nurse within the i^{th} nursing program.

Summary

A nonexperimental explanatory research design was used in this study. A causal relationship was being sought between the nurses' knowledge in adrenocorticosteroid physiology, side-effects, nursing implications and administration techniques, and the documented nursing assessments which were made on patients receiving an adrenocorticosteroid drug.

Chapter IV describes the analysis of data. Analysis was accomplished by calculating correlation and linear regression coefficients. In Chapter V, a summary of findings and conclusions are presented. Implications for nursing and recommendations for further studies are included.

CHAPTER IV

ANALYSIS OF DATA

Introduction

The analysis of data is presented in Chapter IV. The results and interpretations of the findings in this study are discussed. The statistics used to analyze the data are included.

The analysis of data was guided by the following purposes:

1. To determine what the nurses' level of knowledge was in adrenocorticosteroid physiology, side-effects, nursing implications and administration techniques
2. To determine what kind of documented nursing assessments were made on patients receiving adrenocorticosteroid therapy
3. To determine whether or not there was a relationship between nurses' knowledge in adrenocorticosteroid physiology, side-effects, nursing implications and administration techniques, and the documented nursing assessments which were made on patients receiving an adrenocorticosteroid drug

Characteristics of the Sample

A thirty-six point set of multivariate data on the basic nurse-patient relationships was extracted from the checklists and the questionnaires. The components of each point were the nurse's test score, a patient-assessment score, and various demographic items from the questionnaire. Some preliminary univariate descriptive statistics were calculated for the nurses' test scores and for the patient-assessment scores. Next a scatter plot (Figure 6) of the patient-assessment scores versus the test scores was made to give an idea of the nature of the bivariate data. See Appendix G for original data.

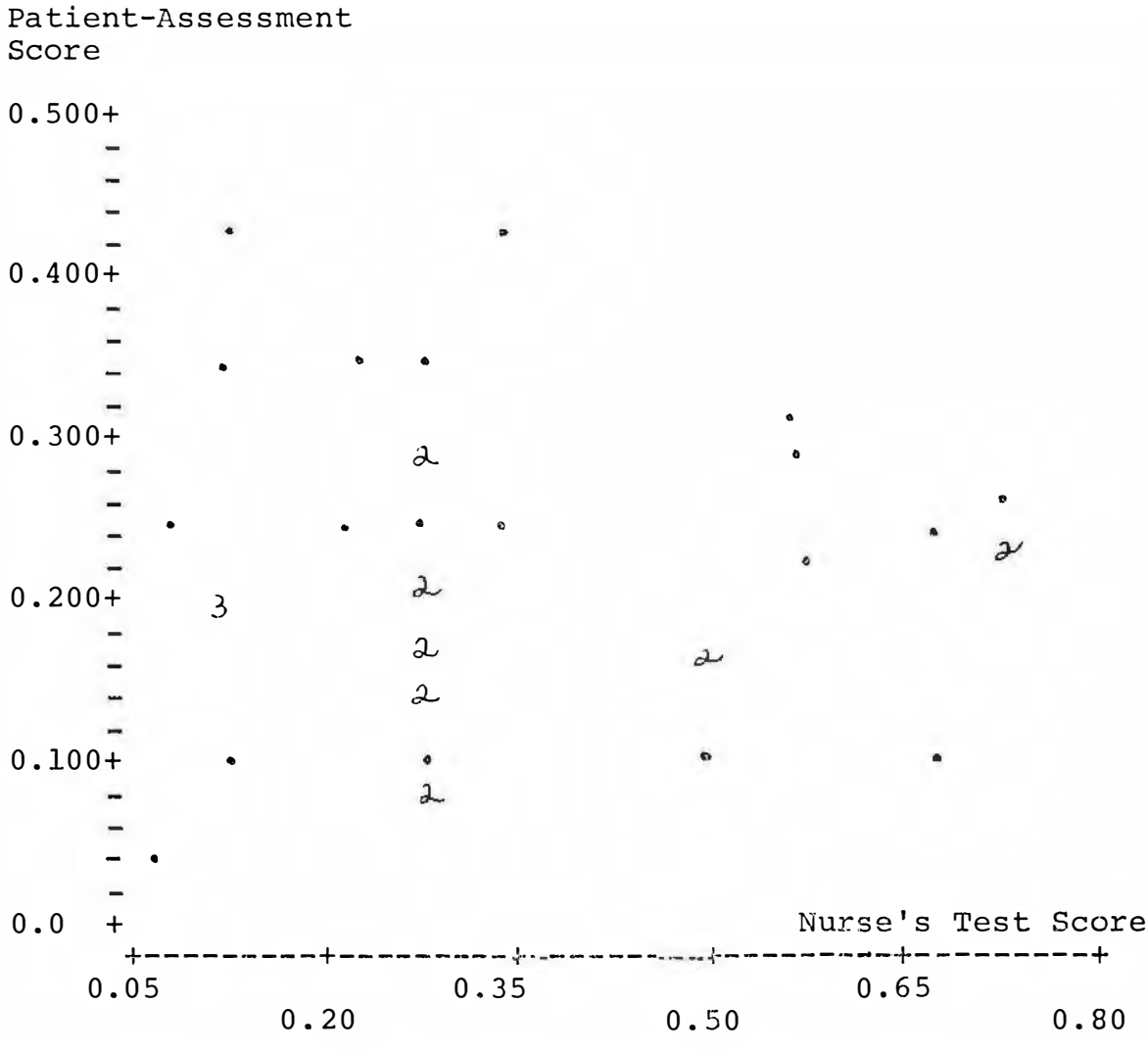
Presentation of Findings

The Linear Correlation Coefficient

The wide spread of the data indicated by the scatter plot is measured by the coefficient of linear correlation. The value of the coefficient for the data illustrated in the scatter-plot is $r=0.014$, so the degree of linear correlation in the data is negligible. Thus the linear regression coefficients calculated were statistically equivalent to zero. (See Figure 6.)

Figure 6

Scatter Plot of Patient-Assessment Score
Versus Nurses' Test Scores



Correlation coefficients were computed for each of three shifts. These coefficients are shown in Table 9. Each of these sample r values is not statistically different from zero at the $\alpha = .05$ level, with 3, 3, and 2 degrees of freedom. This means that from this data, one should not be inclined to reject the hypothesis that

patient-assessment scores are not linearly related to nurses' test scores.

TABLE 6

CORRELATION COEFFICIENTS BETWEEN NURSES' TEST SCORES AND PATIENT-ASSESSMENT SCORES BY SHIFT

Shift	7-3	3-11	11-7	Overall
	-0.495	-0.179	0.652	0.014

Evaluation of the Test Portion of the Questinnaire

Test Difficulty

The desired test difficulty was between a forty percent and fifty percent. The actual calculated test difficulty of the fourteen nurses who participated in the study was thirty-three percent. (See Table 7.) The initial pilot testing of twenty graduate students resulted in a forty-two percent test difficulty. Perhaps the thirty-three percent difficulty result indicates that the test was more difficult than was originally calculated or perhaps indicates that much guessing was done. These results should not be taken as a final indicator of the test's difficulty as the length of the test is rather short, and it has been administered to a total of only thirty-four people. It should be noted that some of the nurses completed their

TABLE 7

Item Success and Discrimination on Test Portion of Questionnaire

ITEM #	# OF HIGHS WHO GOT THE ITEM CORRECT (H)	# OF LOWS WHO GOT THE ITEM CORRECT (L)	H+L	H - L
1.	3	1	4/14 29%	2
2.	5	1	6/14 43%	4
3.	6	5	11/14 79%	1
4.	3	0	3/14 21%	3
5.	3	0	3/14 21%	3
6.	3	1	4/14 29%	2
7.	4	2	6/14 43%	2
8.	7	2	9/14 64%	5
9.	3	0	3/14 21%	3
10.	2	1	3/14 21%	1
11.	2	1	3/14 21%	1
12.	0	0	0/14 0	0
13.	4	0	4/14 29%	4
14.	4	4	8/14 57%	0

Desired discrimination is at least 2. Desired total success should be between 30% and 90%. Calculated average difficulty thirty-three percent.

test while on duty, whereas others took them home to complete. The setting in which the nurses completed their tests could have had a major influence on the test scores.

Item Discrimination

A two point or better discrimination was desired between the high and low scorers on each test item. Items 3, 10, 11, 12, and 14 got less than a two point discrimination. (See Table 7.) The initial pilot test revealed that items 4, 5, 11, and 12 got less than a two point discrimination. The fact that items 11 and 12 got low discriminations after being tested twice indicates that the items need to be reevaluated.

Items and Their Distractors

Out of a total of seventy possible choices, nineteen choices were not selected. (See Table 8.) The initial pilot test showed that ten choices were not selected. Seven of these choices were not selected in either group. An analysis of the seven choices which were not selected by either group is indicated to improve the power of the distractor.

