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## WATER AND SALT DEPLETION\*

BY

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### Comparison of Effects

The main differences are shown in Table V. Their theoretical explanation, as already suggested, appears to derive from the contrast in conditions of extracellular-fluid osmotic pressure. In pure water depletion (Fig. 4A) the extracellular fluid tends to become hypertonic and its volume to be maintained by withdrawal of water from the cells. In consequence of the relative normality of the plasma volume, oligæmic symptoms, such as lassitude,

total body salt; pure salt depletion, as reduction below normal of total body salt without reduction of body water beyond that which is inevitable from loss of extracellular osmotic pressure. Mixed water and salt depletion may be defined as reduction below normal of total body water and total body salt, the water reduction being greater than that consequent upon the secondary dehydration of salt depletion. This occurs in patients in whom there have been abnormal losses of secretions *without free intake of water*; the water loss tends to outstrip salt loss because the secretions lost are either isotonic (gastric or intestinal juices) or hypotonic (saliva, sweat), and, in addition, there is extra depletion of water in the continuous unavoidable losses, averaging about 1.5 litres daily, which occur from the skin and lungs and in the urine. Patients suffering from mixed depletion, as above defined (water loss in excess), have a mixture of the symptoms of pure water and of pure salt depletion. This is because the extracellular-fluid volume is diminished, due to the reduction in total sodium and chlorine, but there is also a tendency for the shrunken extracellular fluid to be hypertonic owing to the disproportionate water loss. The tendency to hypertonicity causes some withdrawal of water from the cells. The clinical picture, therefore, is such that patients show the circulatory and other features due to reduction of extracellular-fluid volume, but are also thirsty, have dry mouths, and have early oliguria.

Any condition causing secretion losses unaccompanied by adequate water intake will result in mixed depletion. Cases of acute vomiting provide the commonest examples. In protracted vomiting, such as occurs in pyloric stenosis, the effects of salt depletion tend to dominate the picture because water intake usually goes on until the late stages, so that there is a prolonged phase of selective drain of chlorine and sodium. Estimations of plasma concentrations of sodium and chlorine are even more fallacious as indicators of total loss of these ions in mixed depletion (water loss in excess) than in pure salt depletion, because of the tendency to hypertonicity in the diminished extracellular fluid. In any case, plasma chlorine estimations in vomiting, as already pointed out, are no guide to the concentration of sodium ions, because of the much greater loss of chlorine than of sodium (see Fig. 8). In mixed depletion due to vomiting it is possible for the concentration of sodium ions, largely partnered by HCO<sub>3</sub>, and the plasma osmotic pressure to be raised when the concentration of chlorine ions is decreased. In severe diarrhoea without vomiting (see Fig. 8) the converse may be true. The concentration of chlorine ions may be a little lower, or normal, or even raised while the concentration of sodium ions is decreased. Rogers (1921) found serum chlorides in cases of cholera before treatment to be as high as 0.79%.

TABLE V.—Comparison of Effects of Water and Salt Depletion

Manifestation	Pure Water Depletion	Pure Salt Depletion
Dehydration .. ..	+++ primary or simple	+++ secondary or extracellular
Thirst .. ..	+++	Absent
Lassitude .. ..	+	+++
Orthostatic fainting ..	Absent till late	+++
Urine volume .. ..	Scanty	Normal till late
NaCl in urine .. ..	Often +	Always absent except in Addison's disease
Vomiting .. ..	Absent	May be +++
Cramps .. ..	+	+++
Plasma NaCl .. ..	Slight increase or normal	Diminished +++
Blood urea .. ..	+	+++
Plasma volume .. ..	Normal till late	Decreased +++
Haemococoncentration ..	Not till late and slight	+++
Blood viscosity .. ..	Normal till late	Increased +++
Blood pressure .. ..	+	Fall +++
Water absorption .. ..	Rapid	Slow
Mode of death .. ..	? due to rise of osmotic pressure	Peripheral circulatory failure

giddiness, fainting, and fall of blood pressure, are absent or do not develop until a late stage. Thirst is pronounced because of cellular desiccation. In pure salt depletion (Fig. 4B) the extracellular fluid tends to become hypotonic and so water is not sucked from the cells and there is little or no thirst. The kidneys, working to preserve isotonicity of the extracellular fluid, excrete water; therefore, while water is being ingested and absorbed, urine volume is not significantly decreased, and may be increased. The volume of the extracellular fluid falls. The decrease of plasma volume causes prominent symptoms of peripheral circulatory failure due to oligæmia and increase of blood viscosity.

