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DANGERS IN LOW SALT DIET THERAPY
RAY F. BEERS, M.D.

THE concept of the salt retaining kidney has provided the impetus for the use of low salt diets in a variety of clinical conditions. Kempner has recommended the rice-fruit diet for the treatment of benign and malignant hypertension and their renal complications, as well as for arteriosclerotic heart and kidney disease. Recent literature has stressed the importance of salt restriction and mercurial diuretics in the management of congestive heart failure, and the statement has been made that these two procedures are of more importance than digitalis. Salt restriction has been used with success in the treatment of pre-eclampsia and eclampsia, the nephrotic stage of nephritis, in cirrhosis of the liver with ascites, in D.O.C.A. overdosage and in conjunction with therapy with ACTHAR and Cortone. That most therapeutic procedures are potentially two edged swords is axiomatic and it is the purpose of this paper to demonstrate the dangers of misuse of salt restriction.

While the average daily intake of salt varies from 4-8 Grams/day, it has been estimated that a normal person requires about 6 Grams/day. Strict low salt diets may reduce the intake to as little as 500 mgm./day (or 200 mgm. sodium). Intakes as low as this even when not accompanied by mercurial diuresis must necessarily make the danger of salt depletion a real one.

In an effort to find the incidence of complications due to salt depletion, in patients on low salt diets, we reviewed all the patients who had been on salt restriction in the Allentown Hospital during the year 1950-1951. Cases reviewed in detail totalled 473, and of these, 142 cases were considered to be usable material. All cases that had been on salt restriction for less than eight hospital days were not used in this analysis.

<table>
<thead>
<tr>
<th>Total cases of salt depletion (of any severity)</th>
<th>30</th>
</tr>
</thead>
<tbody>
<tr>
<td>Per cent of cases showing salt depletion</td>
<td>21.1%</td>
</tr>
<tr>
<td>Salt depletion on low salt diet alone</td>
<td>10 (7.0%)</td>
</tr>
<tr>
<td>Salt depletion on low salt diet plus mercurials</td>
<td>20 (14.1%)</td>
</tr>
<tr>
<td>Salt depletion plus previous renal damage</td>
<td>25 (17.6%)</td>
</tr>
<tr>
<td>Salt depletion without previous renal damage</td>
<td>5 (3.5%)</td>
</tr>
<tr>
<td>Percent of salt depletion cases showing previous renal damage</td>
<td>83.3%</td>
</tr>
</tbody>
</table>
Overall mortality with salt depletion as a precipitating or contributing cause .......................... 12 (8.4%)
Mortality in salt depletion cases ........................................ 12 (30%)
Average age of salt depletion cases .................................. 59 yrs.
Age range of salt depletion cases ..................................... 23 to 83 yrs.
Recognized cases ............................................................. 5 (16.6%)

The statistical analysis adequately demonstrates that patients with signs of previous renal impairment are more liable to salt depletion. However the figures also show that previous renal damage is not necessary for this complication to arise, contradicting somewhat the work of Soloff and Zatuchni, who stress the necessity of previous renal damage, though often mild. We see no theoretical ground for assuming the necessity of renal impairment, for heat cramps can and do develop in patients with perfectly normal renal function. Proger and O'Conner have noted the syndrome in patients with normal kidneys and in their series, as in ours, it is noteworthy that very few cases showed true renal insufficiency prior to the development of salt depletion, the signs of damage being restricted in most cases to trace albuminuria and moderate fixation of the urine specific gravity. B.U.N. determinations were for the most part normal. Schroeder comments on this in his analysis as well. Therefore the danger of salt depletion is real in patients with but minor changes in renal function, and the use of low salt diets in patients with advanced renal insufficiency is exceedingly hazardous. Our series includes cases that were kept on low salt diets even in the uremic state with very low CO2 powers, as well as cases of malignant hypertension with renal failure who were placed on low salt diets to their detriment.

Kempner's diet, while low in salt, is also low in fat and protein, and it cannot be legitimately assumed that the low salt part of the diet is the only thing of value. Furthermore, no one else has been able to reduplicate Kempner's results even when following his therapy in strict accordance with his own rules. He himself warns of the danger of electrolyte depletion in patients on his diet and insists on frequent examinations of the blood and urine chlorides to detect it. He cites instances of its occurring, though he gives no idea as to the frequency of its occurrence, and no inkling as to how to prevent or treat the salt depletion when it does occur. Rice-fruit diets are not without danger even in ambulatory cases of benign essential hypertension. The group at the hypertensive clinic at the Massachusetts General Hospital finds that a significant number of benign hypertensives have a "salt losing" kidney, i.e. despite rigid salt restriction the
patient continues to excrete normal concentrations of salt with no effort towards salt conservation, the net result for this type of patient can only be salt depletion. Salt conservation function is lost by any damaged kidney hence the great danger of depletion. Furthermore, the Massachusetts group cannot find any particular advantage to Kempner’s regime as opposed to conservative medical care in a control group.

It is of significance that of the 30 cases of salt depletion encountered in this series, 20 had been on mercurial diuretics. The primary action of mercurials is on the renal tubules and any extra-renal effects are negligible. Good evidence shows that mercury primarily inhibits the reabsorption of chlorides and water, but regardless of primary action it inhibits the reabsorption of sodium and potassium as well. Many investigators have noted urinary concentrations of sodium chloride greater than plasma concentrations during a mercurial diuresis, and in therapy we usually allow water ad libitum; the net result can only be dilution of the extra-cellular electrolytes. The primary action of the mercurials being chloride elimination, the earliest and most commonly encountered part of the low-salt syndrome is hypochloremia with a normal CO₂ power, but as the syndrome becomes more advanced, the serum sodium becomes depleted and the CO₂ power drops. Giving chlorides alone, as ammonium chloride, or potassium chloride will not prevent the development of the low salt syndrome, and symptoms will develop in the face of virtually normal serum chlorides. These cases will show a depressed CO₂ power only, but clinically the picture is the same as when both sodium and chloride are depressed.

