winter time may be necessary. If scleroderma is present and Raynaud's disease has anteceded the scleroderma, sympathectomy should be performed.

Sympathectomy, including the first to the fifth thoracic segments, benefits scleroderma of the yoke, face and mouth.

If scleroderma of the esophagus is present, causing dysphagia and proved by barium swallow, complete bilateral thoracic sympathectomy, preserving the splanchnic connections on one side to avoid postural hypotension, is sometimes beneficial.

We submit most of our patients with Buerger's disease and arteriosclerosis of the lower extremities to sympathectomy, believing it to be the most efficacious palliative measure at our disposal. However, in the older age group with arteriosclerotic endarteritis obliterans who still have good collateral circulation and whose limbs we judge will stay viable for their life expectancy, we do not advocate sympathectomy. The operation should be sold on the basis of life insurance for the limb and without promising to relieve entirely the intermittent claudication. This limitation is often a disappointment to the patient who comes to us for relief of his inability to walk far, but the operation usually triples or quadruples his exercise tolerance. In a patient with Buerger's disease we must also guard ourselves in not promising relief from recurrent superficial phlebitis migrans. These attacks we now treat by ambulatory anticoagulation therapy.

Sympathectomy has been effective in relieving 76% of 29 patients with reflex sympathetic dystrophy.

References

The Early Diagnosis of Bronchogenic Carcinoma.
Reliable evidence indicates that bronchogenic carcinoma is increasing in incidence; in males it is exceeded in frequency only by cancer of the stomach. Since a clear-cut symptom complex is rarely present, one must constantly suspect carcinoma in the presence of any pulmonary symptoms or signs of doubtful etiology. Once a reasonable suspicion of lung carcinoma exists, the investigator should pursue a vigorous investigative course, using all available facilities necessary to establish a conclusive diagnosis.—Reitz, H. E., U.S. Nav. M. Bull., 48: 198, 1948.

Scurvy—Recent Experiences

N. Barrie Coward, M.D.
Halifax, N.S.

The subject of this paper may cause some bewilderment because scurvy has for a long time been considered an extinct disease, and also because of a veiled suggestion that there may be some new line of therapy. The reason, however, for presenting this subject is the relatively large number of cases of scurvy which we have seen at the Children's Hospital, Halifax, in recent years, and particularly during the past twelve months. There is no new form of therapy in the treatment of scurvy, and indeed none is needed. The disquieting fact is that the disease should be prevalent at all, when it is one which can be so easily prevented.

A review of our cases of advanced clinical scurvy since June 1, 1945, shows:

Table I.

<table>
<thead>
<tr>
<th>Year</th>
<th>Cases</th>
</tr>
</thead>
<tbody>
<tr>
<td>1945</td>
<td>3</td>
</tr>
<tr>
<td>1946</td>
<td>2</td>
</tr>
<tr>
<td>1947</td>
<td>11</td>
</tr>
<tr>
<td>1948</td>
<td>2</td>
</tr>
<tr>
<td>1949</td>
<td>14</td>
</tr>
<tr>
<td>1950</td>
<td>5</td>
</tr>
</tbody>
</table>

No accountable reason has been found for the relatively large number of cases in 1947 and 1949.

There is no specific seasonal arrangement generally given for scurvy, and in our cases they were scattered over the year with the greater number occurring between the months of June and December.

Table II.

<table>
<thead>
<tr>
<th></th>
<th>1945</th>
<th>1946</th>
<th>1947</th>
<th>1948</th>
<th>1949</th>
<th>1950</th>
</tr>
</thead>
<tbody>
<tr>
<td>January</td>
<td>1</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>February</td>
<td>1</td>
<td>2</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>March</td>
<td>1</td>
<td></td>
<td>2</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>April</td>
<td>1</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>May</td>
<td>1</td>
<td>1</td>
<td>1</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>June</td>
<td></td>
<td></td>
<td>1</td>
<td>5</td>
<td></td>
<td></td>
</tr>
<tr>
<td>July</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td>2</td>
</tr>
<tr>
<td>August</td>
<td>1</td>
<td>1</td>
<td>1</td>
<td>2</td>
<td></td>
<td></td>
</tr>
<tr>
<td>September</td>
<td>3</td>
<td></td>
<td>2</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>October</td>
<td>1</td>
<td>2</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>November</td>
<td>1</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td>2</td>
</tr>
<tr>
<td>December</td>
<td>1</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td>2</td>
</tr>
</tbody>
</table>

The average length of time for scurvy to develop following cessation of vitamin C is 6 to 10 months. A review of the age group shows

*Presented at the 81st Annual Meeting of the Canadian Medical Association, sub-section of Pediatrics.
89% of the cases occurring within the latter half of the first year of life.