### Mixed Water and Salt Depletion

Pure water depletion may be defined as reduction below normal of total body water without significant reduction of

### Occurrence of Pure and Mixed Depletions in Practice

Pure water depletion is frequent in the circumstances already described; pure salt depletion or mixed water and salt depletion, however, are still more common because of the many causes of vomiting and diarrhoea. It is necessary to enumerate only a few of these causes to realize the frequency with which water and salt may be lost from the body. For example, vomiting may occur from gastritis, gastro-enteritis, appendicitis, pyloric or intestinal obstruction, and peritonitis; associated with pregnancy, after administration of anaesthetics, in alcoholism, in uraemia, in diabetic ketosis, in sea-sickness; and in such intracranial conditions as concussion, meningitis, cerebral tumours, and cerebral vascular lesions. Diarrhoea may occur from enteritis and colitis, from dysentery and cholera, from pancreatic insufficiency, in sprue, from ulcerative and neoplastic conditions of the intestines, and in patients in whom

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ileostomy has been performed (Cave, 1946). The special frequency of vomiting and diarrhoea in children is well known; their particular vulnerability to water and salt loss has already been mentioned.

The present frequency of pure salt depletion resulting from the treatment with water alone in those who have lost both salt and water has been emphasized. In recent months I have seen such unrecognized salt depletion in ten patients: two with severe gastro-enteritis, one with ulcerative colitis, two with chronic alcoholism, and in five after operation (gastrectomy, cholecystectomy, ileostomy, and two gastro-enterostomies). All ten had been diagnosed as suffering from dehydration. Four of the five post-operative cases had been treated with intermittent spells of gastro-duodenal suction drainage. All the patients had been on forced fluids, and intake and output charts were being kept. All had become extremely ill and were drowsy, apathetic, or stuporous. None complained of thirst, and none had chloride in the urine. All had lowered blood pressures; in three the systolic pressure was below 90 mm. Hg. The blood urea levels were raised, the highest being 280 mg. per 100 ml. After the diagnosis of salt depletion had been made, all, except one of the alcoholics, were treated by the administration of large amounts of intravenous saline (5 to 9 litres of isotonic saline) and all, except the untreated alcoholic and the ileostomy case (plasma Na 242 mg. %), rapidly recovered. In the Tropics I saw many instances of the salt-depletion syndrome, from simple sweating to sweating plus vomiting and diarrhoea, develop in patients suffering from various forms of medical and surgical illness.

The failure to recognize the true nature of the salt depletion syndrome often occurs because the patient's deterioration is explained in terms of pathological states which are essentially secondary but are mistakenly thought to be primary. Thus in many cases the frequent high blood urea levels lead to a diagnosis of uraemia. The tendency to vomiting in the late stages sometimes suggests a diagnosis of acute dilatation of the stomach or paralytic ileus; the lowered blood pressure and the manifestations of peripheral circulatory failure may cause the condition to be ascribed to "shock."

### Diagnosis

The differential diagnosis between water and salt depletion and mixed depletion is a comparatively easy matter, depending on recognition of their different modes of causation and awareness of the facts of their clinical and pathological differences.

Estimation of the chloride content of the urine is a most important diagnostic test. Qualitative diagnosis of the type of depletion is not enough. It is essential also that the attempt should be made to arrive at a quantitative diagnosis of the amount of the deficiency of water or salt or of water and salt.

### Prevention

This, again, is mainly a matter of awareness of the possible development of water and salt depletion. Fortunately, this state of alert anticipation now generally exists so far as water is concerned. It has become nearly as automatic to chart fluid intake and output of seriously ill patients as to record their temperature and pulse rate.

The best guarantee that water intake is sufficient is an adequate urine volume, in regard to which there are two points of practical importance. The first has already been mentioned: it is that in patients who have inefficient kidneys, incapable of secreting concentrated urine, the daily output of urine may need to be not less than 1.5 litres. Apart from permanent renal inefficiency, temporary

damage is frequent in illness; therefore it is seldom advisable to be satisfied with a urine volume of less than 3 pints or 60 oz. (1.7 litres) daily. The point is that, when gross secretion losses are occurred, 24 hours is too long a unit of time to allot for a review of the output; within its span the patient can die of dehydration. In such cases the urine volume and chloride content should be reported to the doctor eight-hourly. The aim should be a pint (570 ml.) per eight hours.