Item Difficulty

Out of fourteen questions, five received between a forty-three percent and a seventy-nine percent difficulty. (See Table 9.) Four of these five questions are pure

TABLE 8

ITEM ANALYSIS ON TEST PORTION OF QUESTIONNAIRE

ITEM #	A High	A Low	B High	B Low	C High	C Low	D High	D Low	E High	E Low
1	0	2	1	2	3	2	3 *	1	0	0
Total	2		3		5		4		0	
2	5 *	1	0	0	1	2	1	2	0	2
Total	6		0		3		3		2	
3	1	0	6 *	5	0	2	0	0	0	0
Total	1		11		2		0		0	
4	0	0	0	2	4	5	3 *	0	0	0
Total	0		2		9		3		0	
5	1	1	0	2	3 *	0	0	1	3	3
Total	2		2		3		1		6	
6	0	0	0	0	0	5	3 *	1	4	1
Total	0		0		5		4		5	
7	4 *	2	2	0	1	4	0	1	0	0
Total	6		2		5		1		0	
8	0	3	7 *	2	0	1	0	0	0	1
Total	3		9		1		0		1	
9	4	2	0	0	0	2	3 *	0	0	3
Total	6		0		2		3		3	

TABLE 8 (continued)

ITEM #	A High	A Low	B High	B Low	C High	C Low	D High	D Low	E High	E Low
10	4	4	0	1	1	1	0	0	2 *	1
Total	8		1		2		0		3	
11	2 *	1	4	2	0	4	1	0	0	0
Total	3		6		4		1		0	
12	0	1	0	0	0 *	0	3	3	4	3
Total	1		0		0		6		7	
13	0	0	4 *	0	3	7	0	0	0	0
Total	0		4		10		0		0	
14	0	0	2	0	1	3	0	0	4 *	4
Total	0		2		4		0		8	

* indicates the correct answer for the given question.

TABLE 9

MAP OF ITEMS ON TEST PORTION OF QUESTIONNAIRE

ITEM #	CONTENT OF QUESTION	TYPE QUESTION	TOTAL # WHO GOT ITEM CORRECT H+L
1	physiology	knowledge	4/14 = 29%
2	physiology	knowledge	6/14 = 43%
3	physiology	knowledge	11/14 = 79%
4	side-effects	application	3/14 = 21%
5	side-effects	application	3/14 = 21%
6	physiology	knowledge	4/14 = 29%
7	physiology	knowledge	6/14 = 43%
8	physiology	knowledge	9/14 = 64%
9	side-effects	application	3/14 = 21%
10	side-effects	application	3/14 = 21%
11	side-effects	application	3/14 = 21%
12	physiology	application	0/14 = 0
13	administration of drug	application	4/14 = 29%
14	nursing implications	application	8/14 = 57%

knowledge questions about physiology. The fifth question required the application of knowledge in a nursing situati The test had a total of six knowledge questions and eight questions which required the application of knowledge. The nurses did much better in answering the pure knowledge questions than they did in answering the questions which required the application of knowledge. Perhaps this indicated that nurses possessed knowledge of physiology; howe they were unable to apply it.

Univariate Statistical Analysis of Nurses' Test Scores

The nurses' scores on the test were examined statistically by univariate procedures and through separate one-way analyses of variance applied to the Distribution of Nurses by Test Score and Basic Nursing Program, to the Distribution of Nurses by Test Score and Years of Experie and to the Distribution of Nurses by Test Score and Shift.

TABLE 10

HISTOGRAM OF NURSES' TEST SCORES

TEST SCORES (MIDPOINT OF THE INTERIOR)	NUMBER OF OBSERVATIONS	HISTOGRAM
0.10	4	****
0.20	1	*
0.30	4	****
0.40	1	*
0.50	1	*
0.60	2	**
0.70	1	*

The nurses' scores on the test portion of the questionnaires ranged from one to ten correct answers (from 0.071 to 0.714) on fourteen questions, with a mean score of 0.332, an average of slightly less than five correct answers. Four nurses achieved the modal score of four correct answers. The median score was also four correct answers (0.286) while the sample standard deviation of the fourteen scores was 0.201. (See Table 11.)

The only separate one-way analysis of variance of a distribution of nurses' test scores and a demographic characteristic which showed a significant result was the analysis of variance (ANOVA) of the Distribution of Nurses' by Test Scores and Basic Nursing Program; therefore, the only ANOVA table and Distribution reproduced is that one. Similar procedures were followed for the Distribution of Nurses by Test Score and Years of Experience and for the Distribution of Nurses by Test Score and Shift.

The analysis of variance of the Distribution of Nurses by Test Score and Basic Nursing Program tested the null hypothesis that mean scores of nurses in each of the represented kinds of nursing programs were all the same. Assuming random samples were taken from the three populations of nurses in each of the nursing programs, the probability of observing data with an associated calculated

TABLE 11

DISTRIBUTION OF NURSES BY TEST SCORE

AND BASIC NURSING PROGRAM

TEST SCORE	BASIC NURSING PROGRAM			
	Two-year associate degree	Three-year diploma	Four-year B.S. in Nursing	Total
1/14 (0.071)	-	1	-	1
2/14 (0.143)	2	1	-	3
3/14 (0.214)	-	1	-	1
4/14 (0.286)	-	4	-	4
5/14 (0.357)	-	-	1	1
7/14 (0.500)	-	-	1	1
8/14 (0.571)	-	-	1	1
9/14 (0.643)	-	-	1	1
10/14	-	-	1	1
Total # of nurses	2	7	5	14
Mean Score	0.143	0.225	0.557	-

F statistic greater than or equal to 18.345 is less than 0.01. The null hypothesis was rejected that all three populations have the same mean test score. This means that at least one of the test scores is different from the other two.

TABLE 12

ANALYSIS OF VARIANCE OF NURSES' TEST SCORES SEGREGATED BY BASIC NURSING PROGRAM

Source	Degrees of Freedom	Sum of Squares	Mean Square	F
Basic Nursing Program	2	0.405	0.203	18.435
Sampling Error	11	0.121	0.011	
Total	13	0.526		

To compare the mean test scores of nurses by Basic Nursing Program simultaneously, a Duncan's Multiple Range Test with unequal sample sizes was performed with $\alpha = \alpha_2 = .05$ for comparison of two means. Table 13 presents the results of this test.

TABLE 13

DUNCAN'S MULTIPLE RANGE TEST OF BASIC NURSING PROGRAMS*

Average Test Score		
2 yr. Asso.	3 yr. Dip.	4 yr. B.S.N.
.143	.225	.557

*Any averages underscored by the same line are statistically equal. Those not underscored by the same line are statistically unequal.

The analysis of variance of the Distribution of Nurses by Test Score and Years of Experience tested the null hypothesis that all nurses tested would score the same, regardless of their years of experience. The ANOVA indicated an F statistic of 3.194. Under purely random sampling from a population where the null hypothesis is true the observed result could have occurred between five percent and ten percent of the time. This was not deemed significant; it was concluded that the data did not call for rejection of the null hypothesis.

The separate one-way analysis of variance of the Distribution of Nurses by Test Score and Shift tested the hypothesis that the mean test scores of all the nurses tested were the same, regardless of shift. The ANOVA indicated an F statistic of 0.737, which is not statistically

significant. The data do not call for the rejection of the null hypothesis in favor of any alternative hypothesis.

Evaluation of the Chart Checklist and the Patient Interview Checklist

In looking at the compliance achieved for each criterion, a fifty percent or better compliance was attained on the following criteria:

1. Are the vital signs recorded at least once a shift
2. Is a total intake and output recorded each shift
7. Does patient have a surgical incision or a wound which is currently being treated
   If so, has a note been made concerning its healing progress within the past 24 hours

The remaining criteria had less than a fifty percent compliance.

The criteria that were met with at least a fifty percent compliance are those which are routinely met on patients in the hospital. On the criteria with less than a fifty percent compliance, some kind of nursing assessment would have to have been made in order to satisfy these criteria. The patients' physiological responses to the corticosteroid drugs were not adequately charted, as is demonstrated by low compliance scores in recording urine sugar and acetones, daily weights, skin conditions, mental status,

abnormal lab values, and changes in weights. No records were made in regard to how any of the drugs were administered, i.e., deep IM in gluteal area or with food or milk. There was a complete disregard to charting any of the signs and/or symptoms that the patients were experiencing. The low compliance to these criteria indicated that very few assessments of the patients were recorded. See Appendix H for a summary of the responses to items on the chart checklist and the patient interview checklist. Appendix I contains a summary of percentage compliance on the chart checklist and the patient-interview checklist.