Though rapid diuresis with mercurials is the usual cause of salt depletion in congestive heart failure, it has been shown that the syndrome may develop when no mercurials have been used, and one case in our series illustrates this point.

The clinical picture of a patient who is suffering from the low salt syndrome usually takes three to five days to develop, though it may take longer if mercurials are not being used, and the picture is as follows:

1. Drowsiness, weakness, lethargy—the patient becomes very apathetic.
2. Anorexia and nausea—occasionally vomiting.
3. Muscular and abdominal cramps develop.
4. Confusion and headache develop and may progress to psychosis and/or coma.
5. Concentration of chlorides in the urine falls progressively.
6. Urine output falls to oliguria and even anuria.
7. Patient becomes refractory to further mercurials—“mercury fast”.
8. Symptoms secondary to increasing edema develop.
   a. rapid weight gain
   b. increased peripheral edema if present
   c. pulmonary edema may develop
9. Signs of cardiovascular collapse with cyanosis, blood pressure drop, rapid pulse, etc.

   The blood chemistry shows distinctive alterations in a progressive manner, the serum chlorides being the first to fall, followed in more severe cases by a fall in the CO2 comb. power. Concomitantly the B.U.N. begins to rise and may reach uremic levels. Cases which have been on ammonium chloride therapy will have all the clinical signs but the serum chlorides may be within normal range. But these cases show the fall in CO2 power and the rise in B.U.N. quite clearly and this should not lead to confusion.

   Commonly, this situation presents itself; a patient in marked congestive heart failure who has made an initial good response to salt restriction and mercurial diuretics, begins to look “bad” and further mercurials fail to evoke any response, the patient becoming even more apathetic and the expected diuresis becomes actual oliguria. If now the situation is not recognized and treated, and as often happens, further mercurials are given in an effort to establish a diuresis, the patient goes steadily down hill to his death. If recognized and treated properly the patient will improve rapidly, as the syndrome is completely reversible. Furthermore with salt restored, the patient will once again be sensitive to mercurial diuretics.

   Another manner in which the low salt syndrome can be produced is by the use of the newly promulgated cat-ion exchange resins, and two of our cases illustrated this well. One of these cases was a “salt-snitcher” who had taken large doses of mercurials without any signs of depletion, but seven days after the resin was started he showed all the signs of early salt depletion. Since the resins remove only cat-ions the serum chlorides are not affected by them but the CO2 power falls and the B.U.N. rises just as in the cases treated with ammonium chloride. This variation of the low salt syndrome is probably better termed “low sodium syndrome”.

   Salt depletion has been precipitated by the withdrawal of large quantities of ascitic fluid from patients with cirrhosis of the liver who
had been on low salt diets and mercurial diuretics. One case in our series demonstrates a similar mechanism, that has not been previously reported to our knowledge. This boy was in the nephretic stage of nephritis with massive anasarca. He was placed on a low salt diet, without any mercurials whatsoever. Massive amounts of fluid were removed by Southey tubes, following which the signs and blood chemistry findings of salt depletion promptly presented themselves.

Cardiac surgical cases requiring intubation often show signs of salt depletion, due to the replacing of fluids containing glucose alone. As a matter of fact this is the quickest method of producing the low salt syndrome experimentally on dogs.

Hot weather has in our experience been especially favorable for the occurrence of electrolyte depletion even when mercurials are not being used. This is especially true of the more elderly patients, and we routinely raise their salt intake to 1 to 2 Grams/day during July and August.

Treatment of the low salt syndrome is little different from the treatment of water intoxication which it so closely resembles. In mild cases, merely restoring the normal salt intake back to the diet is sufficient. However in the more severe cases, more direct and speedy therapy is often required. Since the syndrome consists in its essence of a hypotonic condition of the extra cellular fluids, the treatment resolves itself into giving as much salt as is necessary, with as little water as possible. For this reason 5% saline solution is the fluid of choice. Usually 250-500 cc, depending on the severity of the salt depletion, suffice. The solution is given by slow intravenous drip and studies have shown no increase in the venous pressure during these infusions. Theoretically there is no reason to expect the venous pressure to rise, for salt is readily diffusible and no rise has been encountered under clinical conditions. The patient treated with this solution responds promptly, the effect often being quite dramatic, and the B.U.N. drops concomitantly with the clinical improvement. Mercurials become effective once again. While the syndrome is usually reversible; if unrecognized until cardiovascular collapse is encountered it is doubtful if many of these will respond, hence the importance of recognizing the syndrome as quickly as possible and of treatment as soon as the diagnosis is established.

