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Adrenal Function in Surgical Patients

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I. ADRENAL FUNCTION IN SURGICAL PATIENTS

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Introduction

Recent dramatic developments have brought the adrenal glands to the notice of both doctors and laymen. Much of this information has been based on observation concerning the functions of the adrenals during severe stress. It has been 14 years since Selye's postulation of the "adaptation syndrome", and a great deal of the recent work has confirmed his theory. Although much remains to be learned about adrenal function during stress, it seems logical to apply some of the facts that have been learned to the understanding of the physiological adaptations in post-operative surgical patients.

The present discussion will not be a report of a completed investigation, but merely a progress report on studies underway together with the author's highly tentative ideas concerning the significance of adrenal activity in patients during and following surgical procedures. Although organic and inorganic metabolism are equally worthy of consideration in this regard, limitations of time make it desirable to limit the discussion to mineral and water metabolism.

In the consideration of any physiologic derangement, it is of great importance to distinguish between the manifestations of the primary abnormal process and those of compensatory or defensive processes. This is the type of distinction which must be made in consideration of the metabolic responses to trauma or surgery. Rational treatment must obviously be based on this. Perhaps the most significant data on the importance of adrenal activity as a defensive mechanism in surgical patients are the results of operations on patients with adrenal insufficiency.

Tolerance of the Adrenal-Deficient Patient to Surgery

From the reported outcome of surgery on patients with unsuspected or untreated Addison's, it is apparent that adrenal function is necessary for the survival of even minimal surgical procedures.[1,2,3,4,5] Such patients have died from exploratory laparotomy, appendectomy or mere induction of anesthesia. This situation was brought to our attention this year when a 76-year old robust man was admitted to this hospital with abdominal pain. Since an acute surgical situation was suspected, exploration was carried out and a chronically inflamed gallbladder removed. He went into profound collapse after operation and was dead within 36 hours. Autopsy showed bilateral adrenal tuberculosis though no symptoms of Addison's or any illness had existed until a few days before his admission. It was apparent that, although the man had been able to survive under ordinary circumstances, the additional stress of a relatively small surgical operation had caused his death.

We must consider now the mechanisms by which the adrenals permit survival of surgical trauma in the normal individual.

The "Adaptation Syndrome" of Selye

The series of events which follow subjection of the organism to unusual stress have been elaborated under the heading of the "General Adaptation Syndrome."[6] This concept, initiated by Selye, has now become quite familiar and cannot be discussed in detail here. Of importance to the present consideration, however, Selye's description of a "shock phase" immediately following exposure to trauma which is followed by "counter-shock" and a "resistance" phase. The shock phase describes the direct manifestations of trauma to the animal. The counter shock phase represents the reactions on the part of the organism which render it able to resist or compensate for the deleterious effects of the shock phase. The phenomena of counter-shock and resistance are mediated by the pituitary and the adrenal glands.

Some of the changes which occur during these stages of adaptation are of importance in our later consideration of the

surgical patient. Blood chloride levels, for example, fall sharply during the shock stage and rise during the stage of resistance.[7] Sodium behaves similarly. Also during the shock phase there is a tendency to development of edema, inhibition of diuresis and alterations in capillary permeability.[8,9] Since these effects of trauma are much more severe in the adrenalectomized animal and can be favorably influenced by administration of adrenal substances[8], they must be considered primary effects of trauma rather than manifestations of resistance. Although the blood levels of sodium and chloride fall immediately, there develops a strong tendency to sodium retention. Whereas the former is apparently a primary effect of trauma, that latter may well be a compensatory process and mediated by the adrenal.

The cellular changes which follow exposure to trauma are also of importance. Lymphopenia and eosinopenia develop rapidly after exposure and are sensitive indices of the production of one type of adrenal hormones. The eosinophil count has become a useful basis for the estimation of pituitary and adrenal activity and reserve.[10,11]

Further manifestations of adrenal activity in the response to trauma are increased excretion of nitrogen, increased blood sugar and anatomical changes such as involution of the thymus and increased adrenal size. The excretion of cortin-like substances in the urine under the circumstances of stress has also been studied extensively.[8,12]

Adrenal Hormones and Pituitary-Adrenal Relationship

Recent developments in the field of pituitary-adrenal physiology have increased our understanding of the nature of the response to trauma. The availability of pure corticotrophic hormone has lent great impetus to this advance.[13,14] Adrenocorticotrophic hormone is released from the pituitary under the circumstances of stress causing the liberation of adrenal substances which mediate the compensatory reactions outlined above. The exact series of events leading to the release of ACTH is not wholly clear, but Sayers has shown in animals that the output of ACTH bears an inverse relationship to the level of circulating adrenal steroids.[15] According to this concept, stress, perhaps through the intermediate action of epinephrine[16], causes an increased consumption of steroids by the tissues and the resulting reduction of their blood levels stimulates the output of ACTH.

With Dr. C. F. Williams we have adduced evidence that such a mechanism exists in the human being. Desoxycorticosterone has been found to diminish the eosinopenic response to epinephrine and histamine when given 3-4 hours before the administration of these "stress agents". (Figure I and II) DOCA did not, however, influence the response to ACTH (Figure III). It was concluded that DOCA, which by itself has no effect on the circulating eosinophils, partially inhibits the release of ACTH by raising the blood level of adrenal steroids.

Three general types of hormones are believed to be produced by the adrenal cortices.

1. 11-desoxy substances (e.g., desoxycorticosterone) the primary function of which is the regulation of salt and water balance.

2. 11-oxy substances with or without 17-hydroxyl groups (e.g., corticosterone and Kendall's compounds E & F.) Although these substances are capable of influencing mineral balance in various ways, they are primarily concerned with nitrogen and carbohydrate metabolism. They are also responsible for the changes in lymphoid tissue and the eosinophil depression following stress.