Univariate Statistical Analysis of Patient-Assessment Scores

The patient-assessment scores were also analyzed as univariate data. The patient-assessment scores ranged from 0.038 to 0.429. The mean score was 0.205; the median score was 0.207; the modal score, 0.250, occurred five times. The sample standard deviation of the thirty-six scores was 0.093635.

TABLE 14

HISTOGRAM OF PATIENT-ASSESSMENT SCORES

PATIENT-ASSESSMENT SCORES (MIDPOINT OF INTERVAL)	NUMBER OF OBSERVATIONS	HISTOGRAM
0.050	1	*
0.100	6	******
0.150	6	******
0.200	8	********
0.250	6	******
0.300	4	****
0.350	3	***
0.400	1	*
0.450	1	*

Table 15 shows the distribution of nurses by program. The analysis of variance on this data tested the hypothesis of no difference in patient-assessment scores due to different Basic Nursing Programs. Since the computed F-ratio (.405) is less than one, H_o is not rejected. It is therefore concluded that there is no variation in patient-assessment scores due to the type of basic nursing program from which the nurse graduated.

TABLE 15

DISTRIBUTION OF NURSES BY BASIC NURSING PROGRAM*

2 yr. Asso.	3 yr. Dip.	4 yr. B.S.
8	2	1
14	6	3
	7	4
	9	5
	10	13
	11	
	12	
$n_1=2$	$n_2=7$	$n_3=5$

*Table contains identification number of each nurse.

Multiple Regression

A multiple linear regression was performed to determine if it were possible to predict nurses' test scores from the demographic variables. Since the variable "inservice training program" was zero for all nurses, it was eliminated from the multiple regression. Also, the variable "highest degree held" was eliminated since it duplicated the variable "type of basic nursing program." The multiple regression model finally adopted was

$$y = -0.254 + 0.143\ X_1 + .0876\ X_2$$

where y is the predicted nurse's test score,

X_1 is the variable "Basic Nursing Program," and

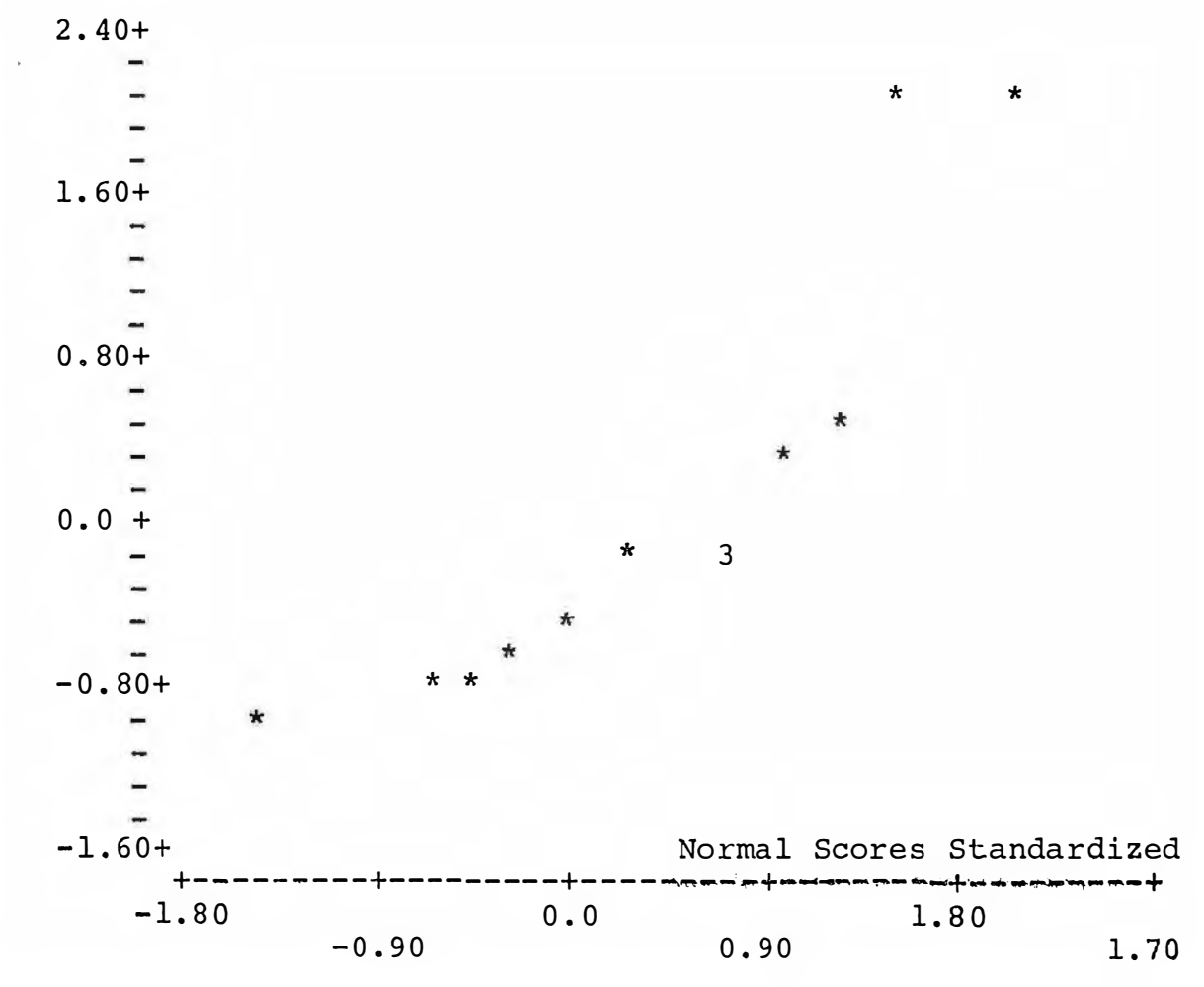
X_2 is the variable "Frequency of Administration of Drug."

The P-value for each of the three coefficients in the regression equation is less than 0.02 with 11 degrees of freedom; i.e., each of the coefficients are significant at the 98% level. This regression yielded a value of $R^2 = 0.784$ adjusted for the degrees of freedom; i.e., this regression model accounts for 78.4% of the variation in the data.

Figure 7 is a plot of the standardized residuals. This normal probability plot is close enough to linearity to allow use of the model given above to predict a nurse's test score.

FIGURE 7

Plot of standardized residuals vs. normal scores of standardized residuals.



Summary

Linear correlation coefficients between nurses' test scores and patient-assessment scores were computed to determine if linear relationships between these variables existed. It was determined that no such relationship was indicated by the data sample.

An analysis of variance (at the α=.01 level) indicated that a nurse's test score was influenced by the type of basic nursing program completed. A Duncan's Multiple Range Test indicated that those nurses receiving a baccalaureate degree in nursing made significantly higher scores than those nurses from either of the other two programs. Other analyses of variance indicated no significant variation in test scores due to either shift assignment or to years of experience.

An analysis of variance of patient-assessment scores by basic nursing program indicated no significant variation due to the different types of nursing programs. A multiple regression of the nurses' test scores on a number of demographic variables indicated an adequate fit of the model. (See page 108.)

In Chapter V, a summary of findings and conclusions were drawn as a result of this study. Implications for nursing and recommendations for further studies are included.

CHAPTER V

SUMMARY, CONCLUSIONS, IMPLICATIONS, AND RECOMMENDATIONS

Introduction

One of the purposes of this study was to determine whether or not there was a relationship between nurses' knowledge in adrenocorticosteroid physiology, side-effects, nursing implications and administration techniques, and the documented nursing assessments which were made on patients receiving an adrenocorticosteroid drug. A summary, conclusions, implications, and recommendations are presented in Chapter V.

Summary

A nonexperimental explanatory research design was used in this study. A causal relationship was being sought between the nurses' knowledge in adrenocorticosteroid physiology, side-effects, nursing implications, and administration techniques, and the documented nursing assessments which were made on patients receiving an adrenocorticosteroid drug.

The setting was an 800 bed county, acute-care, teaching hospital located in the Southwestern United States. Seven general adult, non-intensive care medical-surgical units were sampled.

Data were collected by the utilization of two original tools. One tool was a questionnaire on adrenocorticosteroid therapy which assessed the nurses' level of knowledge in adrenocorticosteroid physiology, side-effects, nursing implications and administration techniques. The second tool utilized was a checklist designed to audit the documented nursing assessments made on patients receiving an adrenocorticosteroid drug.

Linear correlation coefficients between nurses' test scores and patient-assessment scores were computed to determine if linear relationships between these variables existed. It was determined that no such relationship existed.

An analysis of variance (at the $\alpha = .01$ level) indicated that a nurse's test score is influenced by the type of basic nursing program completed by the nurse. A Duncan's Multiple Range Test indicates that those nurses receiving a baccalaureate degree in nursing made significantly higher scores than those nurses from either associate degree or diploma programs. Other analyses of variance indicate no significant variation in test scores due to either shift assignment or to years of experience. An analysis of variance of patient-assessment scores by basic nursing program indicated no significant variation due to the different types of nursing programs.