There is good evidence that moderate deficiencies of vitamin C impair health without giving rise to the classical signs which result in a diagnosis of scurvy.

The fully developed picture of scurvy is generally readily and easily recognized. The early symptoms, however, do vary considerably. This, together with the fact that the weight of a scurbutic infant is often high, due to the presence of oedema, may lead one to a false appreciation of the nutritional and physical state, and so delay diagnosis in the earlier stages.

The symptoms first noted by the parent and those observed prior to admission to hospital are divided as follows:

**Table IV.**

<table>
<thead>
<tr>
<th>First symptoms</th>
<th>Later symptoms</th>
</tr>
</thead>
<tbody>
<tr>
<td>Pain, lower limbs only</td>
<td>18</td>
</tr>
<tr>
<td>upper limbs only</td>
<td>1</td>
</tr>
<tr>
<td>body as a whole</td>
<td>6</td>
</tr>
<tr>
<td>Irritability</td>
<td>17</td>
</tr>
<tr>
<td>Anorexia</td>
<td>6</td>
</tr>
<tr>
<td>Swelling of extremities noted</td>
<td>2</td>
</tr>
<tr>
<td>Swelling elsewhere noted</td>
<td>3</td>
</tr>
<tr>
<td>Bleeding gums</td>
<td>1</td>
</tr>
<tr>
<td>Vomiting</td>
<td>3</td>
</tr>
<tr>
<td>Increased perspiration</td>
<td>2</td>
</tr>
<tr>
<td>Rash on skin</td>
<td>1</td>
</tr>
<tr>
<td>Loss of weight</td>
<td>1</td>
</tr>
<tr>
<td>Sleeplessness</td>
<td>0</td>
</tr>
<tr>
<td>Diarrhoea</td>
<td>0</td>
</tr>
<tr>
<td>Fever</td>
<td>0</td>
</tr>
<tr>
<td>Haematuria</td>
<td>0</td>
</tr>
<tr>
<td>Earache</td>
<td>0</td>
</tr>
</tbody>
</table>

Whereas these may be the more common symptoms observed by parents, there are other signs of impairment to health which are present at the same time, such as decreased capacity to form and maintain a normal collagen level, slow healing of wounds, decreased capillary strength, decreased capacity to combat infections and a decreased capacity to metabolize the amino acids, tyrosine and phenylalanine.

The complaint of earache is not one often associated with scurvy except as part of a co-existing upper respiratory infection. One writer has drawn attention, however, to the relationship of scurvy to grippe or influenzal otitis and myringitis bullosa hemorrhagica. Although all the reported cases were children and adults, there is no reason to assume that they could not occur in infants.

The diagnosis in each case was made purely on clinical grounds and x-ray. No vitamin C estimations of the blood were done. X-ray evidence was present in the long bones in all cases. These included some or all of the following: cortical atrophy, ground glass atrophy of the ends of the shaft, infarctions of the epiphysis, periosteal elevation and the typical white line. Many also showed evidence of swelling of the soft tissues of the lower extremities and, later, evidence of subperiosteal hemorrhage.

Much has been written on anaemia in scurvy and all our cases showed varying degrees of this condition. The haemoglobin ranged from 22 to 77% and the red count from 2 to 3.5 million. Except for one case, which was a macrocytic hyperchromic anaemia, all were essentially normocytic normochromic in type. The white count varied between 6,000 and 26,000. This wide range is undoubtedly due to the presence of secondary infection, and 12, or 32.5%, of the cases showed some additional infection of one type or another on admission to hospital.