We are not generally aware of the potential hazard of salt depletion. However, it is to be hoped that its importance will soon be realized in this country in all cases suffering losses of alimentary secretions. In the Tropics, when patients are sweating, its menace enters into every form of medical and surgical illness. Whenever salt depletion may occur, the routine testing of the urine for chloride is a matter of first importance—more important, indeed, than the taking of the temperature or the counting of the pulse; not, of course, that these essential procedures should be omitted. The aim in adults should be an NaCl concentration of 3–5 g. per litre.

### Treatment of Established Depletions

At the outset it is desirable to call attention to the grave consequences of wrong treatment. In pure water depletion patients require water, and water only, until the deficit has been replaced. The administration of isotonic saline tends to increase existing extracellular hypertonicity, to intensify thirst, and to add to the difficulties of already embarrassed kidneys. It may be lethal. McCance and Young (1944) may be quoted: "Let us suppose that a patient is unable to take anything by mouth and that, in consequence, he is given, during 24 hours, 1,500 ml. of normal saline intravenously—that is, 1,500 ml. of water and (approximately) 15 g. of salt. He will require 1,000 ml. of this water to make good the unavoidable losses from his lungs and skin, and so his kidneys will have the impossible task of excreting 15 g. of salt in 500 ml. of water. . . . The casual administration of saline to young infants is even more dangerous than it is in adult life, for the infant kidney is much less capable of dealing with the superfluity of salt (Young, Hallum, and McCance, 1941; McCance and Young, 1941)." Wrong treatment in salt depletion, on the other hand, chiefly consists in failure to administer saline; in consequence the state of salt deficiency is not relieved and the patient may die of oligaemic circulatory failure. It is probable that hundreds, if not thousands, of patients in this country die annually from salt depletion. In the Tropics the mortality must be much greater.

Though the common and most important therapeutic error in salt depletion is the negative one of omitting to give salt, sometimes there are serious consequences due to the positive error of pressing the administration of water beyond the amount required to compensate unavoidable water losses. When the patient is collapsed and the pylorus closed, the administration of much water by mouth may lead to overfilling of the stomach and to regurgitation or vomiting of the gastric contents, in which the patient may drown, so gravely depressed is the cough reflex. I have seen it happen. Again, when the stage of low blood pressure has been reached anuria develops. Water introduced into the blood stream, whether by absorption from the alimentary tract or direct as intravenous glucose solution, cannot then be excreted in the urine and may cause death from water intoxication (Weir, Larson, and Rowntree, 1922; Helwig, Schutz, and Curry, 1935). Since the plasma and tissue fluid are hypotonic the water must enter the cells. It is probable that turgescence of the cerebral cells is the cause of death, which may be preceded by convulsions. It

is well known that "stoker's cramps" of salt-depletion heat exhaustion particularly tend to be precipitated by the ingestion of large amounts of water.

The special parenteral methods of administration of glucose solution subcutaneously and intraperitoneally—for example, in infants—may precipitate collapse in severe salt depletion by removal of salt from the already depleted plasma. This is because sodium and chlorine ions rapidly diffuse into a tissue depot of injected glucose solution (Darrow and Yannet, 1935). The administration of potassium salts is dangerous in sodium depletion. (In this connexion one has only to remember their danger in Addison's disease.) Sometimes the error of giving potassium to salt-depleted patients who are receiving sulphonamides arises from the routine administration of potassium citrate to alkalinize the urine. In such cases sodium citrate should be used.

Enough has been said about the consequences of wrong treatment to show that there are serious pitfalls in the treatment of different types of dehydration due to water and to salt depletion. The subject is far from being as simple as is often assumed—an assumption which may lead to the control of water and salt administration being left to nurses or junior residents. The essential principle in the treatment of water and salt depletion is that the patient shall be given water or salt, or both, in the amounts that each is lacking. Therefore, as in all conditions, correct diagnosis—in this instance quantitative as well as qualitative—is essential to correct treatment. The facts and principles on which diagnosis depends have been dealt with and need not be recapitulated. The main questions which arise in practical treatment are: (1) What fluid should be administered? (2) How much? (3) By what route? (4) At what rate?