Conclusions

As a result of this study the following conclusions are made:

1. No statistical relationship between nurses' test scores and patient-assessment scores was isolated in the data
2. Nurses who had baccalaureate degrees made significantly higher scores on the test portion of the questionnaire than nurses who had an Associate Degree or diploma
3. No significant variation was indicated among test scores segregated by shift
4. No significant variation was indicated among test scores segregated by years of experience
5. It was not possible to attribute a significant difference in patient-assessment scores to the different nurses in the different types of basic nursing programs
6. Nurses' test scores can be predicted from the two demographic variables basic nursing program and frequency of administration of adrenocorticosteroids
7. Few assessments of patients receiving an adrenocorticosteroid drug were recorded
8. Nurses possessed knowledge of physiology, however, the application of this knowledge was not demonstrated

Implications

This study has implications for instructors of nursing. The documentation of nursing assessments is of vital importance in that it communicates information regarding the patient's needs, problems, progress, and planned nursing interventions. The importance of documenting nursing assessments must be taught and stressed to all basic nursing students.

Nursing inservice educators have a responsibility to continue to emphasize the importance of documenting nursing assessments. Continuing education for the graduate nurse reinforces past learning experiences and provides an opportunity to build upon her knowledge. Accurate and comprehensive documentation also provides legal protection in the clinical setting.

Implications for nurse researchers are to determine why the Bachelor of Science in Nursing students scored higher on the test portion of the questionnaire as compared to the Associate Degree and diploma nurses. Another possible area for investigation is to determine whether or not nurses are actually assessing patients and just not recording these assessments. Further investigation could be done into why nurses are not charting.

Recommendations

The following recommendations are suggested to improve a replication of this study.

1. Use larger samples including other medical-surgical areas
2. Control the setting in which the nurses completed the questionnaires

The following recommendations are offered as possible studies related to the findings in this study.

1. Measure the staff-patient ratio in relation to the nursing time required by the patients on the unit being sampled and its effect on charting
2. Compare and contrast the overall charting habits of the bachelor of science in nursing, Associate Degree, and diploma nurses
3. Investigate whether or not nurses are actually assessing patients, but not charting these assessments

APPENDIX A

TABLE 16

Side-Effects and Nursing Implications for Patients Receiving Corticosteroid Therapy

Body System/ Side-Effects	Monitor	Nursing Implications: Preventor of Complications	Patient Educator
I. Fluid and Electrolytes			
Sodium retention Fluid retention Congestive heart failure in susceptible patients Potassium loss Hypokalemic alkalosis Hypertension Hypotension or shock-like reaction Calcium and phosphorus loss	Check results of serum sodium Weigh patient daily Record intake and output each shift Observe for edema of feet and/or sacral area Check results of serum potassium Check results of any arterial blood gases Check vital signs each shift	Collaborate with physician and dietician concerning a low sodium, high potassium diet Avoid the use of saline solutions to prepare injectable medications Collaborate with physician to restrict activity in patients who are susceptible to congestive heart failure Collaborate with physician for calcium replacement when indicated	Explain the need for a low sodium high potassium diet Explain what foods are acceptable on his diet Explain the need for restricted activity if ordered Explain the need for calcium replacement if ordered

TABLE 16 (continued)

Body System/ Side-Effects	Monitor	Nursing Implications	
		Preventor of Complications	Patient Educator
	Check results of serum calcium		
II. Musculo-skeletal			
Muscle weakness Steroid myopathy Loss of muscle mass Osteoporosis Vertebral compression fractures Aseptic necrosis of femoral and humeral heads Negative nitrogen balance	Observe for muscle weakness	Report complaints of back, hip or shoulder pain to physician Encourage patient to follow a routine of range of motion exercises Stress the importance of preventing accidents Collaborate with physician and dietician concerning a high protein diet	Explain the importance of following an exercise regime Teach patient some of the more common accident hazards and the ways to avoid them Explain the need for a high protein diet Teach what foods are high in protein

TABLE 16 (continued)

Body System/ Side-Effects	Monitor	Nursing Implications: Preventor of Complications	Nursing Implications: Patient Educator
		Collaborate with physician in obtaining an order for a bed board	
III. Gastrointestinal			
Peptic ulcer with possible perforation and hemorrhage Pancreatitis Abdominal distention Ulcerative esophagitis	Check all stools and vomitus for blood	Give all oral preparations after a meal or with milk Report any dyspepsia to the physician Collaborate with physician for prophylactic antacid Report any complaints of abdominal discomfort to physician	Teach patient to take oral medications with meals or milk only Explain the importance of taking antacid therapy if ordered Teach patient the importance of reporting dyspepsia and tarry stools

TABLE 16 (continued)

Body System/ Side-Effects	Monitor	Nursing Implications: Preventor of Complications	Patient Educator
IV. Dermatologic			
Impaired wound healing	Check the progress of wound healing daily, especially surgical incisions	Advise patient to avoid injury	Explain why it's important to avoid injury
Thin fragile skin		Collaborate with physician for a topical cream to treat acne	If patient experiences perineal burning after IV injection of steroid explain that this is an expected occurance
Petechiae and ecchymoses	Inspect skin condition daily	Give meticulous skin care using a desired skin lotion	Explain that acne and hirutism is a side-effect of the steroid and will disappear when the drug is discontinued
Erythema		Provide emotional support especially for females experiencing hirsutism and acne	
Increased sweating		Use a minimal amount of adhesive tape and always remove it with gentleness	
Burning or tingling especially in perineal area after IV injection			
Hirsutism			
Acne			

TABLE 16 (continued)

Body System/ Side-Effects	Monitor	Nursing Implications: Preventor of Complications	Nursing Implications: Patient Educator
V. Neurological			
Convulsions Increased intracranial pressure with papilledema Vertigo Headache Insomnia Psychoses	Observe patient for impending psychotic episodes, manic or depression states, paranoia, a change in sleep patterns, nervousness and excessive motor activity	Provide environment as is indicated by patient's mood	Teach family that changes in mood are a result of the steroid therapy Teach family how to deal with patient in his altered mental state
VI. Endocrine			
Menstrual irregularity Cushing syndrome Decreased growth (in children)	Check blood sugar reports Do urine sugar and acetone tests each shift	Collaborate with physician and dietician concerning a diet low in saturated fats and carbohydrates	Teach patient why it is necessary to be on a low saturated fat low carbohydrate diet Teach patient what foods are acceptable on his diet

TABLE 16 (continued)

Body System/ Side-Effects	Monitor	Nursing Implications Preventor of Complications	Patient Educator
Adreno-pituitary suppression Decreased carbohydrate tolerance Latent diabetes Increased requirements for insulin Hypothyroidism	Observe patient for polydypsia and polyuria During period of decreasing steroid dose observe patient for signs of adrenal insufficiency, headache, lethargy, weakness, hypotension, diarrhea	Help patient identify stressors especially during time of steroid withdrawal Help patient find ways of coping with stressors Reassure females that menstrual irregularities may be caused by these drugs	If patient is to go home on a steroid drug teach him: A. why it is vital to take his medication as ordered B. not to give his medication to anyone else C. to avoid stressful situations D. If he gets a cold, elevated temperature or is under much psychological stress, he must notify his physician at once to have his dose regulated E. wear a medic-alert bracelet with the name of his physician, the drug he is taking, the dose and instructions for emergency situations

TABLE 16 (continued)

Body System/ Side-Effects	Monitor	Nursing Implications: Preventor of Complications	Nursing Implications: Patient Educator
VII. Opthalmic			
Posterior subcapsular cataracts Increased intraocular pressure Glaucoma Exopthalmos	Collaborate with physician to order regular tonometry readings	Check to see if patient has a history of glaucoma and if he is currently treating it Collaborate with physician to order patient's glaucoma therapy Report visual disturbances or eye pain to physician	If patient has glaucoma, reinforce the need to take his eye drops and to have regular tonometry tests Teach patient to avoid increasing ocular pressure by avoiding tight collars, straining, lifting, emotional upsets and excessive fluid intake
VIII. Other			
Hypersensitivity Thromboembolism	Observe patient for signs of anaphylaxis reaction	Report complaints of a sore throat coryza or a 'sick' feeling to physician	Teach patient to report any 'sick' feelings

TABLE 16 (continued)

Body System/ Side-Effects	Monitor	Nursing Implications: Preventor of Complications	Patient Educator
Mask signs of infection Suppression of immune response Increased appetite due to parenterial therapy Hypo-hyper pigmentation Subcutaneous atrophy Sterile abscess	Check CBC for elevated WBC count Check urinanalysis reports for the presence of bacteria Check for an elevated temperature	Obtain a history of allergies, clotting disorders and medications which were being taken routinely Encourage patient to exercise his legs to prevent blood stasis Collaborate with physician and dietician to increase vitamin C intake Use good handwashing technique Give IM meds deep in gluteal muscle not in the deltoid muscle	Teach patient good handwashing technique Instruct patient to avoid crowds and people with known infections Teach why it is necessary to increase vitamin C intake and what foods are high in vitamin C content

TABLE 16 (continued)

Body System/ Side-Effects	Monitor	Nursing Implications: Preventor of Complicatons	Patient Educator
		Discard unused preparations 48 hours after opening Check IV compatibility if medication is to be mixed with other drugs in a solution Report any calf pain or tenderness to physician	

SOURCES: Barbara Strobele, "How to Counsel Patients on Cortisone," R. N. Magazine 38 (July 1975):57-60.