Administration of vitamin C in the form of orange juice showed:

**Table V.**

<table>
<thead>
<tr>
<th>A. Orange juice given</th>
<th>Supposedly satisfactory amounts</th>
<th>Insufficient amounts (1 tsp. daily)</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>9</td>
<td>2</td>
</tr>
<tr>
<td>B. No orange juice given</td>
<td>Never given</td>
<td>14</td>
</tr>
<tr>
<td></td>
<td>Vomited the orange juice</td>
<td>2</td>
</tr>
<tr>
<td></td>
<td>Baby did not like orange juice</td>
<td>1</td>
</tr>
<tr>
<td></td>
<td>Caused diarrhoea</td>
<td>1</td>
</tr>
<tr>
<td></td>
<td>Told not to because of laxative effect</td>
<td>1</td>
</tr>
<tr>
<td></td>
<td>Had been getting orange juice but stopped because mother thought not necessary</td>
<td>1</td>
</tr>
<tr>
<td>C. Orange juice given on occasion only</td>
<td>1</td>
<td></td>
</tr>
<tr>
<td>D. No information</td>
<td>5</td>
<td></td>
</tr>
</tbody>
</table>

The majority of the nine cases who developed scurvy in spite of getting a supposedly sufficient supply of orange juice had been warming or heating the juice before giving it to the baby. This fact is often not brought out except by careful questioning of the family and, there-
fore, may as a result lead to a delay in diagnosis. In this regard there must exist a definite misunderstanding concerning the stability of vitamin C. As is well known, oxidation of vitamin C goes on as soon as an orange is cut but the process is accelerated by heat, light and alkalinity, and, if copper is present, the process goes on still more rapidly.

The group which demands attention is the one where orange juice was either never given or stopped early because of some associated factors such as vomiting, dislike by the baby, and so on. In spite of these occurrences it is difficult to understand why, with all that is written today in the popular journals, such an indifference to the use of vitamin C should still persist. It may be that while most parents are familiar with the idea of giving orange juice, many still do not understand why it is important to do so. In any event, it is more likely to be a lack of appreciation of the need of vitamin C than inaccessibility to the means of prevention.

In many cases it happens that parents are simply told on one occasion to give orange juice to the baby, but because of one or more of the above-mentioned associated factors, it is discontinued. On subsequent visits to the physician there exists the assumption that the infant is getting some form of vitamin C, but no further check is made to ensure that this is the case. This state of affairs may go on for many months, and because there are no abnormal signs or symptoms both parents and physicians are lulled into a false sense of security owing to the long latent period which exists between the beginning of the deficiency in the diet and the onset of symptoms.

With this in mind, and remembering that even moderate deficiencies impair health, the physician should, therefore, as a routine procedure, always question the parent regarding the intake of vitamin C. His instructions in this regard should be implicit, and should stress the reasons for the giving of orange juice.

Because most cases of deficient vitamin intake are found in the poorer economic group, the question of meeting the infants’ needs with fresh orange juice becomes a factor. Also, as these cases are found entirely among the artificially fed and essentially in urban areas, being almost unknown in the breast fed and rare in rural districts, if there is any question of the infant not taking or tolerating orange juice, or if there is any doubt about the co-operation of the parent, or if economic reasons are a factor, it would be advisable for the physician to use the easily procurable and well tolerated ascorbic acid tablets.

On the basis of published evidence, the Food and Nutrition Board of the National Research Council (USA) recommends the following intake:

<table>
<thead>
<tr>
<th>Table VI.</th>
</tr>
</thead>
<tbody>
<tr>
<td>Children under 1 year</td>
</tr>
<tr>
<td>“ 1 to 3 years ”</td>
</tr>
<tr>
<td>“ 4 to 6 “</td>
</tr>
<tr>
<td>“ 7 to 9 “</td>
</tr>
<tr>
<td>“ 10 to 12 “</td>
</tr>
<tr>
<td>As fresh orange juice contains approximately 0.5 mgm. per c.c., the infant’s requirements can only be met by giving at least two ounces of orange juice daily, which may indeed prove a burden to certain economic groups.</td>
</tr>
<tr>
<td>Again, one should always bear in mind that if clinical scurvy exists, there are many more cases of latent scurvy which remain unrecognized. Published reports indicate that this applies not only to the infant but to the older age group as well.</td>
</tr>
<tr>
<td>One might naturally expect that the older infant and child, with their broader, even though deficient, diets, would be more prone to suffer from latent scurvy with its various ills and substandard nutritional states. There is always the possibility that some of these might go on at any moment to show the picture of classical scurvy if any infection should develop. The probable reason that more of them do not is because the physician’s routine instructions to force fruit juices in the presence of an infection prevents the development of manifest scurvy and unwittingly improves the deficiency. A temporary stay in the course of the disease may thus be obtained but unless the physician bears the condition in mind and permanently corrects the deficiency, the impairment to health continues.</td>
</tr>
<tr>
<td>Much can be done in this respect by improving the balance of the diet and the use of selected foods even in the poorer economic groups. It is true that fresh cow’s milk will normally prevent scurvy and this is one reason why it is seldom found in rural areas. Pasteurization, on the other hand, renders it useless in this respect. However, the use of fresh un-</td>
</tr>
</tbody>
</table>
pasteurized milk as an antiscorbutic food is open to question because of the possibility that such milk may cause other infections.