### 1. The Fluid to Administer

In water depletion, water should be administered by mouth or per rectum, unless the circumstances of the case compel the use of alternative routes—intravenous, subcutaneous, or intraperitoneal. If one or other of these latter routes must be used, then the best vehicle for water is isotonic glucose solution (5%). In salt depletion, isotonic saline solution (0.85%) should be administered when the salt deficiency is giving rise to symptoms and while chloride is still absent from the urine. In very severe salt depletion, with vomiting and circulatory collapse, intravenous administration is indicated, as other routes may be too slow to save life, while oral administration carries the special risk of drowning or, short of drowning, of infecting the bronchial tree. When symptoms are relieved, or when chloride reappears in the urine, isotonic saline should be discontinued in favour of hypotonic saline. It is never desirable to continue isotonic saline administration longer than is necessary, because as soon as salt depletion is relieved there is a tendency for patients receiving isotonic saline to retain sodium and chlorine out of proportion to water (Coller, Job, Vaughan, Kalder, and Moyer, 1945). For adults the hypotonic concentration of choice is 1/2 isotonic (0.425%) until the salt balance appears to be fully restored, and then, if food is not being taken and salt intake must be derived from fluid 1/3 isotonic for maintenance. If parenteral routes are being employed these strengths may be achieved by mixing one part of isotonic saline solution with one or two parts of isotonic glucose solution. In the case of children particular care should be taken not to continue too long with isotonic saline; indeed, some authorities consider the possible dangers to infants to be so great that isotonic saline should never be used. Suitable hypotonic concentrations for infants are 1/4 or 1/5 isotonic—that is, for parenteral use, one part of isotonic saline mixed with three or four parts of isotonic glucose solution. In mixed water and salt depletion, or if there is any doubt as to the nature of the depletion, the patient should from the start receive hypotonic saline in the strengths above indicated.

It may be asked whether simple saline solution is sufficient to repair electrolyte deficiencies consequent upon loss of alimentary secretions, and, in particular, whether or not adjustments in the nature of the solution should be made to rectify alterations in the acid-base balance. This question is not really finally settled, but most authorities would, I think, agree with Gamble (1942) that saline or saline mixed with glucose solution is all that is needed. For a discussion of the considerations involved Gamble's monograph should be consulted.

### 2. Amount of Fluid

The essential principle is that the amount given, whether of water or saline, shall be enough to restore the patient to a normal state of balance. As shown in preceding sections, the initial deficit in a 70-kg. (or 11-stone) man may range up to a maximum of 10 litres (18 pints) of water in water depletion, or the equivalent of 10 litres of isotonic saline in salt depletion. The clinical criteria for the rough estimation of the volume of depletion have been given. In addition to the initial deficit, allowance must be made for losses going on during the period of fluid administration. The best check on restoration of water and salt is provided by eight-hourly measurements of the volume of urine and estimation of its chloride content. The aim per eight hours in an adult should be a urine volume of not less than a pint (570 ml.) and a salt content of 3 to 5 g. per litre. In infants urine measurement and testing for chloride present special practical difficulties. Yet they would seem to be even more important than in adults, because infants die so quickly from fluid depletion and because their kidneys have not the same power of correcting therapeutic errors. I have not the practical experience needed to attempt to define figures. All that can be said is that infants need to pass relatively much larger volumes of urine with lower chloride content.

During the intravenous administration of saline some NaCl may spill into the urine even though the patient is still grossly salt-deficient. For this reason if testing the urine shows the presence of chlorine it is wise to discontinue the saline, substituting glucose solution if it is desired to continue a drip infusion, and then half an hour and an hour and a half later instruct the patient to pass urine. The 30-minute specimen should be discarded and the 90-minute specimen tested for chlorine.

In fluid restoration a quantitative attitude of mind is essential, and it is necessary to realize clearly the considerable magnitude of fluid deficit which may be present in a case of water, salt, or mixed depletion. Fortunately, we are passing out of the days when patients deficient of perhaps 1½ to 2 gallons (6.8 to 9 litres), and suffering continuing losses, received a pint or two of fluid, though the approach is still often too homoeopathic.