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Marlene McGann, "Cushing's Syndrome: Its Complexities and Care," R. N. Magazine 38 (August 1975):40-43.

Mary Evans Hamdi, "Nursing Interventions for Patients Receiving Corticosteroid Therapy," in Advanced Concepts in Clinical Nursing, ed. Kay Corman Kintzel (Philadelphia: J. B. Lippincott Company, 1971):241-244.

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Windsor C. Cutting, Handbook of Pharmacology, 5th ed. (New York: Appleton-Century-Crofts, 1972):338.

APPENDIX B

TEXAS WOMAN'S UNIVERSITY
COLLEGE OF NURSING
DENTON, TEXAS

DALLAS CENTER
1810 Inwood Road
Dallas, Texas 75235

HOUSTON CENTER
1130 M.D. Anderson Blvd.
Houston, Texas 77025

AGENCY PERMISSION FOR CONDUCTING STUDY*

THE Parkland Memorial Hospital

GRANTS TO Christine Seftchick Houde, R.N., B.S.N.

a student enrolled in a program of nursing leading to a Master's Degree at Texas Woman's University, the privilege of its facilities in order to study the following problem:

The problem of this study will be to determine whether or not there is a relationship between nurses' knowledge in adrenocorticosteroid physiology, side-effects, nursing implications, and administration techniques, and the documented nursing assessments which are made on patients receiving an adrenocorticosteroid drug.

The conditions mutually agreed upon are as follows:

1. The agency (may) (~~may not~~) be identified in the final report.
2. The names of consultative or administrative personnel in the agency (may) (~~may not~~) be identified in the final report.
3. The agency (wants) (~~does not want~~) a conference with the student when the report is completed.
4. The agency is (willing) (~~unwilling~~) to allow the completed report to be circulated through interlibrary loan.
5. Other: Need title upon of analysis of data

Date July 19, 1976

Signature of Agency Personnel

Christine Houde
Signature of student

Judith N. Jones
Signature of Faculty Advisor

*Fill out and sign three copies to be distributed as follows: Original -- Student; first copy -- agency; second copy -- T.W.U. College of Nursing.

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APPENDIX C

CHART CHECKLIST

DIRECTIONS: Follow previously stated criteria for selecting the patients' charts which are to be audited. Check the chart for the desired information during the twenty-four hour period immediately preceding the time of audit.

I. ADEQUATE MONITORING OF PATIENT'S PHYSIOLOGICAL RESPONSE TO ADRENOCORTICOSTEROID THERAPY

	Instructions	Shift 1 Y	Shift 1 N	Shift 1 NA	Shift 2 Y	Shift 2 N	Shift 2 NA	Shift 3 Y	Shift 3 N	Shift 3 NA
1. Are the vital signs recorded at least once a shift?	Check graphic sheet									
2. Is a total intake and output recorded each shift?	Check intake/ output record sheet									
3. Is a urine sugar and acetone recorded at least once a shift?	Check diabetic record sheet									
4. Is a daily weight recorded?	Check graphic sheet									
5. Has a note been made concerning the patient's skin condition within the past 24 hours?	Check nurses' notes									
6. Has a note been made concerning the patient's mental status within the past 24 hours?	Check nurses' notes									

	Instructions	Shift 1 Y	Shift 1 N	Shift 1 NA	Shift 2 Y	Shift 2 N	Shift 2 NA	Shift 3 Y	Shift 3 N	Shift 3 NA
7. Does patient have a surgical incision or a wound which is currently being treated?	Check on Kardex									
If so, has a note been made concerning its healing progress within the past 24 hours?	Check nurses' notes									
Check Lab Work for Past 24 Hours										
8. Was sodium elevated above normal limits?										
If so, was a note made regarding the abnormal lab data?	Check nurses' notes									
9. Was potassium decreased below normal limits?										
If so, was a note made regarding the abnormal lab data?	Check nurses' notes									
10. Was blood sugar elevated above normal limits?										
If so, was a note made regarding the abnormal lab data?	Check nurses' notes									
11. Was temperature elevated 2 degrees above 98^6 orally over past 24 hrs.	Check graphic sheet									
If so, was a note made of this?	Check nurses' notes									
12 Were the WBCs elevated above normal range in past 24 hours?	Check lab sheets									
If so, was a note made regarding this?	Check nurses' notes									

	Instructions	Shift 1 Y	Shift 1 N	Shift 1 NA	Shift 2 Y	Shift 2 N	Shift 2 NA	Shift 3 Y	Shift 3 N	Shift 3 NA
13. Has the patient gained a ½ pound since the previous day?	Check graphic sheet									
If so, was a note made regarding this?	Check nurses' notes									
II. PROPER ADMINISTRATION OF ADRENOCORTICOSTEROIDS										
1. Does the patient receive an oral adrenocorticosteroid preparation?										
If so, has a note been made that this preparation was administered with food or milk?	Check medication sheet and nurses' notes									
2. Does the patient receive an adrenocorticosteroid preparation intramuscularly?										
If so, has a note been made this preparation was administered IM in gluteal area only?	Check medication sheet and nurses' notes									

PATIENT INTERVIEW CHECKLIST

DIRECTIONS: Interview the patient after explaining the goal of the interview and securing his permission. If the patient responded yes to any of the symptoms, check the nurses' notes to see if a note was made regarding that symptom during the past twenty-four hours.

III. QUESTIONS FOR PATIENT

ASK: In the past twenty-four hours have you had any problems with or noticed:

	Y	N	Shift 1	Shift 2	Shift 3
1. acne					
2. edema or swelling of ankles or lower back					
3. muscle weakness					
4. bone or joint discomfort					
5. easy bruising					
6. increase in thirst					
7. increase in amount of urine excreted					
8. stomach upset or heartburn					
9. dark black tarry stools					
10. change in mental attitude					
11. feel like you are getting a cold or chills					

APPENDIX D

QUESTIONNAIRE ON ADRENOCORTICOSTEROIDS

The purpose of this questionnaire is to determine what the nurses' basic background knowledge is in adrenocorticosteroid physiology, side-effects, nursing implications and administration techniques. The results of this questionnaire will be used concurrently with the results of a checklist to determine whether or not there is a relationship between the nurses' knowledge in adrenocorticosteroid therapy and the documented nursing assessments which are made on patients receiving an adrenocorticosteroid drug.

The questionnaire takes about thirty to forty minutes to complete. Please be as careful as possible in selecting your answers. Take your time! DO NOT put your name on this paper!! Your co-operation with this study is most appreciated. Thank you so much.

PERSONAL INFORMATION

Instructions: Please circle the appropriate answer to each of the following questions.

1. What type of basic nursing program did you graduate from?

   A. 2 year associate degree

   B. 3 year diploma

   C. 4 year baccalaureate degree

   D. other (please specify)

2. What is the highest degree you hold at the present time?

   A. diploma

   B. associate degree

   C. baccalaureate degree

   D. master degree

   E. other (please specify)

3. What is your primary nursing specialty area?

   A. medical

   B. surgical

   C. other (please specify)

4. How many years of experience have you had as an R.N. in your current nursing specialty area?

   A. 0 - 1 year

   B. 1 - 3 years

   C. 3 - 6 years

   D. 6 - 9 years

   E. over 9 years

5. In your present position, do you administer any type of steroid medication?

   A. yes

   B. no

6. If you answered yes to the previous question, about how frequently do you administer these drugs?

   A. rarely

   B. seldom

   C. occasionally

   D. frequently

   E. very often

7. Have you taken a course or an in-service type program on steroid therapy within the past six months?

   A. yes (if yes please specify the course and it's length)

   B. no

8. Have you read any articles pertaining to steroid therapy within the past six months?

   A. yes (if yes please specify the source of the article)

   B. no

9. How would you rate your over-all knowledge of steroid therapy?

   A. poor

   B. fair

   C. average

   D. good

   E. excellent

TEST QUESTIONS

Instructions: The following section is designed to test your knowledge about adrenocorticosteroids. There is only ONE correct answer for each question. Please circle only ONE answer per question.

