# Evidence for intestinal toxemia— An inescapable clinical phenomenon

Alan Immerman, BS Lombard, Illinois



Alan Immerman

A diet high in protein causes predominance in the Intestine of proteolytic putrefactive bacteria which produce highly toxic compounds, some of which are absorbed. These compounds are incompletely detoxified by the liver, and therefore enter the systemic circulation. The toxins cause or aggravate many disease states. Alleviation of pathology can be accomplished by use of such nontraumatic measures as exercise, fasting, and proper diet. Intoxication arising from the intestine is a common occurrence and, if eliminated, health status may be expected to improve. Many aspects of the concept of intestinal toxemia are discussed.

# Introduction

This paper will focus on the concept of intoxication of intestinal origin. The subject is of wide-ranging clinical importance and should be emphasized. As will be made clear, intestinal toxemia is frequently found as either a basic cause of or contributing factor to many clinical phenomena. In 1933, Dr Anthony Bassler, a professor of Gastroenterology at Fordham University Medical College and New York Polyclinic Medical College, and consulting gastroenterologist of Christs, Polyclinic and Peoples Hospitals in New York, stated, after a 25-year study of over 5000 cases, that: "Every physician should realize that the intestinal toxemias are the most important primary and contributing causes of many disorders and diseases of the human body.''<sup>1</sup> Dr H.H. Boeker, in 1928, went so far as to say, "it is now universally conceded that autointoxication is the underlying cause of an exceptionally large group of symptom complexes."<sup>2</sup>

Intestinal toxemia is a process resulting from a certain type of diet or from intestinal obstruction. Various toxic chemicals are produced in the lumen by

Alan Immerman is presently enrolled in the National College of Chiropractic in Lombard, Illinois, where he is an honor student. He graduated in 1977 from Northern Arizona University, Magna Cum Laude with a BS in Chemistry, and was elected to the Phi Kappa Phi Honor Society. After graduation from chiropractic college in 1980, Immerman plans to practice in Phoenix, Arizona. His current address is Box 138, 200 E. Roosevelt Road, Lombard, Illinois 60148. bacteria. These toxins are absorbed into the bloodstream either as a result of a pathological or a nonpathological state of the mucosa. Some of the toxins escape the detoxifying action of the liver because of pathological, functional (cannot act upon all of the toxin present), or physiological (normally does not act upon this toxin) liver insufficiency. These chemical poisons then enter the general circulation and exert deleterious effects before being excreted by the kidneys. The result of the intoxication is to produce a pathological change in the tissues, or to aggravate a previously existing condition. Each of these steps will be considered.

A thorough review of the literature was undertaken in the preparation of this paper. **Index Medicus,** from the years 1879 to 1978, was checked under multiple listings; many medical texts were perused to find leads to articles; the **Citation Index** was also checked. The result of this search was to find that almost no clinically-oriented articles have been written in the English language about intestinal toxemia since the late 1950s. In fact, few papers have been published since 1940 which directly address this subject. It is for this reason that most of the clinically-oriented papers reviewed in this paper are dated from before 1940.

The reader may wonder why the theory of intestinal toxemia has not been discussed in recent times, and is not well known today. The fact is that a change in opinion over the years, not based on any new scientific research, has led to the abandoning of this idea by the medical profession. It is important to emphasize that this has not resulted from new scientific research proving the error of the theory of intestinal toxemia. It is well known that certain ideas fall into and out of favor as the years pass, regardless of their validity, which has been the present case. An analogy may be drawn from the theory of chiropractic which, despite its great clinical usefulness, has been repeatedly "discovered" then abandoned or forgotten since ancient times; D.D. Palmer was the most recent individual to revive this old healing practice and bring it to our attention.

Some of the articles cited in this paper were written over 50 years ago. Quite naturally, a question may arise in the reader's mind as to whether or not such research could be reliable, having been done so many years ago. The answer to this is unequivocally affirmative. Portions of this paper as well as a great wealth of scientific information available today were uncovered many years ago, and are still valid. Modern textbooks in microbiology and biochemistry discuss many of the facts presented in this paper as true information; one text in each area is cited herein to demonstrate this point. Although scientific technology was not as sophisticated in the 1920s, the scientific intellect was equally keen; many important discoveries were made and theories confirmed. It is most important to emphasize that older research and

clinical observations are not necessarily invalid because of age.

The writer is well aware of the fact that current opinion in the medical world does not agree with the conclusions reached in this paper. However, the theory of intestinal toxemia as presented is scientifically and clinically sound; this ultimately is the main concern of an objective scientist. Opinion should be based on solid scientific research, and it is the purpose of this paper to aid in the formation of a scientifically-based opinion.

#### **Effect of Diet**

The experiments of Herter and Kendall<sup>3</sup>, performed in 1909 at the Rockefeller Institute for Medical Research, were among the first to prove a definite connection between the nature of the diet and the type of bacterial flora found in the intestine. In experiments on cats (chosen because they are carnivores) and monkeys (chosen because of their biological similarity to man), it was proven that the intake of a high protein diet resulted in dominance of a strongly proteolyzing putrefactive type of flora (note: the term putrefaction refers to "decomposition of proteins by anaerobic organisms"4); also that conversion to a high carbohydrate/low protein diet resulted in dominance of a non-putrefactive type of flora. Fecal samples were cultured after a change in diet, and concentrations of endproducts of bacterial metabolism were measured in the urine to determine the type and character of the flora. The change in flora resulting from the change in diet was the same regardless of the animal type. The products of the putrefactive flora included indole and skatole (from tryptophan), phenol (from tyrosine), and hydrogen sulfide from the products of protein breakdown. Of importance to health care professionals is the following observation seen after a change in diet from high protein to high carbohydrate: "Clinically, the most striking feature of the change in diet [in monkeys] is an improvement in spirits and activity which may safely be construed as showing a markedly improved sense of bodily and psychological well being."<sup>5</sup> The results of these experiments were confirmed by other investigators of that same time period. <sup>6,7,8,9</sup> Many recent experiments have shown the presence of products in the urine of the putrefactive flora following ingestion of a high protein diet.<sup>141,142,143,144,145,146,147,148</sup> Other experiments have shown that ingestion of fermentable carbohydrates such as glucose, fructose or lactose results in delay of or complete inhibition of the putrefactive process.<sup>141,148,149</sup> Inhibition of the putrefactive process is reflected by decreased urinary output of putrefactive products.

# **Intestinal Obstruction**

Obstruction in the intestine causes toxemia only in a small percentage of cases. However, it is far more

likely to be rapidly fatal than toxemia resulting from diet. The main reason for considering obstruction is derived from the insight gained into the study of intestinal toxemia.