3. Androgenic substances (e.g., adrenosterone) and possibly estrogens influence nitrogen balance positively.

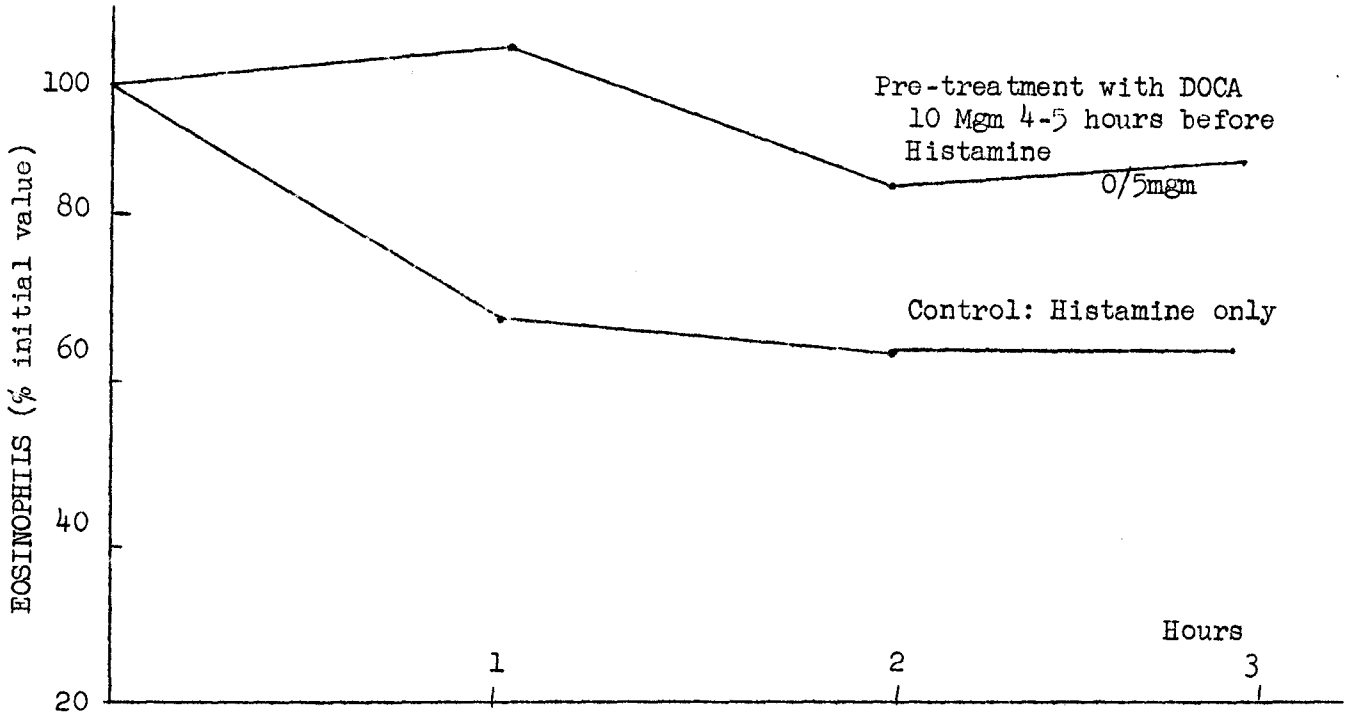

Fig. I. Effect of DOCA on eosinopenic response to Histamine: mean values in 8 subjects, each done with and without pre-treatment with DOCA.