1. The adrenal cortex secretes which of the following groups of compounds?

   A. catecholamines, androgens, glucocorticoids

   B. glucocorticoids, mineralocorticoids, cholinergics

   C. glucocorticoids, catecholamines, mineralocorticoids

   D. glucocorticoids, androgens, mineralocorticoids

   E. cholinergics, mineralocorticoids, androgens

2. The primary effect(s) of the glucocorticoids is on:

   A. fat, protein and carbohydrate metabolism

   B. protein synthesis enhancement

   C. fluid and electrolyte balance

   D. the release of catecholamines

   E. the control of cholinergic activity

3. ACTH is stimulated to be released when:

   A. cortisol levels are high

   B. cortisol levels are low

   C. corticotropin-releasing factor is low

   D. renin levels are high

   E. renin levels are low

4. Which of the following symptoms would you NOT expect to find in a patient receiving a PURE glucocorticoid drug for a prolonged period of time?

   A. petechiae

   B. loss of muscle mass

   C. cataracts

   D. Hyperkalemia

   E. edema

5. Which of the following groups of symptoms might you observe in a patient receiving a PURE glucocorticoid drug for a prolonged period of time?

   A. decrease in wound healing, menstrual irregularities, hyponatremia

   B. increase in muscle mass, hypernatromia, hyperglycemia

   C. peptic ulcer, pathological fractures of the long bones, glaucoma

   D. headache, hyperkalemia, thin fragile skin

   E. hypokalemia, hypotension, decrease in resistance to infection

6. The renin-angiotensin mechanism stimulates the adrenal cortex to release:

   A. cortisol

   B. angiotensin II

   C. catecholamines

   D. aldosterone

   E. angiotensinogen

7. The release of renin in a normal healthy person is stimulated by:

   A. low renal pressure in afferent arterioles

   B. high renal pressure in afferent arterioles

   C. low cortisol blood levels

   D. aldosterone excess

   E. angiotensin II excess

8. The major action of the mineralocorticoids is which of the following?

   A. regulation of carbohydrate-fat metabolism

   B. regulation of sodium-potassium balance

   C. regulation of catecholamine release

   D. regulation of protein metabolism

   E. regulation of cholinergic release

9. Which of the following groups of symptoms would you expect to find in a patient who has just been started on a PURE mineralocorticoid drug?

   A. hypernatremia, hyperkalemia, oliguria

   B. decreased wound healing, hypotension, diaphoresis

   C. hypoglycemia, diuresis, hypernatremic

   D. oliguria, hypokalemia, hypertension

   E. moon face, hirsutism, dry skin

10. Which of the following groups of symptoms would you expect to find in a patient with adrenal cortex hyperplasia?

   A. hirsutism, hyperkalemia, moon face

   B. acne, hyperglycemia, increase in skin pigmentation

   C. truncal fat distribution, amenorrhea, hypotension

   D. diuresis, hyponatremia, striae

   E. buffalo hump, hypokalemia, acne

11. Which of the following symptoms would you expect to find in a patient with adrenal cortex atrophy?

    A. abdominal cramps, increase in skin pigmentation, hypotension

    B. hypoglycemia, fatigue, hypernatremia

    C. diarrhea, hyperkalemia, decrease in levels of ACTH

    D. weight loss, hypertension, increase in levels of ACTH

    E. increase in levels of aldosterone, vomiting, hyperglycemia

12. If a patient with adrenal cortex atrophy goes untreated he will probably go into an adrenal crisis. Which of the following groups of medications would you expect the physician to order for a patient in a crisis state?

    A. intravenous adrenocorticosteroid, potassium chloride, insulin

    B. sodium bicarbonate, insulin, potassium chloride

    C. kayexalate, intravenous adrenocorticosteroid, .9% sodium chloride IV solution

    D. 50% dextrose solution, kayexalate, ACTH

    E. ACTH, .9% sodium chloride IV solution, sodium bicarbonate

13. Which one of the following statements is TRUE?

    A. Some adrenocorticosteroid preparations may be added to most standard IV solutions to which another drug has already been added.

    B. Some adrenocorticosteroid preparations may be given intravenously over one minute.

    C. When an adrenocorticosteroid compound is incompatible in solution, crystalization and/or clouding of the solution will always be seen.

    D. Adrenocorticosteroid therapy may be discontinued immediately when it has been determined that the patient no longer needs the drug's therapeutic effects.

14. To be better able to monitor a patient on prolonged steroid therapy, the nurse should do:

A. daily intake and output, urine for sugar and acetone, vital signs at least once a shift

B. daily intake and output, daily weight, vital signs at least once a shift

C. daily intake and output, daily weight, daily height, urine for sugar and acetone, vital signs at least once a shift

D. daily weight, urine for sugar and acetone, vital signs at least once a shift

E. daily intake and output, daily weight, urine for sugar and acetone, vital signs at least once a shift

APPENDIX E

ITEM ANALYSIS

ITEM #	A High	A Low	B High	B Low	C High	C Low	D High	D Low	E High	E Low	OTHER High	OTHER Low
1	1	0	0	0	2	5	7 *	5	0	0	0	0
Total	1		0		7		12		0		0	
2	10 *	5	0	0	0	1	0	4	0	0	0	0
Total	15		0		1		4		0		0	
3	0	1	4 *	2	6	3	0	1	0	3	0	0
Total	1		6		9		1		3		0	
4	2	4	1	1	5	3	2 *	1	0	0	A-C-E	
Total	6		2		8		3		0		Marked 3	
5	2	5	3	1	0 *	4	1	0	4	0	0	0
Total	7		4		4		1		4		0	
6	0	0	1	0	0	1	9 *	5	0	4	0	0
Total	0		1		1		14		4		0	
7	8 *	4	0	4	0	1	1	1	1	0	0	0
Total	12		4		1		2		1		0	
8	0	0	9 *	5	1	2	0	2	0	1	0	0
Total	0		14		3		2		1		0	
9	3	5	1	2	0	3	4 *	0	2	0	0	0
Total	8		3		3		4		2		0	
10	3	2	2	2	2	3	0	2	3 *	1	0	0
Total	5		4		5		2		4		0	

ITEMS ANALYSIS (continued)

ITEM #	A High	A Low	B High	B Low	C High	C Low	D High	D Low	E High	E Low	OTHER High	OTHER Low
11	2	* 2	1	3	1	4	4	1	2	0	0	0
Total	4		4		5		5		2		0	
12	4	5	1	0	3	* 2	0	1	2	2	0	0
Total	9		1		5		1		4		0	
13	0	3	6	* 3	2	3	0	2	0	0	A-B-C	
Total	3		9		5		2		0		A-B	
14	0	0	0	2	1	2	0	0	9	* 6		
Total	0		2		3		0		15			

* indicates the correct answer for the given question

	ITEM SUCCESS AND DISCRIMINATION			
ITEM #	# OF HIGHS WHO GOT THE ITEM CORRECT (H)	# OF LOWS WHO GOT THE ITEM CORRECT (L)	H+L	H - L
1.	7	5	12/20 60%	2
2.	10	5	15/20 75%	5
3.	4	2	6/20 30%	2
4.	2	1	3/20 15%	1
5.	0	4	4/20 20%	-4
6.	9	5	14/20 70%	4
7.	8	4	12/20 60%	4
8.	9	5	14/20 70%	4
9.	4	0	4/20 20%	4
10.	3	1	4/20 20%	2
11.	2	2	4/20 20%	0
12.	3	2	5/20 25%	1
13.	7	3	10/20 50%	4
14.	9	6	15/20 75%	3

Desired discrimination is at least 2.
Desired total success should be between 30% and 90%.
Calculated average difficulty .42.

APPENDIX F

TEXAS WOMAN'S UNIVERSITY

(Form B-- Oral presentation to subject)

Consent to Act as a Subject for Research and Investigation:

I have received an oral description of this study, including a fair explanation of the procedures and their purpose, any associated discomforts or risks, and a description of the possible benefits. An offer has been made to me to answer all questions about the study. I understand that my name will not be used in any release of the data and that I am free to withdraw at any time.

Signature _____ Date _____

Witness _____ Date _____

Certification by Person Explaining the Study:

This is to certify that I have fully informed and explained to the above named person a description of the listed elements of informed consent.