We may therefore conclude that the maintenance of an adequate vitamin C intake is the responsibility of all those interested in the growth and development of children, and that the question of a reliable intake in all age groups and economic brackets is one of practical concern.

**BIBLIOGRAPHY**

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5. BRENNMANN, J.: Practice of Pediatrics, Hagerstown, Prior, 1933.

**RÉSUMÉ**

L'auteur présente 37 cas de scorbut constatés depuis 1945. Malgré les progrès dans l'alimentation des enfants le scorbut n'est pas encore une maladie entièrement disparue. Tous les cas ont été diagnostiqués cliniquement et radiologiquement, sans dosage de la vitamine C. Les signes les plus fréquents furent, des douleurs dans les jambes, de l'irritabilité et de l'oedème. Très peu souvent on trouva des génives hémorragiques. L'anémie fut constatée dans la plupart des cas. L'auteur explique ces déficiences en vitamine C par les faits suivants. Ou bien les enfants reçoivent une quantité suffisante de jus d'orange mais ce dernier est chauffé avant l'administration, ou bien ils s'agit de négligence de la part du médecin ou des parents. L'auteur suggère qu'un questionnaire de routine soit toujours fait par le médecin traitant pour s'assurer que l'enfant reçoit une quantité suffisante de vitamine C synthétique ou sous forme de jus d'orange.

**YVES PRIVOST**

**CLINICAL USE OF THE OXIMETER**

Arnold L. Johnson, M.D., O. R. Stephen, M.D.

Paul Sekelj, Ph.D.E.

Montreal, Que.

The degree of oxygen saturation of the peripheral arterial blood is a matter of fundamental importance in health and disease. Clinically, the only definite means of determining that there is oxygen unsaturation of the arterial blood has been the sign of cyanosis. This, unfortunately, is a very crude indication of anoxemia, for it is well established that there must be a considerable degree of unsaturation before the amount of reduced hemoglobin is sufficient to result in a blue colour.1, 2 In a subject with a normal hemoglobin, the oxygen saturation of the arterial blood must be reduced from the average normal of approximately 98% to between 65 and 70%. At this level approximately 10 grams of hemoglobin in each 100 c.c. of blood will bear oxygen and 5 grams will be reduced hemoglobin. These calculations serve to indicate the marked reduction in oxygen saturation which must occur before the classical clinical sign of anoxemia, cyanosis, becomes evident.

The desirability of having a more subtle means of determining the lesser degrees of oxygen unsaturation is apparent. This may be done by arterial puncture with subsequent analysis of the blood by the technique of Van Slyke and Neill.3 This procedure involves discomfort, it is time-consuming, and it is not applicable to routine clinical practice. The efforts of the past few years in devising a clinically applicable method to determine oxygen saturation have resulted in extensive studies in oximetry, a term which is applied to the study of oxygen saturation of circulating blood by photoelectric techniques.

The manner in which an oximeter is employed is as follows. An earpiece is attached to the pinna of the ear. It has been established that when the ear is flushed with heat, the blood contained within it has an oxygen content practically equivalent to that in the peripheral arteries. On one side of the ear is an electric bulb from which light of known intensity is emitted. The heat of this lamp causes the necessary vasodilatation. This illumination passes through the substance of the pinna to fall upon two filters—an infra-red filter and a red filter. Light which has been transmitted through the ear and through these filters then falls upon two light-sensitive photoelectric selenium cells. The potential developed by these cells is impressed upon a sensitive galvanometer and recorded as a spot of light illuminating a scale, from which calculations may be determined to ascertain the percentage saturation.

It is apparent that the pinna varies in its characteristics from one individual to another. Ears differ as to thickness, pigmentation and concentration of the hemoglobin contained within their blood vessels. Various means are employed to account for these differences, the necessary deductions being obtained partly from changes in the incident light which has passed through the infra-red filter. Wood4 employs marked pressure to compress the ear, rendering it bloodless, and taking measure-