Obstruction in laboratory animals is produced by the surgical formation of a closed intestinal loop. The loop is washed to exclude the secretions of the stomach, liver, and pancreas, along with the products of food digestion. The result has been the same in all experiments; the bacteria multiply greatly, the proteolytic bacteria overgrow all others and produce toxic chemicals, the toxins are absorbed, and the animals become sick and die.<sup>10,11,12,13,14,150</sup> The toxins produced include histamine,<sup>15</sup> which is normally present but may be in a greater concentration, and various protein decomposition products.<sup>16</sup>

The toxins produced in the closed intestinal loop have been removed and injected into healthy animals with a reaction "more intense but similar to that developing in a closed-loop" animal.<sup>17,18</sup> Of importance is the fact that injection into the portal vein "gives a reaction similar to intravenous injection"<sup>19</sup> indicating that "the liver plays no essential role as a protective agent against this poison."<sup>20</sup>

These toxins have been produced by putrefactive bacteria which are normally present but have multiplied greatly and overgrown all other bacteria in the obstructed intestine. Therefore, it is within the realm of hypothesis that some amount of toxin is produced and absorbed in the case of intestinal stasis with a high intake of protein but without total obstruction.

It is important to emphasize again that intestinal obstruction similar to that observed in animals with surgical closure of an intestinal loop is rarely seen in man; such complete obstruction is certainly not the basic cause of intestinal toxemia, in most cases. However, insight into the process of intestinal toxemia probably can be gained by the consideration of such an extreme case; therefore, this consideration has been included. When researchers feed lab animals ten times the amount of saccharine a human being would ever consume in the same time period, identical logic is used. This logic is accepted by the scientific community. It is well recognized that much information can be gained from experiments with animals, using extreme conditions.

# Nature and Action of Chemicals Produced by Proteolytic Bacteria

To date, the exact nature of all such chemicals has never been completely identified. Unfortunately, along with the development of instrumentation sophisticated enough to yield a complete answer to this question, there has been a simultaneous loss of interest in the subject of intestinal toxemia. However, much information is available from various research studies.

The reader should bear in mind a basic tenet of modern pathology which is too often forgotten today.

The ACA Journal of Chiropractic/April 1979

This tenet states that inflammation is a response of the body to tissue injury, and that this response serves to protect the body from the injurious agent.<sup>151</sup> Many times it is thought that the basic problem in a tissue involves inflammation, as if such a state existed in a vacuum. That would not be possible. Inflammation is the response of the body to local injury and attention should be paid to removal of the injurious agent, a truly protective response, rather than to suppressing the inflammation.

Some ammonia is formed by bacteria in the intestine, mainly from urea and digestive products of proteins<sup>21</sup>; ammonia is also formed, as is well known, by the liver and kidneys. In liver disease such as cirrhosis, or in disease of portal circulation, "abnormal elevations in the level of ammonia in peripheral blood may occur and these are accompanied by corresponding but lesser elevations of ammonia in the cerebrospinal fluid."<sup>22</sup> Many studies show that this increased concentration of ammonia causes severe neurological symptoms resembling hepatic coma such as mental disturbances, characteristic tremor, and altered EEG pattern.<sup>23,24,25,26</sup> A low protein diet minimized these symptoms.<sup>23,24,26</sup>

This reflects modern medical opinion as well. Harrison's Principles of Internal Medicine states that "both hepatic coma and the chronic form of hepatocerebral disease are characterized by hyperammonemia, which is probably important in their pathogenesis. Ammonium is derived from the bacterial action on intestinal proteins and normally is converted to urea in the liver.<sup>152</sup> Confusion, drowsiness, or other signs of impending hepatic coma should be treated by prompt decrease in protein intake to levels of 20 to 30 grams daily or less."<sup>153</sup> In regards to treatment for hepatic coma: "Reducing the protein intake, cleansing the colon of blood, suppressing the bacterial action on protein in the intestinal tract with neomycin or kanamycin, and administering the acidifying agent lactulose, all of which lower the NH<sub>1</sub> levels in the blood, have been found to restore many of these patients to a relatively normal state." And, "Although the biochemical mechanism is not fully understood, the most plausible hypothesis is that the levels of blood NH<sub>3</sub> are elevated because the diseased or bypassed liver fails to convert it to urea; the high serum NH<sub>3</sub> causes elevated NH<sub>3</sub> levels in the brain which interfere with its metabolism in some obscure way."<sup>154</sup> Ammonia may even be involved with the malignant transformation of cells.<sup>27</sup>

Clostridium perfringen enterotoxin has also been found.<sup>29</sup> This is well known to be highly poisonous.<sup>29</sup>

Indole is formed from tryptophan by proteolytic bacteria,<sup>30</sup> and is known to be toxic from results of experiments on animals,<sup>31</sup> and man.<sup>32</sup> It is also known that certain metabolites of tryptophan can cause bladder tumors.<sup>33,34</sup> With normal liver function, most, if not all indole is detoxified by the process of conjugation. In the case of sickness, this may or may not occur. A high protein, low carbohydrate diet results in increased excretion of conjugated indole, called indican,<sup>7,35,141,146,147,148</sup> as compared with a low protein, high carbohydrate diet. The amount of indican in the urine has been widely used as a measure of intestinal putrefaction.

However, indican determination used by itself is not reliable. For diagnostic purposes, it is interesting to know that combined determination of phenol and indican in the urine has proved to be highly valuable in the detection of the stagnant loop syndrome.<sup>145</sup> Stagnant loop syndrome is defined as bacterial overgrowth in the small intestine, vitamin  $B_{12}$ malabsorption, and steatorrhea, all of which improved by treatment with antibiotics.<sup>155</sup> This syndrome is believed to be present if the levels in the urine of both phenol and indican are abnormally elevated. Tabaqchali cautions, however, that since his experiment utilized only 51 patients, this observation of diagnostic reliability needs confirmation in a larger series.<sup>145</sup>

Phenol (carbolic acid) is formed from tyrosine in the process of putrefaction.<sup>30,142,143,156</sup> It is so extremely poisonous that it is used as an antimicrobial agent. It is both a local corrosive and systemic poison which can cause necrosis of gastrointestinal mucosa, renal and hepatic cells.<sup>36</sup> Phenol is absorbed into the body, and most of it is excreted in a free form — not conjugated and, therefore, not detoxified.<sup>37,38</sup> As is the case with indole, the concentration of phenol in the urine is increased with a high protein diet.<sup>39,40,157,158</sup>