SUMMARY

1. The uses and abuses of low salt diets have been discussed.
2. It has been shown that cases with poor renal function are poor risks for low salt diet therapy.
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<td>Art. Sel. H.D.</td>
<td>70</td>
<td>1.010</td>
<td>Lt.</td>
<td>35.</td>
<td>21.</td>
<td></td>
<td></td>
<td>Salt free</td>
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<tr>
<td></td>
<td>Cong. failure</td>
<td></td>
<td>(high)</td>
<td>Cloud</td>
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<td>Low salt disc.</td>
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<tr>
<td></td>
<td>Auric. fibrillation</td>
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<td>(85)</td>
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<td>Thiomerin</td>
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<td>Hyp. C.V.D.</td>
<td>61</td>
<td>1.018</td>
<td>Heavy</td>
<td>47.</td>
<td>41.</td>
<td>440</td>
<td>Low salt</td>
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<td>congestive fail.</td>
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<td>Cloud</td>
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<td>10 days</td>
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<tr>
<td>257,503</td>
<td>Sub. acute nephritis</td>
<td>29</td>
<td>1.022</td>
<td>3.5</td>
<td>47.</td>
<td>39.</td>
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<td></td>
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<td>gm/1</td>
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<tr>
<td>261,148</td>
<td>Malignant hypertension</td>
<td>29</td>
<td>1.020</td>
<td>Heavy</td>
<td>76.</td>
<td>23.</td>
<td>400</td>
<td>Nor</td>
<td>None</td>
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<td></td>
<td></td>
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<td>Cloud</td>
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<tr>
<td>254,874</td>
<td>Hypertension enceph. cirrhosis of liver</td>
<td>62</td>
<td>1.012</td>
<td>Light Cloud</td>
<td>26.79</td>
<td>470</td>
<td>Low salt</td>
<td>None</td>
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<td>Na. cl. 1 gm.</td>
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<td>T. l. D. (1st 5 days)</td>
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<tr>
<td>248,512</td>
<td>Malig. Hypertension</td>
<td>28</td>
<td>1.008</td>
<td>neg.</td>
<td>55.</td>
<td>26.</td>
<td>500</td>
<td>200 mgm</td>
<td>Mercuzanthine</td>
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<tr>
<td></td>
<td>congestive failure</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td>Na.</td>
<td>1 cc x 9</td>
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<td></td>
<td></td>
<td></td>
<td>36 days</td>
<td>Thiomerin</td>
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<td></td>
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<td></td>
<td>1 cc x 2</td>
</tr>
</tbody>
</table>

Comment:
- Known cardiac on mercurials and low salt diet adm. with edema, paralytic ileus and crampy abd. pain, vomiting necessitating intubation. Pt. died on 10th day without adequate salt replacement. Autopsy revealed no organic obstruction.
- Pt. placed on low salt diet in face of rising B.U.N. returned in 3 months with severe salt depletion, uremic. Expir'd.
- Pt. under Rx for hypt. lyr. prev. to admission. Drowsy, confused and speech slurred, complained of peri-umbilical pain on admission. Improved markedly when salt was given. Rapidly depleted himself again when salt restricted. Sent home without salt restriction. Errorously diagnosed.
- Pt. became progressively worse on Rx. Began to vomit at which time 9 gm. salt given I.V. Improved for a time then got progressively worse again. Signed release and left.
<table>
<thead>
<tr>
<th>Case Number</th>
<th>DIAGNOSIS</th>
<th>Age</th>
<th>Spec.</th>
<th>ALB.</th>
<th>B.U.N.</th>
<th>CO2</th>
<th>CHL</th>
<th>DIET</th>
<th>MERCURIALS</th>
</tr>
</thead>
<tbody>
<tr>
<td>258,824</td>
<td>Nephrotic nephritis</td>
<td>23</td>
<td>1.016</td>
<td>4+</td>
<td>16</td>
<td>54</td>
<td>30</td>
<td>Low salt</td>
<td>None</td>
</tr>
</tbody>
</table>

**COMMENT:** Massive amounts of fluid removed by Southey tubes following which anorexia and weakness developed CO2 and chl were depressed.

<table>
<thead>
<tr>
<th>Case Number</th>
<th>DIAGNOSIS</th>
<th>Age</th>
<th>Spec.</th>
<th>ALB.</th>
<th>B.U.N.</th>
<th>CO2</th>
<th>CHL</th>
<th>DIET</th>
<th>MERCURIALS</th>
</tr>
</thead>
<tbody>
<tr>
<td>261,245</td>
<td>Malig hypertension</td>
<td>36</td>
<td>1.010</td>
<td>4+</td>
<td>73</td>
<td>17</td>
<td>404</td>
<td>Low salt</td>
<td>Thiomerin 1 cc x 6</td>
</tr>
</tbody>
</table>

**COMMENT:** Malig. hypertension with severe renal damage. Low salt diet resulted in a rise in B.U.N. and severe depression of serum salt corrected and brought to normal limits with Na. therapy by enema and mouth. Replaced on low salt diet and given mercurials. 15 days later expired with B.U.N. of 80 and CO2 13.

<table>
<thead>
<tr>
<th>Case Number</th>
<th>DIAGNOSIS</th>
<th>Age</th>
<th>Spec.</th>
<th>ALB.</th>
<th>B.U.N.</th>
<th>CO2</th>
<th>CHL</th>
<th>DIET</th>
<th>MERCURIALS</th>
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<tbody>
<tr>
<td>254,046</td>
<td>Art. Sel. C.V.D.</td>
<td>78</td>
<td>1.004</td>
<td>neg.</td>
<td>29</td>
<td>48</td>
<td>600</td>
<td>Low salt</td>
<td>None given for 1 month prev. to development of symptoms</td>
</tr>
</tbody>
</table>

**COMMENT:** Pt. became apathetic and semi-comatose on a low salt diet. CO2 corrected with 6 M Na. Lactate and definite clinical improvement. Died of Myocardial infarction 2 days after Na. Lactate was given.

<table>
<thead>
<tr>
<th>Case Number</th>
<th>DIAGNOSIS</th>
<th>Age</th>
<th>Spec.</th>
<th>ALB.</th>
<th>B.U.N.</th>
<th>CO2</th>
<th>CHL</th>
<th>DIET</th>
<th>MERCURIALS</th>
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<tbody>
<tr>
<td>265,785</td>
<td>Pulmonary Tubercul.</td>
<td>62</td>
<td>1.009</td>
<td>neg.</td>
<td>@10</td>
<td>468</td>
<td>346</td>
<td>Low salt</td>
<td>Thiomerin 1 cc x 2</td>
</tr>
</tbody>
</table>