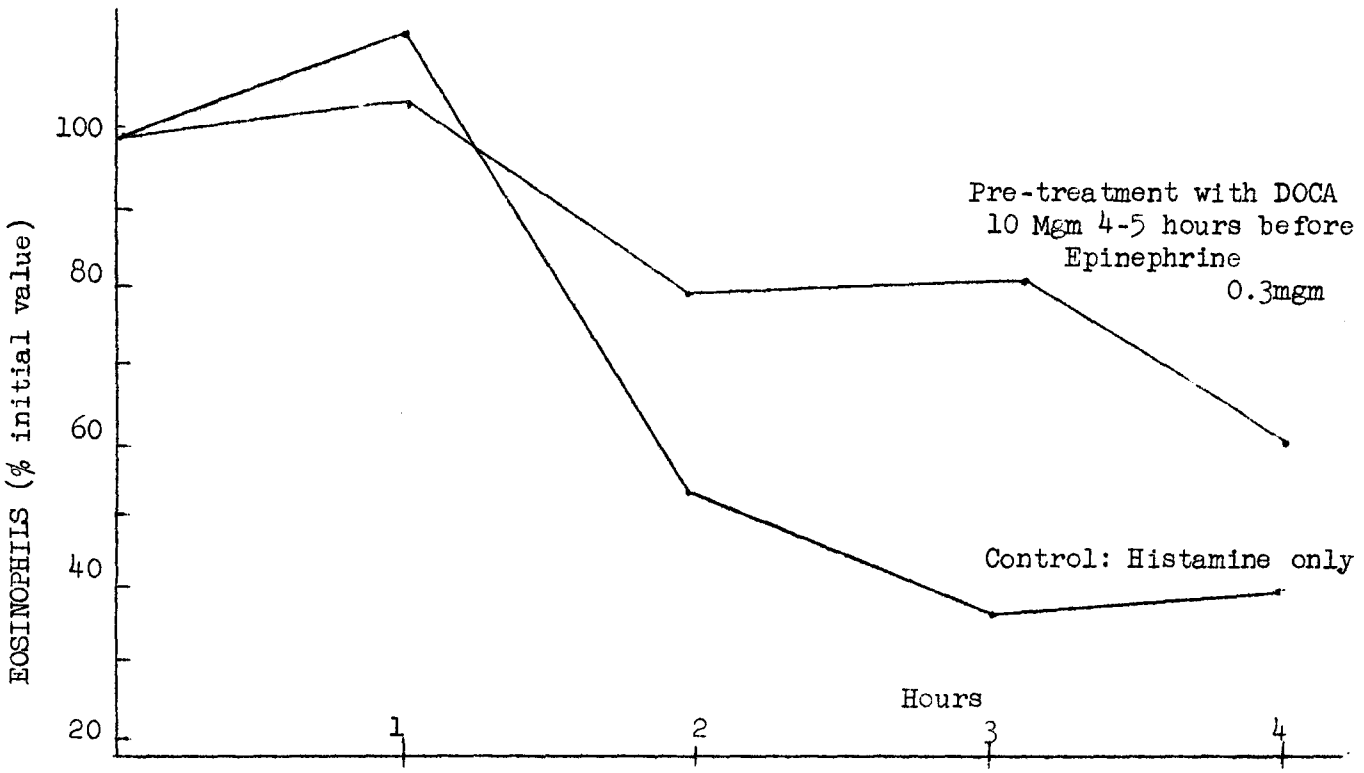

Fig. II. Effect of DOCA on Eosinopenic response to Epinephrine: mean values in 11 subjects, each done with and without pre-treatment with DOCA.

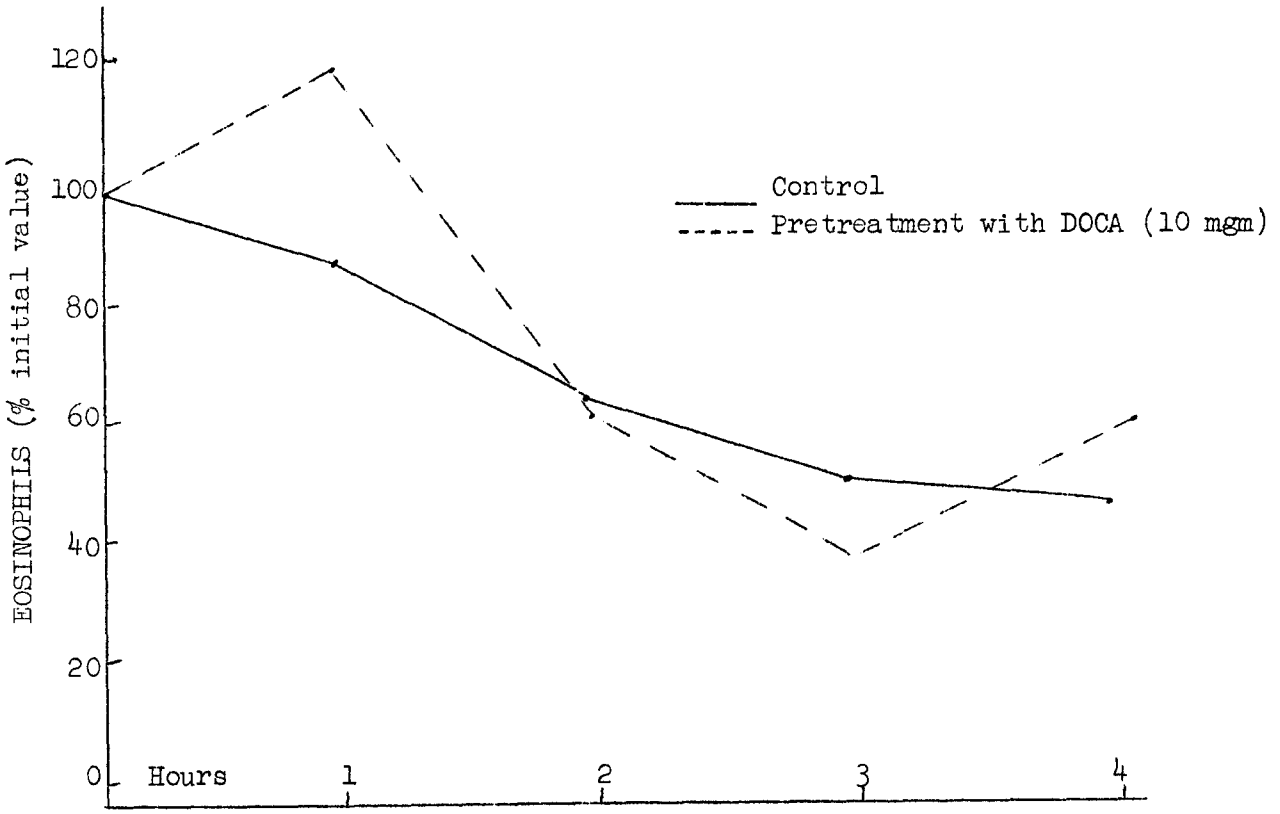

Fig. III. Effect of DOCA on response of eosinophils to ACTH. Mean values in four subjects, each done with and without pretreatment with DOCA.

The question of the control of these hormones is important. Greep and his co-workers have produced convincing evidence that, in the rat, only the 11-oxy group are under the control of the pituitary. These are believed to be produced by the zona fasciculata which undergoes hypertrophy under pituitary corticotrophic stimulation.[17] The desocy or "salt-retaining" hormones, on the other hand, are considered to be formed by the zona glomerulosa and are independent of the pituitary, being responsible only to the Na/K ratio of the serum.[18,19]

This independence has been questioned in the human being because in that species, unlike the rat the administration of ACTH causes sodium retention.[17,20] Further evidence for this view has been produced by Conn and his co-workers in experiments in which it was found that the administration of ACTH in human subjects caused changes in the composition of thermal sweat characteristic of those associated with the "salt-retaining" adrenal hormones.[21] They have found similar changes surgical operations.[22] Leaf and co-workers also felt that the two types of hormones were not independently controlled because limitation of sodium intake in their subjects resulted in increased excretion of urea, uric acid and nitrogen.[23]

Experiments recently carried out here have lead the author to feel that desoxy and 11-oxy corticoids may be separately controlled in the human as they apparently are in the rat. Table I shows eosinophil values on 8 subjects who were subjected to a very low sodium (10-13 M. E. per day) high potassium (250-300 M. E. per day) intake. It will be seen that there was no depression of eosinophils during the experimental period. Figure IV shows urinary 24 hour corticoids (formaldehyde-producing) determined by the method of Mason[24] on three such subjects. There was no increase, and in at least two of the subjects there was a decrease in the corticoid output. If the pituitary were required to participate in the reaction to the "electrolyte stress"

produced in this manner we should expect a decreased eosinophil level and an increased excretion of oxy corticoids. The conclusion is suggested that desoxy-substances which we know are not excreted in a form measurable by this method are mobilized without the participation of adrenocorticcliophic hormone.