Skatole is formed from bacterial action on tryptophan.<sup>30,159</sup> It is regarded as ''a toxic substance causing depression of the circulation and of the central nervous system,"<sup>32,41</sup> and is found in increased concentration in the intestine following a high protein intake.<sup>42</sup> Skatole, "when in excess in the circulating blood through failure of conjugation by the liver," imparts a foul odor to the breath.<sup>43</sup> Skatole and indole are partially responsible for the characteristic odor of feces.<sup>44</sup> Skatole antagonizes acetylcholine and potassium<sup>160</sup> in smooth muscle preparations. Pseudomonas migula has recently been identified as a bacteria capable of converting tryptophan to skatole.<sup>161</sup> Following formation, skatole is converted into 6-hydroxyskatole by the cells of the gut wall, and possibly by other host tissues.<sup>162</sup> 6-hydroxyskatole damages lipid-absorbing cells, the hemoglobin within red blood cells, and hemoglobin found free within the body.<sup>162</sup> This destructive action probably accounts for the presence of metabolites of skatole in the urine of many patients suffering from the malabsorption syndrome and certain anemias.<sup>162,164</sup>

Hydrogen sulfide gas is another byproduct of protein decomposition.<sup>43</sup> In comparable concentrations it is "as toxic as cyanide and interferes with the cytochrome system."<sup>45</sup> It is obvious that this gas can irritate the mucosa; this brings about congestion and makes the mucosa more permeable to intestinal contents because of the presence of gas in solution. It may be responsible for "neurocirculatory myasthenic symptoms, for poisoning by this gas will cause: weakness, nausea, clammy skin, rapid pulse and cyanosis."<sup>43</sup>

Neurine is formed from sphingomyelin by anaerobic bacteria. This compound is toxic to animals.<sup>46,47</sup>

Aminoethyl mercaptan is formed from bacterial decomposition of the amino acid cysteine. It has a "profound hypotensive effect."<sup>48</sup>

Putrescine and cadavarine, formed from putrefaction of tryptophan, can lower blood pressure. Tryptamine, from the same source, raises blood pressure after an initial depression.<sup>49</sup>

Histamine is the last important decomposition product of tryptophan that is well known.<sup>15,49,50</sup> Poisoning with this compound can produce "headache, head congestion, nervous depression, cardiac arrythemia, fall of blood pressure, nausea, and collapse."<sup>32</sup>

Tyramine is a putrefactive product of tyrosine. It is structurally similar to epinephrine and can raise the blood pressure.<sup>49,50</sup>

It should be mentioned that some current writers hypothesize that putrefactive endproducts "irritate the receptor nerve-endings, thereby bombarding the associated spinal cord segments with afferent impulses, which result in muscular response and subluxations in the lower thoracic and upper lumbar areas, as well as predisposing to sacroiliac subluxation." Furthermore, "noxious materials in the bloodstream circulate to the central nervous system and . . . increase its irritability to afferent stimuli leading to increased hypertonicity of muscles and greater likelihood of subluxation."<sup>51</sup> We, therefore, may not have only the circulatory but also the nervous system mediating the effects of toxins to all the cells of the body.

Many other chemicals, too numerous to consider in detail, have been found to be formed in the intestine from bacterial putrefaction. These include guanidine,<sup>52,53</sup> and many others.<sup>54,55</sup> Besides the known substances, many chemicals of unknown character may be produced. Some of these chemicals may be toxic to a degree; of the toxins, some may be completely detoxified by the liver but others not at all. Other substances may be only partially rendered harmless. As is obvious, the subject of the chemistry of intestinal toxemia is highly complex and only partially understood. However, as will be seen, the situation from a clinical perspective is much clearer.

# **Intestinal Stasis**

Delayed intestinal motility of the small and large intestines will now be discussed. Sir W. Arbuthnot Lane, MS, was one of the most knowledgeable men to work in this field. Being a surgeon, and having a

Vol 13, S-28

penchant for surgical treatment of intestinal toxemia, he observed first hand the pathological state of the intestines of many sick people. His therapy, as might be suspected, was to remove the diseased section of the bowel; this was successful in providing temporary relief to the patient. However, as will be seen later, other treatments exist which are as successful but are non-traumatic and do not involve the use of drugs or surgery.

Lane defined chronic intestinal stasis as "an abnormal delay in the transmission of the intestinal contents through some portion or portions of the gastro-intestinal tract, which delay may be accompanied by constipation or by a daily or even more frequent action of the bowels."56 He further states that any such delay facilitates multiplication of organisms and the subsequent development of toxemia in the bloodstream. This leads to "progressive degenerative" changes in every tissue and a very definite and unmistakable series of symptoms."56 It was mentioned in the discussion on intestinal obstruction that bacteria multiply greatly, with the proteolytic-putrefactive bacteria overgrowing all others. Is it not reasonable to consider that some of the features of chronic intestinal stasis are of the same kind but not as severe as those encountered in total obstruction?

Other physicians concur that delay (stasis) may be accompanied by one or more movements of the bowels per day.<sup>57,58,59</sup> This is of great clinical importance. It removes the foundation to the widely held belief that daily movements of the bowel are conducive to health.

Concerning clinical evidence, the following is worthy of mention. In a clinical study of 50 cases of intestinal toxemia, Dr H.J. Bartle found constipation in 72%, and delayed intestinal motility in 60%.<sup>60</sup> Dr Satterlee found constipation in 84% of his cases of intestinal toxemia<sup>61</sup>; in 1916, this doctor was attending physician to Fordham Hospital, Chief of Clinic for Gastroenterological Diseases, University and Bellevue Hospital Medical College, New York.

Consideration of the chemical aspect of intestinal stasis is also worthy of note. In this regard, Drs Underhill and Simpson of the department of Experimental Medicine, Yale University School of Medicine, state the following: "the effect of even mild constipation overshadows the effect of diet on the excretion of phenol and indican. Constipation causes a large increase in the excretion of these substances."<sup>62</sup>

In conclusion, it may be said that many physicians believed that delayed motility provides the foundation for building the condition of intestinal toxemia.<sup>7,63,64,65,66,67</sup>

#### Distension

Distension of the intestine has been attributed by some to be the cause of the symptoms of intestinal toxemia.<sup>68</sup> In experiments performed by Dr Alvarez of

The ACA Journal of Chiropractic/April 1979

the Mayo Clinic, it was shown that pressure upon the rectum by sterile gauze could produce symptoms of intoxication.<sup>69</sup> There is no doubt that the effect of mechanical distension of the intestine is irritation to the nervous system, which is of no small importance. However, the presented evidence shows that mechanical irritation is indeed accompanied by chemical irritation. "Because it has been shown that constipation headache can occur in persons whose spinal cords have been cut, we know that this headache is not caused by nervous impulses from the colon. Therefore, it possibly results from absorbed toxic products or from changes in the circulatory system."<sup>70</sup>

Distension in obstruction of the intestine results in "more or less splanchnic congestion"<sup>71</sup> resulting in "interference with its [the mucosa] normal circulation."<sup>72</sup> This may result in functional or anatomic injury to the mucosa, allowing absorption of toxins to take place faster than the liver can detoxify them. Distension could lead to a "hemorrhagic and frequently necrotic condition of mucosa with a consequently hastened absorption of poisonous substances."<sup>73</sup>

As can be clearly seen, distension is a condition which the clinician will wish to reduce. Treatment will be discussed later.