**COMMENT:** Pt. admitted with pulmonary moisture and 4+ pitting edema—cheat cleared of moisture in 24 hours—peripheral edema disappeared in 48 hours—on 7th day pt. looked critical—pulmonary moisture reappeared—CO2—73 chl-346 500 cc of 5% salt I.V. prompt and dramatic improvement. CO2—73 — Chl—500.
<table>
<thead>
<tr>
<th>Case Number</th>
<th>DIAGNOSIS</th>
<th>Age</th>
<th>Spec. Grav.</th>
<th>ALB.</th>
<th>CO2 Power</th>
<th>CHL</th>
<th>DIET</th>
<th>MERCURIALS</th>
</tr>
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<tbody>
<tr>
<td>264,494</td>
<td>Diabetes mellitus</td>
<td>51</td>
<td>1.012</td>
<td>Light</td>
<td>@22</td>
<td>54</td>
<td>Low salt</td>
<td>Mercurials</td>
</tr>
<tr>
<td></td>
<td>chronic congestive heart failure</td>
<td></td>
<td></td>
<td>Cloud</td>
<td>47</td>
<td>33</td>
<td>44 Days</td>
<td>Thioromerin 2 cc</td>
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<td></td>
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<td></td>
<td></td>
<td>persist</td>
<td>640</td>
<td></td>
<td>plus</td>
<td>x 20</td>
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<td></td>
<td></td>
<td></td>
<td>determinations</td>
<td></td>
<td></td>
<td>Resodex</td>
<td>Thioromerin gr xv</td>
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<td></td>
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<td></td>
<td>for 7 days</td>
<td>T.I.D.</td>
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**COMMENT:** Pt. lost 50 lbs. in 7 weeks. No sign of salt depletion—Resodex started and 7 days later patient complained of anorexia and nausea. CO2—53 B.U.N.—47 Salt replaced in diet and patient improved.

<table>
<thead>
<tr>
<th>Case Number</th>
<th>DIAGNOSIS</th>
<th>Age</th>
<th>Spec. Grav.</th>
<th>ALB.</th>
<th>CO2 Power</th>
<th>CHL</th>
<th>DIET</th>
<th>MERCURIALS</th>
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<td>260,070</td>
<td>Congest. failure</td>
<td>56</td>
<td>1.020</td>
<td>neg.</td>
<td>21</td>
<td>47</td>
<td>Low salt</td>
<td>Thioromerin</td>
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<tr>
<td></td>
<td>persistent rt. hydrothorax</td>
<td></td>
<td></td>
<td></td>
<td>33</td>
<td>39</td>
<td>plus</td>
<td>1 cc x 5</td>
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<tr>
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<td></td>
<td></td>
<td>45</td>
<td>Resodex</td>
<td>2 cc x 15</td>
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<td>610</td>
<td>Gm. 15 t.i.d.</td>
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<td></td>
<td></td>
<td>plus Am. chl.</td>
<td>7 days</td>
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</table>

**COMMENT:** This patient did not become depleted of electrolytes despite massive mercurial therapy—but 7 days after Resodex was started complained of anorexia and lethargy and crampy abd. pain CO2 had dropped to 39 Vols%—corrected by placing salt back in the diet.
3. It has been shown that salt depletion can occur in patients with no objective evidence of impaired renal function.
4. Mercurial diuresis plus low salt diets have been shown to be the commonest causes of the low salt syndrome.
5. Sodium depletion and the development of the low salt syndrome through the use of cation exchange resins has been mentioned.
6. Salt depletion precipitated by use of Southey tubes has been illustrated.
7. The logical treatment of salt depletion has been shown to be hypertonic saline solutions.

BIOGRAPHY

5. Soloff and Zatzchi-Syndrome of Salt Depletion induced by Regimen of Sodium Restriction and Sodium Diuresis J.A.M.A. 139:1135 April 1949.
PAROXYSMAL TACHYCARDIA IN INFANTS

DR. FORREST G. MOYER, M.D., F.A.A.P.

Paroxysmal tachycardia is a condition in which the heart beats very rapidly at a regular rate. In this condition the sinus pacemaker has lost control and the heart beat is initiated by a temporary pacemaker located in the auricles, in the auriculoventricular node, or in the ventricles. The ventricular form is rare in infancy and childhood.

Paroxysmal tachycardia may be regarded as a succession of premature systoles. The onset is abrupt; the duration may be a few moments, hours, or several days, and the end is as abrupt as the beginning. The rate is regular and usually in the neighborhood of 200 beats per minute.

Paroxysmal tachycardia occurs frequently in adults and its recognition is usually easy because of the signs and symptoms which focus attention on the cardiovascular system. The rapid heart rate is easily detected by auscultation.

The occurrence of paroxysmal tachycardia in infants is determined less easily, since a baby does not describe his symptoms and the heart sounds may easily be obscured by crying or noisy respiration. Unless the rapid heart action is interrupted by therapy, the course may be steadily downhill with a fatal termination. It is in this respect that paroxysmal tachycardia in infancy differs from that commonly seen in the older patients. Its importance as a clinical entity somewhat differs from the tachycardia seen in older children and adults which was emphasized for the first time by Hubbard in 1941. Since that time a number of case reports have appeared in the literature.

CAUSE

The cause of paroxysmal tachycardia in infancy is obscure. Certain congenital malformations, particularly defects of the auricular septum, may predispose to various types of arrhythmias, but in the great majority of instances no pathology has been discovered in the heart. Once the attacks have been overcome, the heart appears to be normal. In some instances the attacks seem to arise spontaneously. In others a respiratory or other infection may be a precipitating factor. It may occur at any period during infancy, but it is particularly likely to arise during the first few weeks of life. The attacks are prone to be more severe and prolonged in younger infants if untreated.
SYMPTOMS

The symptoms are fairly characteristic. The baby becomes restless and irritable. Feedings are taken poorly. Vomiting is frequent. The color is ashen gray and distinct cyanosis may appear as the attack progresses. The respiratory rate is increased and may go as high as 150 per minute. Prostration is in proportion to the severity of the attack. Slight fever is often present.