Signature _____ Date _____

Position _____

Witness _____ Date _____

APPENDIX G

ORIGINAL DATA

Patient Number	Nurse Number	Shift	Patient-Assessment Score	Test Score	Educational Level
1	1	7-3	5/12 .417	5/14 .357	4 yr BSN
	2	3-11	3/11 .038	1/14 .071	3 yr dip
2	1	7-3	2/8 .250	5/14 .357	4 yr BSN
	2	3-11	2/8 .250	1/14 .071	3 yr dip
3	3	7-3	4/17 .235	10/14 .714	4 yr BSN
4	4	11-7	2/9 .222	8/14 .571	4 yr BSN
5	3	7-3	3/14 .214	10/14 .714	4 yr BSN
6	4	11-7	2/7 .286	8/14 .571	4 yr BSN
7	3	7-3	2/9 .222	10/14 .714	4 yr BSN
	4	11-7	3/10 .300	8/14 .571	4 yr BSN
8	5	3-11	2/12 .167	7/14 .500	4 yr BSN
	6	11-7	2/12 .167	4/14 .286	3 yr dip
9	5	3-11	2/12 .167	7/14 .500	4 yr BSN
	6	11-7	2/12 .167	4/14 .286	3 yr dip
10	5	3-11	1/11 .091	7/14 .500	4 yr BSN
	6	11-7	1/11 .091	4/14 .286	3 yr dip
11	7	7-3	2/11 .182	2/14 .143	3 yr dip
	8	3-11	2/11 .182	2/14 .143	2 yr AD
12	7	7-3	3/7 .429	2/14 .143	3 yr dip
	8	3-11	2/6 .333	2/14 .143	2 yr AD
13	9	3-11	2/10 .200	4/14 .286	3 yr dip
	10	11-7	2/10 .200	4/14 .286	3 yr dip
14	9	3-11	1/7 .143	4/14 .286	3 yr dip
	10	11-7	1/7 .143	4/14 .286	3 yr dip
15	9	3-11	2/7 .286	4/14 .286	3 yr dip
	10	11-7	2/7 .286	4/14 .286	3 yr dip

Patient Number	Nurse Number	Shift	Patient-Assessment Score		Test Score		Educational Level
16	9	3-11	1/13	.077	4/14	.286	3 yr dip
	10	11-7	1/13	.077	4/14	.286	3 yr dip
17	11	7-3	2/6	.333	3/14	.214	3 yr dip
	12	3-11	2/6	.333	4/14	.286	3 yr dip
18	11	7-3	2/8	.250	3/14	.214	3 yr dip
	12	3-11	2/8	.250	4/14	.286	3 yr dip
19	13	7-3	3/12	.250	9/14	.643	4 yr BSN
	14	11-7	2/11	.132	2/14	.143	2 yr AD
20	13	7-3	1/10	.100	9/14	.643	4 yr BSN
	14	11-7	1/10	.100	2/14	.143	2 yr AD

APPENDIX H

SUMMARY OF RESPONSES TO ITEMS ON
CHART CHECKLIST

DIRECTIONS: Follow previously stated criteria for selecting the patients' charts which are to be audited. Check the chart for the desired information during the twenty-four hour period immediately preceding the time of audit.

I. ADEQUATE MONITORING OF PATIENT'S PHYSIOLOGICAL RESPONSE TO ADRENOCORTICOSTEROID THERAPY

	Instructions	Shift 1			Shift 2			Shift 3		
		Y	N	NA	Y	N	NA	Y	N	NA
1. Are the vital signs recorded at least once a shift?	Check graphic sheet	20			20			20		
2. Is a total intake and output recorded each shift?	Check intake/ output record sheet	14	6		15	5		15	5	
3. Is a urine sugar and acetone recorded at least once a shift?	Check diabetic record sheet	1	19		1	19		1	19	
4. Is a daily weight recorded?	Check graphic sheet	5	15		15	5		15	5	
5. Has a note been made concerning the patient's skin condition within the past 24 hours?	Check nurses' notes	4	16		16	4		16	4	
6. Has a note been made concerning the patient's mental status within the past 24 hours?	Check nurses' notes	9	11		2	12	6	3	12	5

	Instructions	Shift 1 Y	Shift 1 N	Shift 1 NA	Shift 2 Y	Shift 2 N	Shift 2 NA	Shift 3 Y	Shift 3 N	Shift 3 NA
7. Does patient have a surgical incision or a wound which is currently being treated?	Check on Kardex	4	16							
If so, has a note been made concerning its healing progress within the past 24 hours?	Check nurses' notes	3	1			1	3		1	3
Check Lab Work for Past 24 Hours			10	10						
8. Was sodium elevated above normal limits?										
If so, was a note made regarding the abnormal lab data?	Check nurses' notes									
9. Was potassium decreased below normal limits?		1	9	10						
If so, was a note made regarding the abnormal lab data?	Check nurses' notes		1			1			1	
10. Was blood sugar elevated above normal limits?		1	9	10						
If so, was a note made regarding the abnormal lab data?	Check nurses' notes		1			1			1	
11. Was temperature elevated 2 degrees above 98^6 orally over past 24 hrs.	Check graphic sheet	4	14	2						
If so, was a note made of this?	Check nurses' notes	1	2	1		2	2	1	2	1
12 Were the WBCs elevated above normal range in past 24 hours?	Check lab sheets	5	7	8						
If so, was a note made regarding this?	Check nurses' notes		5			5			5	

	Instructions	Shift 1 Y	Shift 1 N	Shift 1 NA	Shift 2 Y	Shift 2 N	Shift 2 NA	Shift 3 Y	Shift 3 N	Shift 3 NA
13. Has the patient gained a ½ pound since the previous day?	Check graphic sheet	2	3	15						
If so, was a note made regarding this?	Check nurses' notes		2			2			2	
II. PROPER ADMINISTRATION OF ADRENOCORTICOSTEROIDS										
1. Does the patient receive an oral adrenocorticosteroid preparation?		14	6							
If so, has a note been made that this preparation was administered with food or milk?	Check medication sheet and nurses' notes		14			14			14	
2. Does the patient receive an adrenocorticosteroid preparation intramuscularly?		2	18							
If so, has a note been made this preparation was administered IM in gluteal area only?	Check medication sheet and nurses' notes		2			2			2	

SUMMARY OF RESPONSES TO ITEMS ON
PATIENT INTERVIEW CHECKLIST

DIRECTIONS: Interview the patient after explaining the goal of the interview and securing his permission. If the patient responded yes to any of the symptoms, check the nurses' notes to see if a note was made regarding that symptom during the past twenty-four hours.

III. QUESTIONS FOR PATIENT

ASK: In the past twenty-four hours have you had any problems with or noticed:

	Y	N	Shift 1	Shift 2	Shift 3
1. acne	1		No	No	No
2. edema or swelling of ankles or lower back	3		No	No	No
3. muscle weakness	7		No	No	No
4. bone or joint discomfort	4		No	No	No
5. easy bruising	9		No	No	No
6. increase in thirst	10		No	No	No
7. increase in amount of urine excreted	7		No	No	No
8. stomach upset or heartburn	5		No	No	No
9. dark black tarry stools	2		No	No	No
10. change in mental attitude	4		No	No	No
11. feel like you are getting a cold or chills	7		No	No	No

APPENDIX I

SUMMARY OF PERCENTAGE COMPLIANCE ON THE
CHART CHECKLIST

DIRECTIONS: Follow previously stated criteria for selecting the patients' charts which are to be audited. Check the chart for the desired information during the twenty-four hour period immediately preceding the time of audit.

I. ADEQUATE MONITORING OF PATIENT'S PHYSIOLOGICAL RESPONSE TO ADRENOCORTICOSTEROID THERAPY

	Instructions	Shift 1 Y	Shift 1 N	Shift 1 NA	Shift 2 Y	Shift 2 N	Shift 2 NA	Shift 3 Y	Shift 3 N	Shift 3 NA
1. Are the vital signs recorded at least once a shift?	Check graphic sheet	100%			100%			100%		
2. Is a total intake and output recorded each shift?	Check intake/ output record sheet	70%			75%			75%		
3. Is a urine sugar and acetone recorded at least once a shift?	Check diabetic record sheet	5%			5%			5%		
4. Is a daily weight recorded?	Check graphic sheet	25%			25%			25%		
5. Has a note been made concerning the patient's skin condition within the past 24 hours?	Check nurses' notes	20%			0%			0%		
6. Has a note been made concerning the patient's mental status within the past 24 hours?	Check nurses' notes	45%			14%			20%		

	Instructions	Shift 1 Y	Shift 1 N	Shift 1 NA	Shift 2 Y	Shift 2 N	Shift 2 NA	Shift 3 Y	Shift 3 N	Shift 3 NA
7. Does patient have a surgical incision or a wound which is currently being treated?	Check on Kardex									
If so, has a note been made concerning its healing progress within the past 24 hours?	Check nurses' notes	75%			0%			0%		
Check Lab Work for Past 24 Hours										
8. Was sodium elevated above normal limits?										
If so, was a note made regarding the abnormal lab data?	Check nurses' notes									
9. Was potassium decreased below normal limits?										
If so, was a note made regarding the abnormal lab data?	Check nurses' notes	0%			0%			0%		
10. Was blood sugar elevated above normal limits?										
If so, was a note made regarding the abnormal lab data?	Check nurses' notes	0%			0%			0%		
11. Was temperature elevated 2 degrees above 98^6 orally over past 24 hrs.	Check graphic sheet									
If so, was a note made of this?	Check nurses' notes	33%						33%		
12 Were the WBCs elevated above normal range in past 24 hours?	Check lab sheets									
If so, was a note made regarding this?	Check nurses' notes	0%			0%			0%		