Table I

Circulating eosinophils in eight subjects on low sodium, high potassium intake. Underlined values indicate control period, i.e., after at least 24 hours of normal intake.

Subject	0	1	2	3	4	5	6 Days
1-	31	37	80	43			
2-	142	111	136	160			
3-	450	516	462	462			
4-	277	320	302	296			
5-	94	100	94	96	63		
6-	233	273	251	235	226	233	218
7-	280	445	515	223	280	400	255
8-	223	325	275	279	190	250	

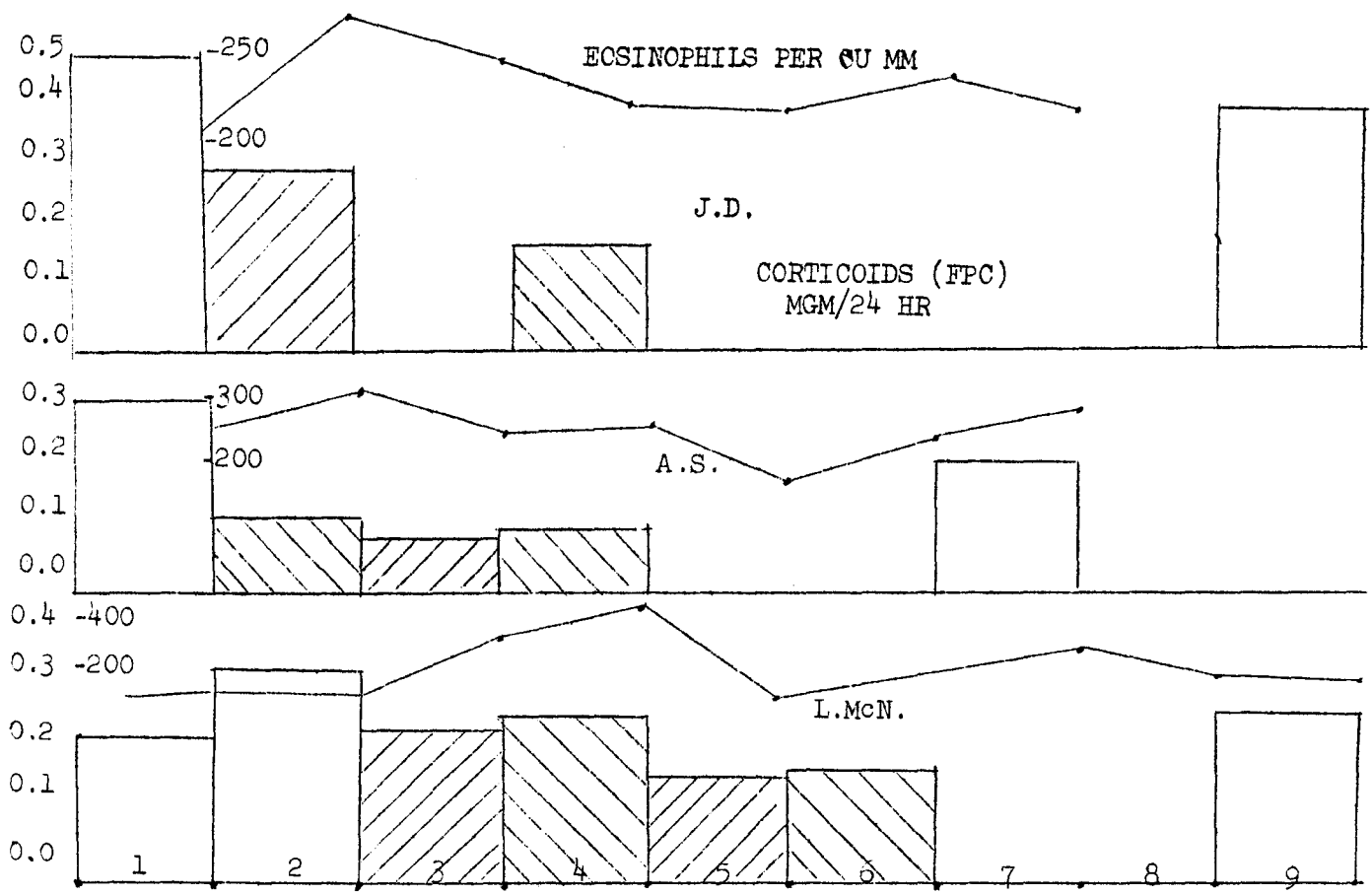

Fig. IV. Eosinophils and 24-hour corticoid excretion in three subjects during high potassium, low sodium intake. Cross-hatched areas: experimental period; open areas: control.

The Post-Operative Patient

It is likely that the changes which have been described as systemic manifestations of non-specific trauma are all of importance in the consideration of the patient following surgery. The changes in nitrogen metabolism in post-operative patients are familiar and cannot be discussed here. The strong tendency of post-operative patients to retain sodium and to excrete large amounts of potassium have been pointed out by Coller and form the basis for the conservative regime we now employ for the administration of salt to such patients.[24,25,26]

Drs. Ariel and Kremen presented before this group in January their observations on the retention of chloride in the extracellular space in post-operative patients.[27] They also discussed the oliguria and failure of diuresis which is seen in varying degrees in patients after operations. Lyon et al have studied the increase in the extracellular space which follows surgery and have observed decreases in serum chloride concentrations.[28]

Dr. Ariel also studied a series of patients who developed convulsions post-operatively in association with low serum and special fluid sodium and chloride levels. He felt these convulsions were on the basis of "water intoxication", and was able to correlate the phenomenon with the retention of a large amount of fluid administered without salt.