The rapid respiration and the occurrence of rales in the chest make auscultation of the heart difficult, and the diagnostic rapid heart rate may be detected only by careful examination. The rate is perfectly regular and so fast that it is almost always above 200 per minute and may be above 300 per minute. X-rays of the chest may show increased bronchovesicular markings together with a widening of the upper mediastinal shadow. A diagnosis of bronchopneumonia and enlarged thymus is frequently made. The signs of congestive heart failure including edema, enlargement of the liver, dyspnea, and dilatation of the heart may occur. Failure to terminate the attack may produce a fatal outcome.

TREATMENT

In the older child and adult the attack normally subsides in the course of time, even in the absence of specific measures. The measures usually employed in these cases, such as massage or compression of the carotid sinus, pressure on the eyeballs, the assumption of some particular position, or the induction of vomiting are rarely effective in the treatment of paroxysmal tachycardia in infants. Furthermore the use of such drugs as quinine, quinidine, pilocarpine, or physostigmine has been disappointing. Fortunately this type of tachycardia in infants seems to respond very well to the use of digitalis. This was first emphasized by Hubbard.

The amount of digitalis necessary to restore normal rhythm varies tremendously. Oftentimes a much larger total dosage is required than would be regarded as necessary to produce full digitalization in accordance with the patient's weight. If the symptoms are not alarming, the digitalis preparation may be given by mouth. Gibson usually employs 50 mgm. (¾ grain) as the initial dose followed by 25 mgm. (½ grain) at 4-hour intervals. Sometimes normal rhythm is established in 24 hours. In other instances two or three days of treatment may be necessary. In more urgent cases, the digitalis has been given intramuscularly. Doses as high as a total of 300 mgm. (4½ grain) have
been given to a baby weighing less than eight pounds. The majority of infants maintain a normal sinus rhythm once the tachycardia has been broken. A few have had repeated attacks, and several cases requiring continued digitalization over several months have been reported.

There are certain cases where digitalis has been ineffective or where the situation is so desperate that digitalis works too slowly. Gibson and Philipshorn employed acetylcholine intravenously in two cases with dramatic effect. Acetylcholine is a powerful parasympathetic-mimetic drug and must be used with caution. A syringe loaded with atropine sulfate should be immediately available as an antidote. The use of mecholyl (acetylbetaethamethylcholine) has been advocated in the treatment of supraventricular tachycardia. It is probably not as safe a drug as acetylcholine, since the latter is destroyed in the body by choline esterase approximately 200 times more rapidly than mecholyl. Furthermore, it is said that the side reactions from the use of acetylcholine are fewer and less severe than those seen with mecholyl.

REPORT OF A CASE

I wish to present the case report of an infant who responded to the use of acetylcholine intravenously after digitalis was ineffective.

C. S. was a one-month old white male infant who was well until two days prior to admission when the mother noted that the baby did not eat well and finally refused all feedings. One day prior to admission the baby became short of breath, breathed rapidly, and slept most of the time. At 4 a.m. on August 8, 1948 he became blue and developed grunting respirations. He was admitted to Allen-town Hospital at that time.

The birth history was uneventful. The baby weighed 7 lb. 8 oz. at delivery and he had done well until August 6, two days prior to his admission. The family history was non-contributory.

Physical examination on admission revealed a well nourished, well developed white male infant who was critically ill. Weight on admission was 9 lb. 5 oz. The skin was cyanotic and mottled. The respirations were grunting and rapid. The body was cold and clammy, and the skin had a doughy feel. The eyes were glazed. The pupils were dilated and reacted sluggishly to light. The chest was clear and resonant to percussion. The breath sounds were normal and no rales were heard. The heart sounds were of fair quality and the rate was
estimated to be above 240 per minute. The heart was not enlarged to percussion. No murmurs were heard. The liver was three inches below the costal margin in the midclavicular line and was quite firm. A diagnosis of paroxysmal tachycardia was made and confirmed by electrocardiogram. The EKG showed no significant abnormalities other than the paroxysmal nodal tachycardia with a rate of 224 per minute.

The child was placed in an oxygen tent and 50 milligrams of digalen was given immediately by intramuscular injection. Thereafter 25 mgm. was given intramuscularly every four hours for a total dosage of .15 Gm. of digalen. Sixteen hours after admission the color was considerably improved, but the heart rate continued between 200-240 per minute. Prostigmine Sulfate, O.2.c.c. of a 1:2000 solution, was given with little effect other than causing the child to cry out, become flushed, and draw up his legs as though he were in abdominal pain. The liver had decreased slightly in size.

Twenty-two hours after admission the child became much worse. The liver extended almost to the pelvic brim and he vomited coffee ground vomitus several times. Because of the baby’s desperate condition it was decided to give the baby intravenous acetylcholine via a cut-down over the internal saphenous vein. During the period of injection an observer listened to the heart. One milligram of acetylcholine was administered slowly. The heart, which had been beating at least 240 per minute, stopped completely and then gradually resumed beating with a rate of 120-130 per minute. A few extra systoles were heard. The child’s color improved almost immediately, but the abdomen remained distended. A catheter was passed into the stomach with difficulty. Much air and about two ounces of coffee ground material were aspirated. Within 10 to 15 minutes the liver receded to 1 1/2 inches below the costal margin. The respirations were still irregular, but the cardiac rate remained about 150 per minute.

Thirty-six hours after treatment, the child’s condition was good. The liver had receded to 3/4 inch below the costal margin. A chest X-ray showed a normal cardiac silhouette. A repeat electrocardiogram showed a normal rate of 120 per minute. The child was discharged on his sixth hospital day in good condition. He has had no recurrences since that time, and his development has been normal.