#### Absorption

Absorption of toxins may occur through an intact gastrointestinal mucosa.74,141 For example, a recent study has shown that indole and skatole can be rapidly absorbed at any level of the small or large intestine. In this experiment, medical students with normal health and patients in the hospital with no symptoms of gastrointestinal tract disease were used as subjects.<sup>141</sup> Many other writers feel that an inflammation of the mucosa is necessary before absorption will occur. Such inflammations of the rectosigmoid colon are described as "common."75 Of 50 cases of intestinal toxemia examined by another physician, 80% had lower colon inflammation, and 62% had inflammation of the duodenum.<sup>76</sup> Others have reported general inflammation of the intestinal wall.<sup>77,78</sup> Inflammation of the terminal ileum or appendix will "elicit intense enterointestinal reflexes resulting in severe inhibition of gastrointestinal motility; as a result, functional obstruction often occurs in the small bowel."79 Of interest is a description of the changes in the colon wall observed during the autopsy of insane patients residing at New Jersey State Hospital. Seen were large areas of destruction of mucosa plus atony and atrophy of smooth muscle.80

It is well known that mucosal injury may result in death from septicemia. Here it is stated that mucosal inflammation alters permeability, thereby allowing absorption of bacterial toxins which cause pathological changes in tissues. It is obvious that in septicemia, the bacteria produce metabolic toxins throughout the body. Many scientists believe these toxins are the cause of death in this type of situation.

# Detoxification

Detoxification is primarily the responsibility of the liver. Though this subject has already been covered to a certain extent, a few additional remarks are necessary.

Detoxification is primarily the responsibility of the liver because the blood flowing from the stomach and intestines is "not returned directly from these organs to the heart, but is conveyed by the portal vein to the liver."<sup>81</sup> Once the portal vein has transported the blood to the liver, "this vein divides like an artery and ultimately ends in capillary-like vessels [sinusoids]."<sup>81</sup> From these sinusoids, substances absorbed from the intestines become exposed to liver parenchymal cells. These cells, hepatocytes, act upon such substances in many ways.

Ideally, hepatocytes will detoxify any poisons present and thereby protect the body. The important point here, however, is that toxins upon which the liver can act and detoxify, may be present in too great a quantity for complete liver action. As Dr Bartle says, the liver must be compared to other organs; "they can do just so much work and no more. In the case of the liver it is always carrying an unusual burden in chronic intoxication states, and naturally the breaking point must now and then be reached. This comes with an acute outburst of intoxicating symptoms. . . . "<sup>82</sup> With mucosal injury especially, "absorption of toxic substances may take place faster than the liver can detoxicate them and toxemia and death ensue."73 Many authors concur in this viewpoint.<sup>56,77</sup> Of course, there are poisons against which "liver plays no essential role as a protective agent."71 Phenol and skatole are examples of poisons which for the most part, escape liver action, 141, 142, 143, 144, 145, 163

#### Symptoms

Information on symptoms arising primarily from intestinal toxemia could fill volumes. Not only have books been written,<sup>84</sup> but literally thousands of articles in many languages have been published in the scientific literature on this topic. Obviously, an exhaustive discussion here is an impossibility.

No claim should be made that every symptom described in the following paragraphs arises from intestinal toxemia. However, by examination of the following, one must note that such toxemia is at the root of more problems than commonly imagined; it is at the root of many conditions more often than might be suspected.

Generally, intestinal toxemia manifests as one or more of the following; fatigue, nervousness, gastrointestinal conditions, impaired nutrition, skin manifestations, endocrine disturbances, neurocirculatory abnormalities and headaches.<sup>43</sup> Arthritis, sciatica and low back pain; allergy, asthma; eye, ear, nose and throat disease; cardiac irregularities; pathological changes in the breasts; all these conditions have responded to therapy directed at a toxemic state in the intestine, and the evidence for this follows. You will note that therapy is described only generally as being aimed at relieving the toxemic state of the intestine. To be more specific in each instance is impossible in this paper. Treatments mentioned will be representative of that which has been used by many doctors, not including surgery and drugs.

A. Allergy: Dr William Lintz, MD, successfully treated 472 patients suffering from "gastrointestinal allergy, a condition of hypersensitivity of the digestive tract," by eliminating the following allergens from the gastrointestinal tract: "bacteria and their toxins, foods and their split products."<sup>85</sup> The most frequent symptoms were endocrine gland disturbances of many types such as hypothyroidism, pituitary malfunction, etc; heart disease; hypertension, and many others.<sup>86</sup>

ŧ

ï

B. Asthma: Dr Allan Eustis, MD, instructor at Tulane University of Medicine in 1912, reports that 121 cases of bronchial asthma were relieved by eliminating the intestinal toxemia universally present.<sup>87</sup> Dr D. Rochester, MD, of the University of Buffalo School of Medicine in 1906, states that it is his conclusion, after 23 years of observation, that toxemia of gastrointestinal tract origin is the underlying cause of asthma. He says, "I believe the results of treatment justify my position."<sup>88</sup>

C. Arthritis: Dr Anthony Bassler treated 44 arthritic patients by relieving intestinal toxemia, and observed "marked improvement in 21, moderate in 19, none in four." With the addition of physical therapy, all but nine cases showed more marked improvement.<sup>89</sup> Twenty years later, Dr Bassler said a similar handling of 300 additional cases "has not modified my opinion a particle," 90 Sir W. Arbuthnot Lane, MS, FRCS (surgeon), believed that arthritis could not develop in the absence of intestinal toxemia, and says there is clinical and x-ray evidence of stasis in such patients. Furthermore, he states that, "the symptoms disappear and the patients recover sometimes with startling rapidity when the condition of stasis has been effectually dealt with."<sup>91</sup> Others confirm the connection between intestinal toxemia and arthritis.<sup>92</sup>

D. Cardiac arrhythmias: Guyton<sup>93</sup> states that "toxic conditions of the heart" can cause arrhythmias. Dr Bassler reports 100% success in eliminating such heart irregularities in 43 patients treated by reducing intestinal toxemia.<sup>94</sup> Dr Bainbridge, MD, stated that "intestinal toxemia is common among the causative factors of so-called functional heart disease.<sup>95</sup> Dr D.J. Barry in 1916, professor of Physiology, Queens College, Cork, England, stated that: "There seems little doubt that substances having a deleterious action on the heart musculature and nerves are formed both in the small and large intes-

tine, even under apparently normal circumstances."%

Toxemia is further implicated in high blood pressure. Dr Hovell states that "toxemia due to intestinal sepsis is a common cause of increased blood pressure."<sup>97</sup>

E. Ear, nose, and throat problems: The experience of three doctors is neatly summed up by Dr J.A. Stucky, MD.<sup>98</sup> "In several hundreds of cases of diseases of the nasal accessory sinuses, middle and internal ear, ... I have found unmistakable and marked evidence of toxemia of intestinal origin as evidenced by excessive indican in the urine, and when the condition causing this was removed there was marked amelioration or entire relief of the disease."<sup>99,100</sup>

F. Eclampsia: R.C. Brown, MB, MS, FRCS, an obstetrical surgeon in England in 1930, linked intestinal toxemia and eclampsia.