The recently-developed methods for evaluating adrenal activity are proving to be of great interest in the study of the post-operative surgical patient. Venning found that post-operative patients excrete from 3 to 30 times the normal daily amount of glycogenic corticoids in the urine. The use of the eosinophil count to follow the adrenal response to surgery, first described by Laragh and Almy[31], has been studied extensively by Roche et al.[32] Roche[32] and Moore[33] have suggested that the eosinopenic response to epinephrine or ACTH could be used as a method for pre-operative evaluation of a patient's ability to respond to surgery.

We have been interested in following the eosinophil levels after a variety of surgical operations in patients on whom serum electrolyte determinations were made simultaneously. Figure V shows the average values on 14 such patients after uncomplicated major surgical procedures. It will be observed that the eosinophils drop promptly to very low levels and do not reach the pre-operative level before the 4th to 6th post-operative day. The duration of eosinopenia is somewhat longer in our cases than in those reported by others.[22,32] Although our data are not adequate to establish this, there seems to be no definite relationship between the magnitude of the operation and the duration of eosinopenia. Chlorides and sodium regularly fall in the post-operative period. In most instances, this is of no serious consequences, but occasionally it may be severe. Lowered levels of serum potassium were quite regularly seen in the post-operative period. This has been reported upon by Randall et al[34] as well as by Snyder and Snyder[35] and gives credence to the idea that the increased excretion of potassium post-operatively is not due merely to tissue destruction. Alkalosis of varying degree and duration also observed during the post-operative period and some elevation of the CO_2 combining power almost always occurred.

In most instances, the period of relative hypochloremia and hyponatremia coincided with the period of eosinophil depression. We feel this represents the period in which unusual stress exists and does not imply any causal relationship between adrenal hyperactivity and hypochloremia. In some cases discrepancies appeared between the electrolyte changes and the eosinophil response. In one man (F.C.) who had undergone an extremely extensive operation for advanced cancer, the eosinophils had returned to normal on the second day. At this time, the chlorides had dropped to 73 ME per L and the sodium to 118 ME per L. Thereupon the patient displayed a convulsion

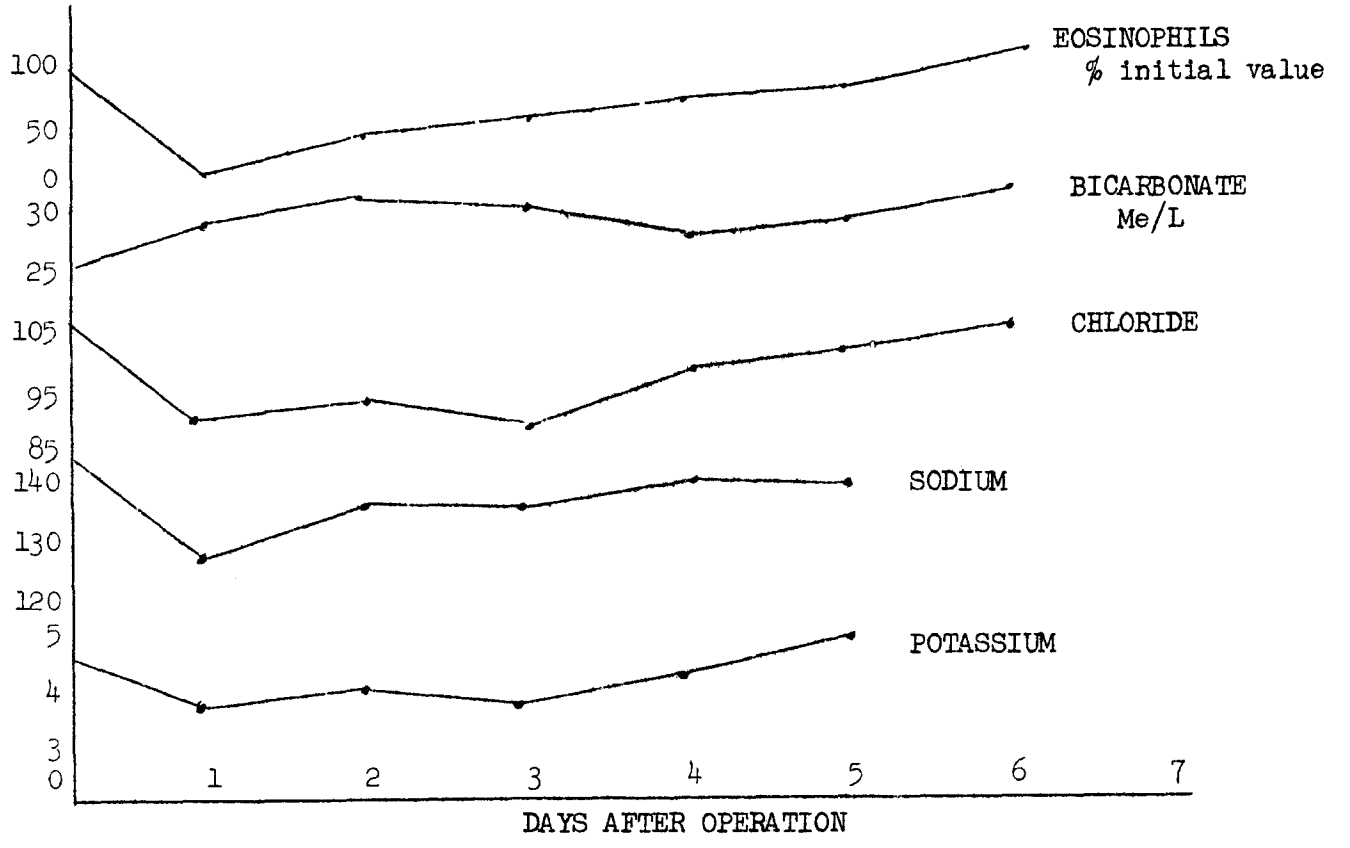

Fig. V. Response of Eosinophils and Electrolyte Concentrations in 14 Patients following uncomplicated major surgical procedures. (Sodium and Potassium studied in 6).

and became comatose. During the next three days, however, the eosinophils fell again and the electrolytes gradually returned to normal. It was our impression in this case that some sort of relative and transitory adrenal failure had developed at the time when the eosinophils rose prematurely and the electrolyte concentrations fell to very low levels.