SUMMARY

A case of supraventricular paroxysmal tachycardia was successfully treated by the intravenous administration of acetylcholine.
INTRODUCTION

Proctology has been a distinct surgical specialty since 1877 when Dr. Joseph MacDowell Matthews adopted this field of surgery. He is known, rightfully, as the father of proctology because he was the first orthodox physician in the world to engage in this work.

Proctosigmoidoscopic examinations on patients, however, should not be limited to proctologists. Every physician and surgeon concerned with rectal and colonic disease should be able to conduct such examinations successfully, because prompt diagnosis is important, and sometimes life-saving, to the patient.

Although crude anal specula have been unearthed at Pompeii, satisfactory sigmoidoscopic instruments are of comparatively recent origin. Throughout the years both the instruments for making examinations and the position of the patient to facilitate them have been improved.

It is almost as important to know that disease of the anus, rectum and colon is not present as it is to know that it is. All of us have seen patients who received treatment for non-existing organic lesions.

HISTORY

The importance of a good medical history needs no emphasis. This has been expounded by authors and teachers to such an extent that reference to it is made with reluctance. Many believe that this phase of the investigation of disease is over-emphasized. This, in the instance of proctology, is true. It is far more important and infinitely more accurate to "look and see". This statement becomes more apparent when we realize that 72 per cent of colonic lesions can be observed through the proctoscope.

INSPECTION

Careful inspection of the skin of the perianal, sacral, perineal and gluteal regions is indicated. Skin lesions common to other parts of the body are frequently observed. Evidence of trauma, discharging sinuses and masses may be observed. Insignificant sacro-coccygeal dimples are frequently seen. These do not indicate evidence of pilonidal
disease. An elevated hyperemic area over the sacrum, however, may indicate pilonidal disease, usually in the form of an abscess. A discharging sinus in this region, containing a tuft of hair is more positive evidence of pilonidal disease. An elevated area containing several discharging sinuses may indicate a carbuncle. Recently a female patient was referred to the hospital with a tentative diagnosis of ischi-anal abscess. The final diagnosis was carbuncle with an associated diabetes mellitus. Thrombosed edematous external hemorrhoids are diagnosed by inspection and must be differentiated from the comparatively rare melano-epithelioma. These tumors are painless and increase in size gradually in contradistinction to hemorrhoids.

Lichenification and thickening of the perianal skin may indicate anal pruritus. In this condition the skin may be excoriated, weeping, fissured, dry or hyperemic depending upon the stage of the disease or the character of the treatment. If the patient has received X-ray therapy the skin may be unusually thick and devoid of hair. Idiopathic pruritus ani is differentiated from psoriasis in that the latter disease is present in other parts of the body surface; from drug dermatitis (notably sulfa and aureomycin) for similar reason and the history; from acanthosis nigricans because of the increase in pigmentation, verrucous and papillomatous character of the lesions and the frequent association of abdominal malignancy; from fungus infection by the microscopic examination, and from leukokeratosis and leukoplakia by the location of the lesion and distinct appearance.

Actinomycosis, sarcoma, carcinoma, tuberculosis and osteomyelitic sinuses may be considered as the perianal area is inspected but further tests are indicated and definite conclusions cannot be reached by mere inspection of the lesions.

Mucosa, internal hemorrhoids, enlarged anal papillae and pedunculated tumors may be seen protruding from the anus.

Moist foul smelling pinkish or greyish cauliflower warty growths in the perianal region may indicate condyloma acuminatum. These simple warts must be distinguished from the warts caused by syphilis. The latter are usually larger and rarely become pedunculated.

Chancres, chancroids, granuloma inguinale and lymphopathia venereum may present nonspecific ulceration and must be differentiated by means other than mere inspection.

Gonorrhea of the anorectal area presents, usually, no other external sign than a purulent discharge. Diagnosis requires careful bacteriological examination, including culture.
The character of the anal orifice should be observed. A spastic anus may mean that the patient is suffering from a painful affliction of the anorectal area such as fissure, cryptitis, ulcer, or abscess. Just as frequently does it indicate tenseness for fear. Patulous anus may indicate recent or old injury to the sphincter, organic disease of the rectum or sigmoid colon and lesions of the spinal cord. This condition was observed recently at our hospital in a case of incomplete bowel obstruction caused by an aneurysm of the lower portion of the abdominal aorta. Some patients have an unusual ability to relax the sphincter muscles and of course in this group patulous anus has no serious significance. The condition of patulous anus was observed in a female child, age 5, who had undergone an operation for repair of spina bifida.

Inspection is important in the diagnosis of the various types of rectal prolapse. It should be remembered that a prolapse of the sigmoid colon does not always present external evidence.

The value of external examination is emphasized by an experience with a patient, age 74, who had a high grade prolapsing cancer of the rectum. The lesion was replaced and because it assumed a position in the hollow of the sacrum, was not observed at subsequent proctosigmoidoscopic examination. Only at operation was the true condition diagnosed. Extirpation with colostomy was performed instead of hemorrhoidectomy as planned. It would be well to call the lowermost posterior portion of the rectum the "blind spot" since this is the portion of the rectum which is most difficult to visualize through the proctoscope.

PALPATION

Gentle palpation is the most popular method of physical diagnosis used by surgeons. Palpation of the perianal area with one or more fingers as the condition requires is an aid in determining the consistency and position of masses, fistulous tracts and other infectious processes. Frequently it is possible to palpate subcutaneous cord-like tracts to the rectum, pilonidal area or, in the case of male patients, to the urethra. The value of information gleaned in this fashion is obvious.

Palpation of the anal canal may reveal the presence or absence of tone in the sphincter muscle, the presence of enlarged anal papillae, cryptitis, internal openings of fistulae, abscesses and both inflammatory and malignant ulcers.