G. Eye problems: Dr C.W. Hawley, MD, treated many cases of eye strain and disease with success once again by relieving intestinal toxemia.<sup>101</sup>

H. Thyroid gland disease: Dr W.S. Reveno, MD, theoretically links exophthalmic goiter to "a toxic process in the intestinal tract."<sup>102</sup> He cites animal studies as evidence. Echoing this view is Dr A. Eustis, MD, who cites case histories of patients who found complete relief from exophthalmic goiter as a result of eliminating intestinal toxemia.<sup>103</sup> Sir W.A. Lane reports a connection between intestinal toxemia and "several changes in the thyroid" such as "adenomatous growths."<sup>104</sup>

I. Nervous system: Many disorders of the nervous system are involved. Dr Carl Von Noorden, MD, professor of the First Medical Clinic, Vienna, Austria, in 1913, described a condition of diffuse sensory polyneuritis with pronounced vagal irritation, treated with positive results by relieving intestinal toxemia. He and his assistant extracted a "poisonous substance from the feces, which in animal experiments produced quite similar symptoms."<sup>105</sup> This substance was found to be formed by a "bacterium of the paratyphus group."<sup>106</sup>

Dr C.A. Herter, MD, in 1892, lecturer on Anatomy and Pathology of the Nervous System, New York Polyclinic, linked intestinal putrefaction to epilepsy in 31 patients. He based this on a successful treatment using drugs to control bacterial activity of the intestine.<sup>107</sup> Agreeing with his conclusion are other doctors.<sup>108,109</sup>

Drs Satterlee and Eldridge, in a paper read to the annual session of the American Medical Association in 1917, reported experience with 518 cases of "mental symptoms" including "mental sluggishness, dullness and stupidity; loss of concentration and/or memory; mental incoordination, irritability, lack of confidence, excessive and useless worry, exaggerated introspection, hypochondriasis and phobias, depression and melancholy, obsessions and delusions, hallucinations, suicidal tendencies, delirium, and stupor."<sup>110</sup> Their success in eliminating these symptoms by surgically relieving intestinal toxemia is truly remarkable in the light of today's commonlyheld beliefs. In the discussion following the presentation of the doctors' paper, other physicians stated that they shared in this experience.<sup>111</sup>

Other authors describe the same pattern, adding headache<sup>66,91,109,112</sup> to the list of symptoms. It would indeed appear true that "the nervous system is almost invariably affected in whole or in part by chronic intestinal toxemia" and that "the nervous symptoms are often the most prominent in the symptomatology."<sup>111</sup>

Of considerable interest is a recent paper entitled, "Biochemical Aspects of Indole Metabolism in Normal and Schizophrenic Subjects" by Herbert Sprince.<sup>163</sup> In this highly sophisticated paper, 11 independent laboratories are noted to have found at least five times more 6-hydroxyskatole in the urine of schizophrenics than in that of normal subjects. Such universal agreement, Sprince says, is highly significant since this is an area where conflict, not agreement, is the rule.<sup>163</sup> It will be remembered that 6-hydroxyskatole arises primarily from skatole in the intestine and that skatole arises from the action of putrefactive bacteria on tryptophan, an essential amino acid.

J. Senility: The following should be mentioned: "Auto-intoxication from intestinal stasis or from constipation due to lessened peristaltic activity, undoubtedly plays a contributing part in the process of senescence."<sup>113</sup>

K. Low back pain and sciatica: Dr R.B. Osgood, MD, cites six cases of patients suffering from these conditions. A few had also received chiropractic manipulative care with no improvement. In these cases, the cause was a toxemic state of the intestine; the pain completely left upon elimination of the toxemia, and returned upon a return of toxemia because of dietary errors. His references point to other physicians who had the same experience.<sup>114</sup>

In this regard, Dr Von Noorden found "pains especially frequent which corresponded to the ordinary sciatic or intercostal neuralgia."<sup>115</sup> The reader would be well advised to review the facts on irritation to the nerves of the low back area during a toxemic state of the intestine.

L. Dermatoses: Dr Hans J. Schwartz, MD, in a statistical analysis of 900 patients suffering from acne, eczema, and many other skin conditions, concluded that "intestinal toxemia is an important etiologic factor in the production of many dermatoses, especially those of the inflammatory type."<sup>116</sup> Dr J.F. Burgess, MB, lecturer in Dermatology, McGill University, associate dermatologist, Montreal General Hospital, reports the results of studying 109 cases of eczema. He states that "on the basis of clinical ob-

servations and sensitivity tests against various amino acids and ptomaine bases, eczema is probably caused by intestinal toxemia."<sup>117</sup> Others concur.<sup>118,119</sup>

M. Breast pathology: Changes in the breast resulting from intestinal toxemia have been described by many doctors. The views of three can be summarized as such<sup>120</sup>: 'The breasts undergo degenerative changes, manifested in the first instance as induration, to be followed by inflammatory and cystic degeneration, and possibly, lastly, by cancerous infection.'<sup>95,121</sup>

N. Cancer: There is no claim that intestinal toxemia is the cause of cancer. However, some physicians believe that "even the beginning of malignant disease of various organs comes within the wide range of intestinal stasis."<sup>92</sup> Sir W.A. Lane recorded his feeling of being "exceedingly impressed by the sequence of cancer and intestinal stasis."<sup>122</sup>

In light of the clonal selection theory of chemical carcinogenesis which states that "all chemical carcinogens will display . . . a greater toxicity for normal tissue cells than for the cells of a tumor derived therefrom by treatment with that carcinogen,"<sup>123</sup> it can be speculated that toxins of intestinal origin may initiate malignant transformations. The malignant cell would be more resistant to the toxic effects than would a normal cell, according to this theory.

#### Treatment

In the treatment of intestinal toxemia various measures have been used. These include surgical removal of inflamed, ulcerated, or infected intestine (sometimes including the entire colon); autogenous vaccines to kill the bacteria responsible for the production of the toxins; colonics and/or laxatives for obvious reasons; diet therapy, and exercise. Different doctors have used various combinations of these therapeutic measures; some have put great emphasis on surgery, while others none at all. Many have put emphasis on the use of vaccines to some degree. Some recommend colonics and/or laxatives; others literally condemn their use. Almost all have used some type of diet therapy, and exercise has often been recommended.