### 3. Route of Administration

Some remarks concerning this question have already been made. In severe salt or in mixed depletion the intravenous route should be used and the continuous-drip method employed. Otherwise, whenever circumstances permit, the oral route is the best. If the patient has difficulty in drinking, a most valuable device is the transnasal intragastric drip. This method consists in fluid administration, by continuous drip, through a Ryle's tube passed via the nose into the stomach. It was particularly exploited by Ransome, Gupta, and Paterson (1944), who have recorded their experience with 355 acute medical cases, and their paper should be consulted for details of the very simple technique. Their work was done at a large military hospital in Assam, and more than 100 of the cases were in-patients in various degrees of coma from cerebral malaria. The nursing facilities were far from being on the scale of British civil hospitals, yet the method was found easy to run in practice. Nauth-Misir (1946) has written on the use of this method in infants, in whom it would seem to be particularly useful. The technique of the continuous intravenous drip, originated by Matas (1924), is too well known to need comment.

I would like to draw attention to the value of rectal fluid administration when properly carried out in accordance with the principles of Murphy (1909, 1916). The technique and also other methods of parenteral fluid administration have been described (Marriott and Kekwick, 1937).

## 4. Rate of Administration

The desirable rate of fluid administration depends on the state of the patient. The need for rapid intravenous administration arises in patients suffering from peripheral circulatory failure due to severe salt depletion or mixed depletion. Such cases are urgent emergencies, and the rate of administration for adults should be of this order: the first pint in 10 minutes, the second pint in 15 minutes, the third pint in 20 minutes, and the fourth pint in 30 minutes; then a pint every two hours till the blood pressure is restored to normal. Thereafter a pint every four hours (more under tropical conditions). In infants the quantities should be on a similar scale in proportion to body weight. In less acute depletions slower restoration is indicated. However, an adult suffering from a degree of either water or salt depletion sufficient to cause symptoms will need to receive within the first 24 hours a minimum of a gallon (4½ litres) of appropriate fluid. Watch must always, of course, be kept on urine excretion and for signs of oedema, especially pulmonary oedema.

## Complicated Cases

Special difficulties and dangers are associated with fluid administration to patients who have suffered from acute haemorrhage or who are anaemic or hypoproteinaemic (Power, Pedersen, and Maddock, 1942); to those in whom anoxia or toxæmia has increased capillary permeability, especially the permeability of pulmonary capillaries (Daly, 1946); and to those whose kidneys are badly damaged. In all such cases most careful consideration must be given to what is done, and very close watch maintained for the development of pulmonary oedema during fluid administration. Patients who have suffered from haemorrhage should receive preliminary blood transfusion to restore blood volume. Those who are seriously anaemic should, if possible, have their haemoglobin restored by drip transfusion (Marriott and Kekwick, 1935, 1940) while fluid administration by other routes proceeds. Patients with hypoproteinaemia should, if possible, similarly receive plasma transfusions, and anoxic patients should be given oxygen.

Water and salt are, perhaps, practically the most important substances it is in our power to administer. They can be so used that they can achieve seeming miracles; or they can be so misused as to lead to a fatal issue. Their proper use is not the simple matter it is often assumed to be. The true approach to understanding is one of humility.

## Postscript

A few days before these lectures were delivered an article by Prof. E. B. Verney appeared (*Lancet*, 1946, 2, 739 and 781) which I did not see until later. In this important paper, recording much precise experimental work, it is shown that liberation of posterior pituitary antidiuretic hormone is determined by the osmotic pressure of the arterial plasma. The hypothesis is advanced that changes in this pressure are appreciated by "osmoreceptors" which are continually engaged in controlling the antidiuretic function of the pituitary. Verney's work thus confirms, from another angle, the paramount importance of the maintenance of the isotonicity of the extracellular fluid. It also implies that renal activity in preserving such isotonicity may be largely or wholly dependent on the functional linkage between the neurohypophysis and the kidneys. The influence of the pituitary antidiuretic hormone on renal water excretion has, of course, been known for many years—largely due to the researches of Verney. However, his latest work, showing that the production of this hormone is related to plasma osmotic pressure, makes a big forward step. His findings do not affect the clinical views expressed in these lectures, but they are important in helping to reveal the physiological mechanism by which the kidneys guard the osmotic constancy of the "internal environment."

I wish to thank Prof. J. L. Gamble, of Harvard, for his generous permission to reproduce his diagrams (Gamble, 1923, 1942) showing the composition of the body fluids. His concise monograph (1942) is a masterpiece of lucid verbal and diagrammatic exposition. To Prof. R. A. McCance I am indebted for supplying references, and I would like to pay tribute to the great importance of the work he has done in this field. I wish also to thank Dr. D. A. K. Black, Dr. A. Kekwick, and Dr. N. F. Maclagan, who have kindly given me references.