The rectum and its adjacent structures may easily be palpated with the examining finger. In many instances even the rectosigmoid
may be explored, especially if the patient is asked to strain. Extrarectal masses may frequently be detected by this simple diagnostic maneuver. Retrorectal, supralever and pelvirectal abscesses may be found by careful digital examination. Thrombotic internal hemorrhoids may be palpated. The stony hard sensation produced by malignancy may easily be distinguished from the tense character of abscesses or the soft polypoid nature of benign or low grade non-ulcerative lesions. Edema and roughness of the mucosa as observed in cases of inflammatory conditions of the bowel may be detected. Strictures, impactions and foreign bodies may be diagnosed.

Among the extra rectal masses which may be detected, are the following: prostatic abscesses and tumors, enlargement of the seminal vesicles, Blumer's shelf, pelvic tumors, sacro-coccygeal tumors and cysts, rope-like loops of bowel as seen in cases of ileitis, fecaliths in proximal portions of the bowel and aneurysms.

Within the past several months a young woman was admitted to our hospital with the history of numerous bouts of intestinal obstruction and several laparotomies for "adhesions". Sigmoidoscopic examination revealed a large rope-like extra-rectal mass in the cul-de-sac. This proved to be an extensive regional enteritis. Resection with immediate anastomosis was performed.

The diagnosis of leukemia cannot be made by a digital examination of the rectum. However, the presence of multiple, unusually painful thrombotic hemorrhoids should be an indication for general physical examination of the patient. During the past year a young male patient was referred to our hospital for the treatment of thrombotic hemorrhoids. The two offending hemorrhoids were removed but after two days the patient developed several more thrombotic hemorrhoids. General examination revealed the presence of leukemia.

ENDOSCOPY

Direct examination of the potential or actual cavity produced by the anal canal, rectum and lower sigmoid colon is known as proctosigmoidoscopy. It is the most valuable single diagnostic aid in the field of proctology.

One of three positions for the patient is usually utilized for examination: the inverted position, knee-chest position or left lateral position. It is advantageous to be able to employ any one of them as the condition of the patient requires. In all instances the patient should be relaxed.
and cooperative. The patient should have been prepared previously with plain water enemas.

Visualization for a distance of at least 24 centimeters with the aid of a well lighted scope should be considered an average examination. Instruments for making higher examinations have been devised, but such examination is seldom necessary and but occasionally possible because unusual cooperation on the part of the patient is required.

The mucosa is carefully examined as the instrument is advanced. This procedure is essential not only from a diagnostic standpoint but it serves as a precaution against perforating the bowel. A diseased bowel is more easily injured than is a normal one. Certain pathologic states of the bowel will frequently prevent introduction of the instrument the usual distance. It is therefore important to anticipate the variations in diameter of the proximal portions of the bowel.

Normal mucosa is pink and glossy. It may be either dry or moist. The web-like pattern created by the narrow blood vessels is easily recognized. The vessels are usually a deep shade of pink in the case of the arteries and bluish in the case of veins. It is not unusual to see large submucosal vessels in the lower part of the rectum. The appearance of the lining of the bowel is not unlike the mucosa of the buccal surface of the cheek. Normal mucosa appears as a thin membrane. It is transparent.

Mucosal and submucosal hemorrhages appear as red blotches of free blood when observed shortly after their occurrence. These areas appear as brown spots when seen a week or more after bleeding has occurred. These ecchymotic areas are flat and devoid of the fullness associated with true hemangiomas. The latter may not have a typical proctoscopic appearance. In a case reported by Buie, the only proctoscopic finding was "mucosal and submucosal thickening."

The mucosa is pale in anemic patients. Other changes in color of the blood may be observed sigmoidoscopically but usually the pathologic processes producing such changes are more easily diagnosed by means other than proctologic examination.

Melanosis coli, a condition which follows the prolonged use of cascara, presents itself as diffuse superficial deposits of brown pigment in the mucosa which otherwise appears normal. Frequently when such a condition is present, the examiner is tempted to increase the intensity of the light attached to the instrument. Apparently the pigment interferes with the usual reflection of the light.
Prominent lymph follicles in the mucosa appear as countless tiny glistening elevations not unlike white sand. They are usually of no pathologic significance.

Normally the anus and rectum are well supported although any one portion can be moved within a restricted area. The sigmoid on the other hand is movable in all directions except in those cases in which the mesosigmoid is short and then movement anteriorly is restricted and induces the complaint of pain.

It is not unusual to see the shadows of loops of small bowel through the transparent sigmoid colon. Movements of the sigmoid synchronous with the pulsation of the large pelvic arteries are frequently seen.

Malignancy of the lower bowel is of great importance from the standpoint of early diagnosis. Adenocarcinoma is the most frequently encountered malignant growth of the bowel. It may be polypoid or ulcerating. It is a pink cauliflower-like lesion which bleeds and may involve part or the entire circumference of the bowel. Size is not always an indication of the grade of malignancy. The lesion may produce excessive amounts of mucus. Ulceration and invasion cause various degrees of fixation. The crater of the ulcer, with its thick hard grayish base may be observed. Frequently the mucosa surrounding the elevated border has a frosted or fish skin appearance and it is not unusual to find a small sentinel polyp at some distance below the malignant lesion. Inflammatory change in the mucosa immediately surrounding the lesion is common. Tumors that arise low in the rectum have a tendency to invade the anal canal and frequently differentiation from a primary tumor in this area must be made.

Although adenocarcinoma of the rectum has a typical appearance it is well to do a biopsy.

The diagnosis of sarcoma, melano-epithelioma and epithelioma is almost impossible by proctoscopic examination alone.

In the presence of atypical ulceration in the rectum or anus, biopsy is essential. Occasionally the presence of submucosal nodules may suggest the presence of early sarcoma or carcinoid tumor but laboratory methods are necessary for final diagnosis. Sarcoma and carcinoids are considered comparatively rare tumors in this region. Carcinoids have been known to metastasize and for that reason they are included in this group of malignant tumors.