The author's conception of an ideal therapy will now be described. It is an amalgam of the ideas of the many clinicians cited in this paper, but with the addition of a few basic holistic concepts in the light of the axiom, "Physician, do no harm."

First, if vaccines and surgery can be avoided by the use of other less traumatic and less side-effect-producing therapies which are at least equally efficacious, then vaccines and surgery should not be used. This does not mean that drug and surgical therapies are never needed in the treatment of intestinal toxemia; however, these therapies are almost never needed.

Second, any therapeutic measure which provides Vol 13, S-32 only the relief of symptoms is inferior to a measure that removes the cause of the symptoms; symptomatic relief is only temporary, whereas, the removal of the cause leads to permanent relief. These concepts are probably self-evident and surely constitute common sense. Surely no chiropractic physician would argue with this general overview of treatment.

Since a majority of physicians have found success in the use of diet therapy and exercise, these measures will be emphasized. Diet therapy is non-traumatic, does not produce side-effects, and removes the cause of the toxemia. It allows for a complete cure to take place which lasts as long as the diet is followed. Dr Satterlee stated that "diet and proper emptying of the bowels has always been the recognized treatment."<sup>124</sup>

Treatment can be summarized by the following: "Patients must be taught how to eat, how to live, how to work and how to play. It must be impressed on them that health will not return through the simple act alone of taking medicine from a bottle. If they can be made to see that a general cleaning-up process is to be inaugurated, and that it is their duty to keep things cleaned up as treatment progresses, and after it is ended, and that the whole management of their trouble is really founded on very ordinary principles, then, with great interest, they usually cooperate quite heartily with their physician."<sup>125</sup>

If the reader will refer to the effect of diet on putrefaction, he will see that animal experiments indicate the benefit of a low protein, high carbohydrate diet. This concept is universally agreed upon by the physicians noted in this paper. How much protein should be consumed? The controversy over this issue is great. However, since "protein in an average food intake of 2,500 calories is about 94 grams per day"<sup>126</sup> and the recommended daily allowance is 50-60 grams for adults, it is obvious that an immediate decrease in intake by almost 50% can be safely accomplished. In light of studies on minimum protein requirements, we may even consider as sufficient intakes of 15-25 grams per day of high quality protein.<sup>127</sup> Obviously, with this intake of protein, other food components are to be relied upon to supply energy needs.

The remainder of the diet should contain a minimal amount of fat, with carbohydrates supplying the bulk of the energy need. It has been shown that fats, especially heated fats, intensify the process of intestinal toxemia.<sup>128</sup> The dietary carbohydrates should not be the refined or starchy form. The more easily digested and more highly nutritious types are preferred. Therefore, after the intestine has healed, fruits and vegetables should be used to provide the greatest amount of the carbohydrate intake, and thus the caloric requirement. These foods have the additional benefit of being rich in the vitamins and minerals.

Intake of certain specific items should be either greatly reduced or eliminated. These include sugar,<sup>129</sup> as implied above; salt, alcohol, condiments,

tobacco<sup>130</sup>; tea, coffee, pastry, fried foods, and "any article of food which is known to disagree."<sup>88</sup>

Eating the correct amount of food is an essential feature of the proper diet. Dr Stucky, MD, says that "all forms of food, when eaten in greater quantities than the digestive fluids can digest, are capable of forming putrefactive poisons, which are deleterious to the human organism. I have, therefore, insisted that all my patients should eat small meals in which the protein foods should form a very small proportion."<sup>131</sup> Dr Rochester, MD, says "the amount of food should be limited to the minimum compatible with maintenance of health and weight."<sup>88</sup> Echoing this is Dr Synnott: "Patients do better and feel better when the caloric intake in their diet is curtailed."

In the acute stage, no food at all is recommended. "Feeding bulky foods causes further distension of the colon, an added burden is put upon its weakened walls, the damaged bowel musculature is taxed beyond the power to respond, the delayed motility and stasis are increased, the gastroptosis and enteroptosis are aggravated, the expected increase in peristalsis does not occur, but on the contrary there is more pronounced paresis of the already atonic, overworked and disabled bowel."<sup>132</sup> Dr Bartle concurs in this view.<sup>130</sup>

Instead of feeding, fasting is recommended. "Fasting is a perfectly sound method of diminishing toxins if combined with copious draughts of water."<sup>133</sup> Fasting has been shown to reduce the phenols to a low level.<sup>134</sup> It has also been shown to increase by one to two days to over two weeks the life span in dogs with duodenal obstruction; the dogs were forced to fast for four days prior to surgical creation of the obstruction.135 In a 76 kilogram man, "intestinal putrefaction as measured by the output of urinary indican was markedly decreased during the fasting interval" of seven days.<sup>136</sup> Fasting will rest the overworked intestine and allow the physiological process of inflammation to proceed onward to the process of repair. One need not worry about starvation in these cases, since the patients already are suffering from excess food intake.

The administration of Lactobacillus acidophilus culture and lactose is said by some to be of great value; but others claim that they are useless. It is interesting to note that the entire basis for the idea that yogurt and its bacteria are beneficial to health is based on the theory of intestinal toxemia. The idea is that the "good bacteria" (Lactobacillus) will displace the "bad," namely the putrefactive proteolytic type. This therapy seems to have some value, at least in a percentage of cases. However, in light of its having dubious value in some doctors' opinion, this therapy should not be relied upon.

The topic of colonic irrigations is surrounded by much argument and controversy. There are some who claim that colonics have great therapeutic value, <sup>137,138</sup> and as might be suspected, others avoid

its use and decry its value.<sup>139</sup> Dr Von Noorden states that such treatments "only hide the pathologic condition of the intestine. Instead of helping, they retard the definite cure. They are justified and advantageous only in acute disease."<sup>105</sup> Dr Bassler says that colonics "relieve the toxemia by clearing the large bowel for a few days but do nothing to control the bacteriology or the chemistry... since the locality for most of the toxic process is in the small intestine. Kept up long enough, they do more harm than good."<sup>140</sup>

Surrounding the subject of exercise there is no controversy, just ignorance or advocacy. Of those in favor, Dr Bassler presents an average view. "Physical exercise, especially in the open, is valuable to keep back the assault of toxins on body tissues. This is accomplished by increasing the conjugating ability by calling forth higher degrees of protoplasmic activity, higher oxidation and increased circulation."<sup>57</sup> But the clinician should be wary, since increased circulation may carry toxins to previously unexposed tissues, thereby multiplying the symptoms. As is usual in the health care profession, prudent clinical judgment is in order.

If the outlined program is followed, both the doctor and patient will be satisfied with the results.