Discussion

A tentative scheme to explain the observed electrolyte changes in the post-operative patient can now be suggested.

In the immediate post-operative period, as a direct result of trauma, a series of changes occur which are analogous to the "shock phase" of Selye's alarm reaction. There is increased capillary permeability and oliguria with associated retention of fluid in the extracellular space as described by Lyon, and probably expansion of the interstitial space. Retention of water results in dilution of the blood electrolytes and a tendency to lowered sodium and chloride levels. The mechanisms responsible for this phase of the response to trauma are not known. This appears to be a stage of <u>relative</u> adrenal insufficiency.

Through a series of steps which have been outlined above, the adrenal is stimulated to secrete a tremendous excess of steroid hormones which have the ability to counteract these unfavorable phenomena. We know from the work of Gaunt and Eversole and many others that adrenal substances are able to promote diuresis[36] and that they are able to restore normal capillary permeability.[37, 38, 39] In addition, their salt-retaining effect prevents the dilution of extracellular sodium and chloride from becoming too severe. In this sense, post-operative salt retention is an important compensatory mechanism and not merely a hazard.

It seems reasonable that a disproportion sometimes occurs between the degree of the immediate and unfavorable results of trauma and the extent of the compensatory adjustments. Such would appear to be the case in those cases which Dr. Ariel brought to our attention as instances of "water intoxication."

Dr. McQuarrie and his department have demonstrated the relationship of low extracellular sodium levels to the convulsive threshold and the ability of adrenal preparations to increase the latter. Swingle[43] and Gaunt[44] have demonstrated their effectiveness in preventing experimental water intoxication in animals.

Finally it is suggested that these responses may be mediated by hormones of the 11-oxy type. They are capable of all these types of activity including salt retention. Although evidence in animals points out that 11-oxy, 17-hydroxy substances cause sodiumphoresis[45,46,47], recent studies in the human have shown that they can cause sodium retention.[48,49] Furthermore Woodbury et al have recently offered evidence to suggest that these substances are able to influence sodium balance either positively or negatively depending on the need for one or the other type of activity.[50]

This scheme is presented because facts are hard to remember without theories to hold them together. Future developments in this rapidly progressing field will undoubtedly require radical changes in our present ideas about the factors at work in the regulation of electrolytes in the surgical patient.

Summary

Clinical experience has demonstrated the necessity for adrenal function for the toleration of even minor surgical procedures. The processes by which the pituitary-adrenal mechanism protects the body against the detrimental effects of unusual stress have been described in terms of the "General Adaptation Syndrome." Evidence has been brought forth to indicate that post-operative alterations in electrolyte and water metabolism are manifestations of the "shock phase" and the "counter-shock phase" of this syndrome. Our experiments so far indicate that the human behaves very similarly to animals studied under the circumstances of stress with regard to the regulation of pituitary adrenocorticotrophic activity, the control of the individual groups of adrenal substances and the changes in water and electrolyte balance following exposure to trauma.

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II. MEDICAL SCHOOL NEWS

Coming Events

May 22-27 Continuation Course in Proctology for General Physicians.

May 25 Special Lecture, "Review of Chemical and Pharmacological Investigations on Cardiac Glycosides", Dr. Arthur Stoll, Professor of Chemistry, University of Basle, Switzerland, 4:00 p.m., Medical Science Amphitheater.

June 12 Minnesota Medical Alumni Association and Minnesota Medical Foundation Dinner - Spaulding Hotel, Duluth, Minnesota, during annual meeting of Minnesota State Medical Association.

* * *

Langsjoen Named Outstanding Senior

Per Harald Langsjoen, senior medical student, was named as the outstanding member of the medical school class of 1950 and will receive the annual award made by the Southern Minnesota Medical Association. Langsjoen was the unanimous choice of the committee in charge of making the selection because of his scholastic record and such personal qualifications as leadership, general interests, and ability.

Langsjoen, who is a resident of St. Peter, Minnesota, completed his pre-medical work at Gustavus Adolphus College where he participated in many extra-curricular activities. His career as a medical student has been outstanding as evidenced by the fact that he was number one man in his class with an honor point ratio of 2.8.

After graduation, he will move to Letterman General Hospital in San Francisco, where he will begin his internship. The Southern Minnesota Medical Association award will be presented to Langsjoen at the Annual Cap and Gown Day Convocation which will be held on Thursday, May 18.

* * *

Faculty News

Doctors J. T. Syverton, T. R. Hamilton, and W. J. Cromartie attended the meeting of the American Association of Pathologists and Bacteriologists at the University of Wisconsin, April 13-15. Dr. Hamilton presented a paper with Dr. Syverton as co-author.

Doctors J. T. Syverton, T. R. Hamilton, W. J. Cromartie, A. A. Werder, and D. T. Imagawa attended the meetings of the American Association for Cancer Research, American Association for Immunologists, and American Society for Experimental Pathologists in Atlantic City at which Doctors Werder, Imagawa, Hamilton, and Syverton gave papers.

Dr. John Adams, Associate Professor of Pediatrics, has announced that he has accepted an appointment as Professor and Head of the Department of Pediatrics at the University of California Medical School, Los Angeles. Dr. Adams will spend a portion of the summer in medical travels in Europe and will take over his new post in California in September. Dr. Adams' many friends wish to join in extending congratulations on this well deserved recognition.