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## THE SWALLOWING OF FOREIGN BODIES

BY

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Recently an epidemic of foreign-body swallowing resulted in the admission of 11 cases into a military hospital in the course of a few months.\* For the purpose of investigation the 11 "swallowers" were compared with 11 psychopaths and 11 clearly stable prisoners as controls. The psychopathic controls were the usual unstable, impetuous, emotional types with overflowing crime sheets; the normal controls were deserters, most of them young men whose nerve had gone during the course of the Italian campaign, but who had otherwise blameless records and were well-behaved in prison. I interviewed the prisoners alone, if necessary several times. I explained that the inquiry was a medical one, that nothing they said would be held against them, and that they had nothing to gain personally, though ultimately all prisoners might benefit from the information obtained.

\* See Major Hallett's paper, which includes cases not in this series. Some of my series had had operations in other hospitals previously, hence any discrepancies.

## Results

The most striking fact which emerged was that the "swallowers" (Group I) fell very definitely into two subgroups: (a) those who had only pretended to swallow, or who had swallowed harmless objects in an entirely calculating manner in the hope of being sent to hospital, which was outside the prison; and (b) those who had swallowed objects when in a highly emotional state with a considerable degree of depression present. In the latter group operation was necessary on various occasions, the six of them having had 15 abdominal operations in all (over a period of months or years) compared with two operations needed by the former. The accompanying tables make a comparison of the swallowers and the control groups on matters which I hoped would throw light on the causes of the swallowing; to facilitate ease of comparison the subgroups Ia and Ib are compared on a basis of 11 (see the figures in parenthesis).

Table I (A) shows the preponderance of bad home conditions in childhood. As the items enumerated—

TABLE I

	Group Ia* (5 Cases)	Group Ib (6 Cases)	Group II (11 Cases)	Group III (11 Cases)
No. of operations required	2 (4)	15 (27)	0	0
A. Home conditions in childhood:				
Father violent .. ..	0	3 (6)	1	0
" alcoholic .. ..	0	2 (4)	2	1
" brutal .. ..	1 (2)	3 (6)	3	1
Parental disharmony ..	1 (2)	2 (4)	4	1
" separation .. ..	0	1 (2)	1	1
Total .. ..	2 (4)	11 (20)	11	4
B. Youthful instability:				
Truancy from school ..	1 (2)	2 (4)	3	0
Adolescent delinquency ..	1 (2)	3 (6)	4	0
Violent-tempered child ..	2 (4)	2 (4)	1	0
Nervous child .. ..	2 (4)	1 (2)	2	0
In reformatory .. ..	2 (4)	0	2	0

\* The results showed Groups Ia and Ib so essentially different that the comparison of figures for the total of Group I seemed valueless. The figures in parenthesis are on a basis of 11 (approx.).

alcoholic father, matrimonial disharmony, etc.—are various aspects of an unsatisfactory home situation I felt it legitimate to add these; it will be seen there is the usual preponderance of these factors in the psychopaths, whether swallowers or not. Table I (B) shows a similar but less striking difference in traits indicative of instability in childhood. The matters recorded in Table II were designed to

TABLE II.—Adult Instability and Affective Reactions

	Group Ia (5 Cases)	Group Ib (6 Cases)	Group II (11 Cases)	Group III (11 Cases)
Anxious in prison .. ..	1 (2)	6 (11)	4	3
Depressed .. ..	0	6 (11)	3	2
Emotional .. ..	2 (4)	4 (8)	4	1
Aggrieved .. ..	0	2 (4)	1	0
Paranoid attitude .. ..	0	2 (4)	1	0
Aggressive .. ..	1 (2)	3 (6)	1	0
Harassed and drawn facies ..	1 (2)	6 (11)	3	1

reveal traits indicative of markedly emotional personalities. Anxiety and depression were recorded from subjective complaints; the other items were my observations. It will be seen that there is a steady decline in the incidence of affective reactions from Group I to Group III, but the most striking difference is between Groups Ia and Ib. Of the latter group of 6 "true swallowers," 6 were anxious and depressed, 4 had drawn facies, and 4 were very emotional, in contrast to 1, 0, 1, 2, for the same traits in Group Ia.

Table III deals with factors which my previous experience of prisoners suggested might be of aetiological significance—namely, treatment by the prison staff, prohibition of smoking, and the food. It will be seen that the results