#### Discussion

Toxemia and subsequent pathology may result from absorption of certain chemicals formed by bacterial action on amino acids. Pathology is classically defined as abnormal function. The sources mentioned in the section on symptoms reveal that there is a clinical, clearly observable relationship between intestinal toxemia and abnormal cellular function. These studies report thousands of cases of people suffering from various ills who became well after clearing up their intestinal toxemia. For example, eczema is a pathological change; it healed time and again when the source of irritation, intestinal toxemia, was removed. The author is aware that this is a controversial idea, today. But, nonetheless, it is certainly conceivable that, for example, the following sequence could occur: high protein diet results in phenol (corrosive poison) production; phenol enters circulation, causing local pathology. Evidence has been presented that a high protein diet causes proliferation of proteolytic bacteria; it is well known that such proteolytic bacteria produce phenol from the amino acid tyrosine, and that phenol can kill cells; papers have been reviewed that prove that the majority of absorbed phenol is not conjugated by the liver, and therefore not detoxified; and many papers have been reviewed which relate pathology to the presence of intestinal toxemia in a statistically significant way.

For example, when previously discussing dermatoses: 1017 patients were examined and treated. In a clinical discussion, it is highly relevant and important that many sick people become well when their intestinal toxemia is cleared up. On the other hand, medical opinion states that the etiology of such diseases as Crohn's, Whipple's, ulcerative colitis, and many others, is unknown (idiopathic). But the irritation that causes the primary inflammation in these afflictions must come from somewhere, or the entire science of pathology is in error. It has been proposed in this paper that the products of intestinal putrefaction are a significant source of such irritation.

# **Summary and Conclusions**

The following have been stated:

- 1) Change in diet induces change in intestinal flora. With a high protein diet, proteolytic putrefactive bacteria predominate.
- 2) Such bacteria produce highly toxic compounds, some of which are absorbed.
- 3) The liver does not detoxify all of these toxins; thus, some escape into the general circulation and can be found in the urine.
- 4) These toxins produce a wide range of symptoms and aggravate preexisting pathologies.
- 5) The problems resulting from such an intoxication may be eliminated in almost all cases by a judicious combination of fasting, exercise, and proper diet consisting of small amounts of protein and fat with enough carbohydrates to supply the caloric need. The best carbohydrates are in fruits and vegetables.
- 6) Intestinal toxemia is frequently found to be either the basic cause of or contributing factor to many clinical phenomena, and the holistic practitioner is advised to bear this in mind.  $\Box$

#### References

- 1. Bassler, A.: "Intestinal Toxemia," Medical Journal and Record, Vol 136, 1933, p 322,
- 2. Boeker, H.H.: "Autointoxication," Medical Journal and Record, Vol 128, Sept 19, 1928, p 293.
- 3. Herter, C.A. and Kendall, A.I.: "The Influence of Dietary Alterations on the Types of Intestinal Flora," Journal of Biological Chemistry, Vol 7, 1909-10, pp 203-235.
- 4. Orten, J.M. and Newhaus, O.W.: Human Biochemistry, 9th ed, C.V. Mosby Company, St Louis, Missouri, 1975, p 469.
- 5. Herter, op cit. p 216.
- 6. Cannon, P.R., Dragstedt, L.R., and Dragstedt, C.A.: "Intestinal Obstruction," Journal of Infectious Disease, Vol 27, 1920, pp 139-144.
- 7. Underhill, F.P. and Simpson, G.E.: "The Effect of Diet on the Excretion of Indican and the Phenols," Journal of Biological Chemistry, Vol 44, 1920, pp 69-97.
- 8. Torrey, J.C.: "The Regulation of the Intestinal Flora of Dogs through Diet, "Journal of Medical Research, Vol 39, 1918-19, pp 415-447.
- 9. Folin, O. and Denis, W.: "The Excretion of Free and Conjugated Phenols and Phenol Derivatives," Journal of Biological Chemistry, Vol 22, 1915, p 309. 10. Gerard, R.W.: "Chemical Studies on Intestinal Intoxication,"
- Journal of Biological Chemistry, Vol 52, 1922, pp 111-124.
- 11. Cannon, op cit, pp 139-144.
- 12. Stone, H.B., Bernheim, B.M., and Whipple, G.H.: "Intestinal Obstruction: A Study of the Toxic Factors," Bulletin of The Johns Hopkins Hospital, Vol 23, No 256, 1912, pp 159-165.

- 13. Gerard, R.W.: "The Lethal Agent in Acute Intestinal Obstruction," Journal of the American Medical Association, Vol 79, No 19, 1922, pp 1581-1584.
- 14. Whipple, G.H., Stone, H.B., and Bernheim, B.M.: "Intestinal Obstruction," Journal of Experimental Medicine, Vol 17, 1913, pp 286-307.
- 15. Gerard, R.W.: "Chemical Studies on Intestinal Intoxication," Journal of Biological Chemistry, Vol 52, 1922, pp 111-124.
- 16. Gerard, R.W.: "The Lethal Agent in Acute Intestinal Obstruction," Journal of the American Medical Association, Vol 79, No 19, 1922, pp 1581-1584.
- 17. Stone, H.B., Bernheim, B.M., and Whipple, G.H.: "Intestinal Obstruction: A Study of the Toxic Factors," Bulletin of The Johns Hopkins Hospital, Vol 23, No 256, 1912, pp 159-165.
- 18. Whipple, G.H., Stone, H.B., and Bernheim, B.M.: "Intestinal Obstruction," Journal of Experimental Medicine, Vol 17, 1913, pp 286-307.
- 19. Whipple, ibid, p 306.
- 20. Whipple, ibid, p 305.
- 21. Wilson, D.R., Ing, T.S., Metcalfe-Gibson, A., and Wrong, O.M.: "In Vivo Dialysis of Faeces as a Method of Stool Analysis, III. The Effect of Intestinal Antibiotics." Journal of Clinical Science, Vol 34, 1968, pp 211-221.
- 22. McDermott Jr, W.V., Adams, R.D., and Riddell, A.G.: "Ammonia Levels in Blood and Cerebrospinal Fluid," Proceedings of Society of Experimental Biology and Medicine, Vol 88, 1955, p 382.
- 23. McDermott, W., Adams, R.D.: "Eck-Fistula A Cause of Episodic Stupor in Humans," Journal of Clinical Investigation, Vol 32, 1953, pp 587-588.
- 24. Phillips, G.B., Schwartz, R., Gabuzda, G.J., and Davidson, C.S.: "The Syndrome of Impending Hepatic Coma in Patients with Cirrhosis of the Liver Given Certain Nitrogenous Substances," The New England Journal of Medicine, Vol 247, No 7, 1952, pp 239-246.
- 25. Berger, R.L., Liversage, R.M., Chalmers, T.C., Graham, J.H., McGoldrick, D.M., and Stohlman Jr, F.S.: "Exchange Transfusion in the Treatment of Fulminating Hepatitis." The New England Journal of Medicine, Vol 274, No 9, 1966, pp 497-499.
- 26. Sherlock, S., Summerskill, W.H.J., White, L.P., Phear, E.A.: "Portal-Systemic Encephalopathy. Neurological Complications of Liver Disease," The Lancet, Vol 2, Sept 4, 1954, pp 453-457.
- 27. Visek, W.J., Kolodny, G.M., and Gross, P.R.: "Ammonia Effects in Cultures of Normal and Transformed 3T3 Cells," Journal of Cell Physiology, Vol 80, 1972, pp 373-382.
- 28. Williams, B.W.: "Importance of Toxemia Due to Anaerobic Organisms in Acute Intestinal Obstruction and Peritonitis,' The Lancet, Vol 1, April 30, 1927, pp 907-912.
- 29. Jawetz, E., Melnick, J.L., Adelberg, E.A.: Review of Medical Microbiology, 12th ed, Lange Medical Publications, Los Altos, California, 1976, pp 189-190.
- 30. Orten, op cit, p 471.
- 31. Herter, C.A.: "An Experimental Study of the Toxic Properties of Indol," NY Medical Journal, Vol 68, July 16, 1898, pp 89-93.
- 32. Korenchevsky, V.: "Autointoxications and Processes of Aging," Texas Rep Biology and Medicine, Vol 12, 1956, p 1016.
- 33. Bryan, G.T., Brown, R.R., and Price, J.M.: "Incidence of Mouse Bladder Tumors Following Implantation of Paraffin Pellets Containing Certain Tryptophan Metabolites," Cancer Research, Vol 24, 1964, pp 582-585.
- 34. Kerr, W.K., Barkin, M., Levers, P.E., Woo, S.K-C, and Menczyk, Z.: "The Effect of Cigarette Smoking on Bladder Carcinogens in Man," The Canadian Medical Association Journal, Vol 93, No 1, 1965, pp 1-7.
- 35. Herter, op cit, pp 203-235.
- 36. Robbins, S.L.: Pathologic Basis of Disease, 1st ed, W.B. Saunders Company, Philadelphia, 1974, p 520.
- 37. Folin, op cit, p 320.