Benign tumors as found during proctologic investigation include polyps, lipomas, oleomas and fecaliths.
Polyps may occur singly or as multiple polyposis. They may be sessile or pedunculated. They may undergo ulceration, necrosis and malignant degeneration. Sessile polyps are regarded with greater concern than are those having a pedicle.

All polyps should be regarded as potentially malignant.

Polyps as well as some other lesions may cause intussusception. The pedunculated ones may occasionally remain unobserved by virtue of their ability to move to different levels depending on the position of the patient and the peristaltic movements of the intestines. A child, age 4, was examined at our hospital because of the presence of rectal bleeding. The first sigmoidoscopic examination for a distance of 24 centimeters revealed normal mucosa. A subsequent examination revealed a rather large pedunculated polyp at 10 centimeters. As the instrument was introduced the polyp advanced ahead of the scope and the pedicle was discovered at about 25 centimeters.

Occasionally a bleeding pedicle is found in the bowel, the end result of a pedunculated polyp which was torn from its attachment by the fecal current.

Lipomas, oleomas and fecaliths appear as submucosal or subcutaneous tumors and must be removed if definite diagnosis is to be made.

The various inflammatory processes of the terminal bowel present the greatest difficulty in diagnosis.

In amebiasis it is usually possible to see an oval or round ulcer with a bleeding base and hyperemic elevated border. Normal mucosa appears between the ulcers. However, the findings are not always typical and in extreme cases the bowel presents only a sloughing necrotic mass. It must be remembered that Balantidium Coli infestation may present a similar picture.

Bacillary dysentery is a common disease. It varies from a mild disturbance to a disease of great severity. Blood and mucus may be absent in the mild forms. Perforation and necrosis of the intestine, although rare, may occur. In children the disease may cause rectal prolapse. Typically, a three-stage progression—seen on first, second and third days, respectively—is noted. Punctate follicular hyperplasia occurs first, then follows punctate follicular necrosis and finally discrete and confluent ulceration.

Bacillary dysentery, obviously, must be differentiated from appendicitis, pneumonia, meningitis, amebiasis, typhoid, salmonella and staphlococcal food infections and focal non-specific enterocolitis.
The term “toxic diarrhea” is rather loosely used to describe a condition resulting from some such indirect cause as peritonitis. Proctosigmoidoscopic examination on a patient in the terminal stages of peritonitis, recently revealed foul fecal discharges, a dilated bowel and mild diffuse hyperemia of the mucosa. Although there are no typical proctologic changes, one can certainly differentiate this condition from intrinsic specific bowel disease.

Actinomycosis presents no typical proctoscopic appearance. The chronic draining sinuses occur from deep abscesses. Proctitis occurs first, then woody infiltration and peri-rectal stenosis, then abscesses and fistulae and stage of complications such as deeper abscesses, septicemia and abscesses of the liver and other parts of the body.

Thrombo-ulcerative colitis, considered streptococcal in origin by Bargen comprises about 67 per cent of cases of chronic ulcerative colitis. There are two phases—active and period of remission. The active phase is divided into three stages. It is the exception rather than the rule to be able to make the diagnosis during the first stage. This is primarily due to the fact that the patient only rarely presents himself for diagnosis at this stage. During the first stage the mucosa is hyperemic and bleeds easily. Edema, characterized by a peculiar bogginess of the mucosa and myriads of greyish-yellow spots, one to two millimeters in diameter are added during the second stage. The third stage presents the typical raw granular diffuse moth-eaten appearance. At this stage there are many pock-like ulcers which result from the miliary abscesses as they rupture through the mucosa. The entire surface of the mucosa is involved. It has the appearance of pink sandpaper. The valves of Houston are no longer sharply outlined.

The period of remission is the result of the previous acute phase. Part or all of the mucosa is destroyed and replaced with fibrous tissue. It is now evident that the disease involves all of the coats of the bowel. The colon has contracted. In fact all that may remain is a bleeding ulcerated tube. Secondary ulceration, pseudopolyposis and even malignant change may be observed. The only remaining characteristic pathologic changes of the original disease are the pock-like scars. These are the foot prints of previous activity. They are pathognomonic and must be sought—frequently with the aid of a magnifying attachment to the scope.

Regional or segmental ulcerative colitis (type 2) must be diagnosed by X-ray as the portion of bowel seen through the proctoscope is normal.
There is a form of chronic ulcerative colitis (type 3) which has an atypical proctoscopic appearance. It involves the entire colon and is considered by many physicians to be not dissimilar to the usual type. Disseminated “punched out” ulcers with normal mucosa between them are observed. It is in these cases that tuberculosis and amebiasis must be ruled out before making a diagnosis of “chronic ulcerative colitis”.

The colitis associated with allergy and vitamin deficiency presents no characteristic sigmoidoscopic picture.

Cases of agranulocytic angina may show an ulcerative rectal process and the possibility should be kept in mind.

Tuberculosis of the colon, although it mimics amebiasis, has no typical sigmoidoscopic appearance.

The irritation set up by parasites may imitate an inflammatory reaction in the colon which may well resemble a true colitis.

Factitial proctitis, a justifiable lesion resulting from irradiation of a lesion in the pelvis, should be recognized. The lesion occurs, usually, on the anterior wall of the rectum. The picture varies from telangiectasis over an edematous mucosa which bleeds on contact to an ulcerating mass involving all of the coats of the bowel which frequently causes a recto-vaginal fistula. The typical appearance, however, is that of a flat irregular ulcer with a greyish-green base. The mucosa surrounding the ulcer shows telangiectasis and edema. The lesion heals slowly and leaves a yellowish scar with telangiectasis. Lesions at the recto-sigmoid may cause obstruction.

SUMMARY

The chief diagnostic signs of the usual diseases encountered in proctologic practice which are recognizable by proctosigmoidoscopic examination, have been described. Effort has been made to present the diagnostic features in a practical and useful fashion.