III. UNIVERSITY OF MINNESOTA MEDICAL SCHOOL
CALENDAR OF EVENTS

May 14 - May 20, 1950

No. 288

Sunday, May 14

9:00 - 10:00 Surgery Grand Rounds; Station 22, U. H.

10:30 - 11:00 Surgical Conference; Cardiac Surgery; Richard L. Varco; Rm. M-109, U.H.

Monday, May 15

9:00 - 9:50 Roentgenology-Medicine Conference; L. G. Rigler, C. J. Watson and Staff; Todd Amphitheater, U. H.

9:00 - 10:50 Obstetrics and Gynecology Conference; J. L. McKelvey and Staff; M-109, U. H.

10:00 - 12:00 Neurology Rounds; A. B. Baker and Staff; Station 50, U. H.

11:00 - Pediatric Rounds; Erling Platou; Sta. I, Minneapolis General Hospital.

11:00 - 11:50 Physical Medicine Seminar; E-101, U. H.

11:00 - 11:50 Roentgenology-Medicine Conference; Veterans Hospital.

11:00 - 12:00 Cancer Clinic; K. Stenstrom and A. Kremen; Eustis Amphitheater, U. H.

12:00 - 1:00 Physiology Seminar; Transcapillary Exchange of Materials; John A. Johnson; 214 M H.

12:15 - 1:20 Obstetrics and Gynecology Journal Club; Staff Dining Room, U. H.

12:30 - 1:20 Pathology Seminar; Sickle Cell Disease; John Rukavina; 104 I. A.

12:30 - 1:30 Surgery Problem Case Conference; A. A. Zierold, C. Dennis and Staff; Small Classroom, Minneapolis General Hospital.

1:30 - 2:30 Surgery Grand Rounds; A. A. Zierold, C. Dennis and Staff; Minneapolis General Hospital.

1:30 - 2:30 Pediatric-Neurological Rounds; R. Jensen, A. B. Baker and Staff; U. H.

4:00 - Pediatric Seminar; Report on American Pediatric Society meetings; I. McQuarrie, et al; 6th Floor West, Child Psychiatry, U. H.

4:00 - Public Health Seminar; 113 Medical Sciences.

4:00 - Medical-Surgical Conference; Bldg. I, Main Conference Room, Veterans Hospital.

4:30 - 5:30 Dermatological Seminar; M-436, U. H.

5:00 - 5:50 Clinical Medical Pathologic Conference; Todd Amphitheater, U. H.

5:00 - 6:00 Urology-Roentgenology Conference; C. D. Creevy, O. J. Baggenstoss and Staffs; M-109, U. H.

Tuesday, May 16

7:30 - 9:00 Fracture Rounds; General Hospital.

8:00 - 9:00 Fracture Conference; Auditorium, Ancker Hospital.

8:30 - 10:20 Surgery Conference; Small Conference Room, Bldg. I, Veterans Hospital.

9:00 - 9:50 Roentgenology Pediatric Conference; L. G. Rigler, I. McQuarrie and Staffs; Todd Amphitheater, U. H.

10:30 - 11:50 Surgical Pathological Conference; Lyle Hay and E. T. Bell; Veterans Hospital.

11:00 - Contagion Rounds; Forrest Adams; Sta. L, General Hospital.

12:30 - Pediatric-Surgery Rounds; Drs. Stoesser, Wyatt, Chisholm, McNelson and Dennis; Sta. I, Minneapolis General Hospital.

12:30 - 1:20 Pathology Conference; Autopsies; J. R. Dawson and Staff, 102 I. A.

1:30 - 2:30 Pediatric-Psychiatry Conference; R. A. Jensen and Staff; 6th Floor, West Wing, U. H.

1:00 - 2:30 X-ray Surgery Conference; Auditorium, Ancker Hospital.

2:00 - 2:50 Dermatology and Syphilology Conference; H. E. Michelson and Staff; Bldg. III, Veterans Hospital.

3:15 - 4:20 Gynecology Chart Conference; J.-L. McKelvey and Staff; Station 54, U. H.

3:30 - 4:20 Clinical Pathological Conference; Staff; Veterans Hospital.

4:00 - 5:00 Physiology-Surgery Conference; Implications of Adrenal Impairment in Two Post-operative Patients; Bernard Zimmermann and Russell Nelson; Eustis Amphitheater, U. H.

4:00 - 5:00 Pediatric Rounds on Wards; I. McQuarrie and Staff; U. H.

5:00 - 6:00 Prophyrin Seminar; C. J. Watson, Samuel Schwartz, et al; Powell Hall Amphitheater.

5:00 - 6:00 X-ray Conference; Presentation of Cases by Veterans Hospital Staff; Drs. Fink, O'Loughlin, et al; Todd Amphitheater, U. H.

Wednesday, May 17

8:00 - 8:50 Surgery Journal Club; O. H. Wangensteen and Staff; M-109, U. H.

8:00 - 9:00 Roentgenology-Surgical- Pathological Conference; L. B. Thomas and L. G. Rigler; Todd Amphitheater, U. H.

8:30 - 9:30 Clinico-Pathological Conference; Auditorium Ancker Hospital.

8:30 - 10:00 Orthopedic-Roentgenologic Conference; Edward T. Evans and Bernard O'Loughlin; Room 1AW, Veterans Hospital.

Wednesday, May 17 (Cont.)

8:30 - 12:00 Neurology Rehabilitation and Case Conference; A. B. Baker, Veterans Hospital.

11:00 - Pediatric Rounds; Erling Platou; Sta. I, General Hospital.

11:00 - 12:00 Pathology-Medicine-Surgery Conference; Surgery Case; O. H. Wangensteen, C. J. Watson and Staffs; Todd Amphitheater, U. H.

12:00 - 1:00 Radio-Isotope Seminar; Report on Current Literature on Radioactive Isotopes; S. Gordon, J. Miree; 113 Medical Sciences.

12:15 - Staff Meeting; Main Classroom, General Hospital.