	Instructions	Shift 1 Y	Shift 1 N	Shift 1 NA	Shift 2 Y	Shift 2 N	Shift 2 NA	Shift 3 Y	Shift 3 N	Shift 3 NA
13. Has the patient gained a ½ pound since the previous day?	Check graphic sheet									
If so, was a note made regarding this?	Check nurses' notes	0%			0%			0%		
II. PROPER ADMINISTRATION OF ADRENOCORTICOSTEROIDS										
1. Does the patient receive an oral adrenocorticosteroid preparation?										
If so, has a note been made that this preparation was administered with food or milk?	Check medication sheet and nurses' notes	0%			0%			0%		
2. Does the patient receive an adrenocorticosteroid preparation intramuscularly?										
If so, has a note been made this preparation was administered IM in gluteal area only?	Check medication sheet and nurses' notes	0%			0%			0%		

SUMMARY OF PERCENTAGE RECOGNITION OF INCIDENCE OF ITEMS ON PATIENT INTERVIEW CHECKLIST

DIRECTIONS: Interview the patient after explaining the goal of the interview and securing his permission. If the patient responded yes to any of the symptoms, check the nurses' notes to see if a note was made regarding that symptom during the past twenty-four hours.

III. QUESTIONS FOR PATIENT

ASK: In the past twenty-four hours have you had any problems with or noticed:

	Y	N	Shift 1	Shift 2	Shift 3
1. acne			0%	0%	0%
2. edema or swelling of ankles or lower back			0%	0%	0%
3. muscle weakness			0%	0%	0%
4. bone or joint discomfort			0%	0%	0%
5. easy bruising			0%	0%	0%
6. increase in thirst			0%	0%	0%
7. increase in amount of urine excreted			0%	0%	0%
8. stomach upset or heartburn			0%	0%	0%
9. dark black tarry stools			0%	0%	0%
10. change in mental attitude			0%	0%	0%
11. feel like you are getting a cold or chills			0%	0%	0%

APPENDIX J

SS# 069-44-4425

TEXAS WOMAN'S UNIVERSITY

DENTON, TEXAS 76204

THE GRADUATE SCHOOL
P.O. BOX 22479, TWU STATION

August 10, 1976

Mrs. Christine Seftchick Houde
181 West 62nd Street
Hialeah, Florida 33012

Dear Mrs. Houde:

I have received and approved the Prospectus for your research project.

Best wishes to you in the research and writing of your project.

Sincerely,

Phyllis Bridges

Phyllis Bridges
Acting Dean

PB:le

cc: Mrs. Judith N. Jones
Dr. Peggy Chinn

APPENDIX K

TEXAS WOMAN'S UNIVERSITY
DALLAS, TEXAS 75235

COLLEGE OF NURSING

July 7, 1976

Christine Seftchick Houde
1005 Metker #46
Irving, Texas 75062

Dear Ms. Houde:

I have read and approved your proposal, "Adrenocorticosteroid Therapy and Documented Nursing Assessment: A Comparison". At this time, I am serving as the only active member of the Dallas Committee. As other members are added, they will read your prospectus and sign the Human Research Review Committee Report. Be prepared to answer any of their questions which may arise regarding your proposal. In the meantime, continue with your plans to collect data.

Thank you.

Sincerely,

Geri Goosen

Geri Goosen, R.N., M.S.
Assistant Professor and Co-ordinator
of Graduate Medical-Surgical Faculty

GG:rw

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RESEARCH AND INVESTIGATION INVOLVING HUMANS

Statement by Program Director and Approved by Department Chairman

This abbreviated form is designed for describing proposed programs in which the investigators consider there will be justifiable minimal risk to human participants. If any member of the Human Research Review Committee should require additional information, the investigator will be so notified.

Copies of this Statement and a specimen Statement of Informed consent should be submitted at the earliest possible time before the planned starting date to the chairman or vice chairmen listed below:

Denton Campus (Submit five copies)

Mr. George Vose, Chairman
Dr. Calvin Janssen, Vice-Chairman
Dr. Marjorie Keele
Dr. Aileene Lockhart
Dr. Carolyn Rozier

Houston Campus (Submit five copies)

Dr. Helen Ptak, Vice-Chairman
Dr. Laura Smith
Mrs. Patricia Smith
Mrs. Irene Robertson

Dallas Campus (Submit one copy ~~five copies~~)

Dr. Opal White, Vice-Chairman
Mrs. Geraldine Logue
Mrs. Patricia Pardies

Title of Study: ADRENOCORTICOSTEROID THERAPY AND DOCUMENTED NURSING ASSESSMENT: A COMPARISON

Program Director(s): Judith Jones, R.N., M.S.

Graduate Student: Christine Seftchick Houde, R.N., B.S.N.

Estimated beginning date of study: 7/10/76 **Estimated duration:** 4 weeks

1. Brief description of the study (use additional pages or attachments, if desired, and include the approximate number and ages of participants, and where they will be obtained).

See attached paper for desired information.

A copy of the proposal is also attached for any further information which may be needed.

A copy of the patient interview questions may be found in Appendix A of the proposal.

2. What are the potential risks to the human subjects involved in this research or investigation? "Risk" includes the possibility of public embarrassment and improper release of data. Even seemingly nonsignificant risks should be stated and the the protective procedures described in (3) below.

The only possible risk to the patients in this study would be that their privacy may be invaded, or public embarrassment may result.

3. Outline the steps to be taken to protect the rights and welfare of the individuals involved:

1. Patients will be given a verbal explaination of why they are being asked these questions
2. All the patients in the study will be assigned a number NO NAMES WILL BE USED!!

4. Outline the method for obtaining informed consent from the subjects or from the person legally responsible for the subjects. Attach documents, i.e., a specimen informed consent form. These may be properly executed through completion of either (a) the written description form, or (b) the oral description form which are available from the committee chairmen or may be reproduced from the attached specimen copies. Other forms which provide the same information will be acceptable.

Form B will be used to obtain consent (See attached Copy)

See attached paper for method of obtaining informed consent.

1. The primary purpose of this study is to determine whether or not there is a relationship between nurses' knowledge in adrenocorticosteroid physiology, side-effects, nursing implications and administration techniques, and the documented nursing assessments which are made on patients receiving an adrenocorticosteroid drug. This will be accomplished by using two original tools. One tool is a questionnaire on adrenocorticosteroid therapy which has a test portion which will be given to the nurses. The second tool that will be used is a two part check list. The first part of the check list was developed to audit charts for the documented nursing assessments which are made on patients receiving an adrenocorticosteroid drug. The second part of the check list was designed to interview a patient receiving an adrenocorticosteroid drug. The interview involves asking the patient if he has had any of eleven symptoms.

   Patients will be chosen from two medical-surgical units according to the following criteria:

   1. Patients who are receiving a form of an adrenocorticosteroid for at least the past twenty-four hours, but no longer than three weeks on a schedule of at least twice a day
   2. Patients who are eighteen years of age or older

3. Patients who are administered an adrenocorticosteroid drug by the oral, intramuscular or intravenous routes
4. Patients who are mentally and physically capable of answering questions

Ten patients in each unit will be interviewed.

4. The method for obtaining informed consent will be:
   1. Have a witness present
   2. The patient will then be given the following information- " My name is Christine Houde. I am a registered nurse who is currently working on a master's degree. A research project must be done as part of the requirements for a master's degree. My research project involves checking to see if the necessary information is being recorded on your chart in regard to your response to the steroid drug which you are currently taking. In order to do this, I would like to ask you eleven questions. I must tell you now that your name will not appear in the study in any way as you will be given a number by which to be identified from here on. Do you have any questions? (Answers to questions will be given here) Before I can ask you any of my questions it is necessary for you to sign a release form giving me permission to in fact ask you any questions. (Give patient consent form B to sign)

5. If the proposed study includes the administration of personality tests, inventories, or questionaires, indicate how the subjects are given the opportunity to express their willingness to participate. If the subjects are less than the age of legal consent, or mentally incapacitated, indicate how consent of parents, guardians, or other qualified representatives will be obtained:

Once the patient has been given the introduction explaining what the study is about, he will be told that he does not have to participate in answering the questions if he chooses not to. He will be instructed not to sign the release form if he does not wish to participate in the study.

(Signed) _____ _____
Program Director Date

(Signed) (Christine) Houde, R.N., B.S.N. July 4, 1976
Graduate Student Date

(Signed) _____ _____
Dean, Department Head, or Director Date

Date received by committee chairman: _____

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