3:00 - Pediatric Rounds; C. J. Huenekens; Sta. I, General Hospital.

3:30 - 4:30 Journal Club; Surgery Office, Ancker Hospital.

4:00 - 5:00 Infectious Disease Rounds; Todd Amphitheater, University Hospitals.

4:00 - 5:30 Cardiovascular Conference; Rheumatic Fever: Biochemistry Studies, D. Glick and F. Adams; Endocarditis Produced by A-V Fistula, C. Lillehei; 106 Temporary Court, U. H.

5:00 - 5:50 Urology-Pathological Conference; C. D. Creevy and Staff; E-101, U. H.

5:00 - 7:00 Dermatology Clinical Seminar; Dining Room, U. H.

8:00 - Dermatological Pathology Conference; Todd Amphitheater, U. H.

Thursday, May 18

8:30 - 10:20 Surgery Grand Rounds; Lyle Hay and Staff; Veterans Hospital.

9:00 - 9:50 Medicine Case Presentationr C. J. Watson and Staff; M-109, U. H.

10:00 - 11:50 Medicine Ward Rounds; C. J. Watson and Staff; E-221, U. H.

10:30 - 11:50 Surgery-Radiology Conference; Daniel Fink and Lyle Hay; Veterans Hospital.

11:00 - 12:00 Cancer Clinic; K. Stenstrom and A. Kremen; Todd Amphitheater, U. H.

11:30 - Pathology Conference Clinic; Main Classroom; General Hospital.

11:30 - 12:30 Clinical Pathology Conference; Steven Barron, C. Dennis, George Fahr, A. V. Stoesser and Staffs; Large Classroom, Minneapolis General Hospital.

12:00 - 1:00 Physiological Chemistry Seminar; Flotation Lipids and Atherosclerosis; E. Shockett; 214 M. H.

1:00 - 1:50 Fracture Conference; A. A. Zierold and Staff; Minneapolis General Hospital.

4:15 - 5:00 Bacteriology Seminar; The Effects of pH upon Utilization of Riboflavin by Lactobacillus casei; D. Waldhalm; 214 M. H.

Thursday, May 18 (Cont.)

4:30 - 5:20 Ophthalmology Ward Rounds; Erling W. Hansen and Staff; E-534, U. H.

5:00 - 6:00 X-ray Seminar; Cystitis Emphysematosa; Joseph Faingold; Todd Amphitheater, U. H.

7:30 - 9:30 Pediatrics Cardiology Conference and Journal Club; Review of Current Literature 1st hour and Review of Patients 2nd hour; 206 Temporary West Hospital.

Friday, May 19

8:30 - 10:00 Neurology Grand Rounds; A. B. Baker and Staff; Station 50, U. H.

9:00 - 9:50 Medicine Grand Rounds; C. J. Watson and Staff; Todd Amphitheater, U. H.

10:00 - 11:50 Medicine Ward Rounds; C. J. Watson and Staff; E-221, U. H.

10:30 - 11:20 Medicine Grand Rounds; Veterans Hospital.

10:30 - 11:50 Otolaryngology Case Studies; L. R. Boies and Staff; Out-Patient Department, U. H.

11:00 - Pediatric Rounds; Erling Platou; Sta. I, General Hospital.

11:00 - 12:00 Surgery-Pediatric Conference; C. Dennis, O. S. Wyatt, A. V. Stoesser, and Staffs; Minneapolis General Hospital.

11:45 - 12:50 University of Minnesota Hospitals General Staff Meeting; Problems of Oxygen Therapy in Medicine; F. J Kottke, W. B. Kubicek, G. Gullickson, and G. K. Stillwell; Powell Hall Amphitheater.

12:00 - 1:00 Surgery Clinical Pathological Conference; A. A. Zierold, Clarence Dennis and Staff; Large Classroom, Minneapolis General Hospital.

2:00 - 3:00 Dermatology and Syphilology Conference; Presentation of Selected Cases of the Week; H. E. Michelson and Staff; W-312, U. H.

1:00 - 2:50 Neurosurgery-Roentgenology Conference; W. T. Peyton, Harold O. Peterson and Staff; Todd Amphitheater, U. H.

1:00 - 3:00 Pathology-Surgery Conference; Auditorium, Ancker Hospital.

3:00 - 4:00 Neuropathology Conference; F. Tichy; Todd Amphitheater, U. H.

4:00 - 5:00 Clinical Pathological Conference; A. B. Baker; Todd Amphitheater, U. H.

4:15 - 5:15 Electrocardiographic Conference; 106 Temp. Bldg., Hospital Court, U. H.

4:30 - 5:30 Journal Club; M-436, U. H.

Saturday, May 20

7:45 - 8:50 Orthopedics Conference; Wallace H. Cole and Staff; M-109, U. H.

8:30 - 9:30 Surgery Conference; Auditorium, Ancker Hospital.

9:00 - 9:50 Medicine Case Presentation; C. J. Watson and Staff; E-221, U. H.

9:00 - 10:30 Pediatric Grand Rounds; I. McQuarrie and Staff; Eustis Amphitheater, U. H.

9:15 - 10:00 Surgery-Roentgenology Conference; F. Ruzicka, O. H. Wangensteen and Staff; Todd Amphitheater, U. H.

10:00 - 11:30 Surgery Conference; O. H. Wangensteen and Staff; Todd Amphitheater, U. H.

10:00 - 11:50 Medicine Ward Rounds; C. J. Watson and Staff; E-221, U. H.

10:00 - 12:50 Obstetrics and Gynecology Grand Rounds; J. L. McKelvey and Staff; Station 44, U. H.

11:00 - Contagion Rounds; Forrest Adams; Sta. L, General Hospital.

11:00 - 12:00 Anatomy Seminar; Morphology of the Oculomotor Nucleus in the Cat; Harold Taft: Hypophyseal-adrenal Cortical Relationship; Harry Monsen; 226 I. A.