The ACA Journal of Chiropractic/April 1979

Vol 13, S-34

- 38. Dubin, H.: "Physiology of the Phenols," Journal of Biological Chemistry, Vol 26, 1916, p 99.
- 39. Folin, op cit, p 317.
- 40. Underhill, op cit, p 96.
- 41. Salant, W. and Kleitman, N.: "The Toxicity of Skatol," Journal of Pharmacology and Experimental Therapeutics, Vol 19, 1922, p 313.
- 42. Herter, op cit, p 215. 43. Bartle, H.J.: "Protein Intoxication," Medical Journal and Record, Vol 128, July 4, 1928, p 30.
- 44. Orten, op cit, p 335. 45. Challenger, F. and Walshe, J.M.: "Foeter Hepaticus," The
- Lancet, Vol 1, 1955, p 1240.
- 46. Orten, op cit, p 467.47. Haubold, H.A.: "General Considerations Regarding Self-Intoxication," The NY Medical Journal, Vol 60, Dec 25, 1897, р 857.
- 48. Orten, op cit, p 468.
- 49. Orten, ibid, p 472.
- 50. Baker, C.E.: "The Physiological Effects of Certain Toxic Substances of Gastro-Intestinal Origin," Illinois Medical Journal, Vol 51, April 1927, pp 325-327.
- 51. Homewood, A.E.: The Neurodynamics of the Vertebral Subluxation, 3rd ed, Valkyrie Press, St Petersburg, Florida, 1977, pp 77-78.
- 52. Korenchevsky, op cit, p 1815.
- 53. Major, R.H.: "Relationship Between Certain Products of Metabolism and Arterial Hypertension," Journal of the American Medical Association, Vol 83, No 2, 1924, pp 81-84.
- 54. Barger, G. and Dale, H.H.: "Chemical Structure and Sympathomimetic Action of Amines," Journal of Physiological Chemistry, Vol 41, 1910-1911, pp 19-59.
- 55. Korenchevsky, op cit, pp 1006-1036.
- 56. Lane, W.A.: "Chronic Intestinal Stasis," Journal of Surgery, Gynecology and Obstetrics, Vol 16, 1913, p 600.
- 57. Bassler, A.: "Chronic Intestinal Toxemia," Medical Record, Vol 145, 1937, p 160.
- 58. Hertz, A.H.: "Chronic Intestinal Stasis," The British Medical Journal, Vol 1, April 19, 1913, p 817.
- 59. Binnie, J.F.: "Symptoms of Colonic Intoxication," Journal of American Medical Association, Vol 58, No 26, 1912, p 2011.
- 60. Bartle, op cit, p 63.
- 61. Satterlee, G.R.: "Chronic Intestinal Stasis," American Journal of Medical Science, Vol 152, 1916, p 729.
- 62. Underhill, op cit, p 96.
- 63. Lane, op cit, pp 600-606.
- 64. Satterlee, op cit, pp 727-738.
- 65. Hertz, op cit, pp 817-821.
- 66. Lucas, C.G.; "Symptomatology of Chronic Intestinal Stasis," Southern Medical Journal, Vol 17, No 9, 1924, pp 659-661. 67. Bartle, op cit, pp 63-66.
- 68. Tucker, John: "Intestinal Toxemia," Medical Clinics of North America, Vol 19, May 1936, pp 1819-1830.
- 69. Alverez, W.C.: An Introduction to Gastro-enterology, 4th ed, Paul B. Hoeber, Inc. New York; NY, 1948, p 638.
- 70. Guyton, A.C.: Textbook of Medical Physiology, 5th ed, W.B. Saunders Company, Philadelphia, 1976, p 674.
- 71. Whipple, G.H., Stone, H.B., and Bernheim, B.M.: "Intestinal Obstruction," Journal of Experimental Medicine, Vol 17, 1913, p 305.
- 72. Gerard, R.W.: "The Lethal Agent in Acute Intestinal Obstruction," Journal of the American Medical Association, Vol 79, No 19, 1922, p 1583.
- 73. Cannon, op cit, p 143.
- 74. Whipple, G.H., Stone, H.B., and Bernheim, B.M.: "Intestinal Obstruction," Journal of Experimental Medicine, Vol 17, 1913, p 322.
- 75. Soper, H.W.: "The Mucosa of the Rectum and Sigmoid Colon as a Focus of Infection," Boston Medical and Surgical Journal, Vol 176, No 22, 1917, p 766.
- 76. Bartle, op cit, p 64.

- 77. Lucas, op cit, p 660.
- 78. Woolley, P.G.: "Intestinal Stasis and Intestinal Intoxications: A Critical Review," Journal of Laboratory and Clinical Medicine, Vol 1, 1915-1916, p 50.
- 79. Guyton, op cit, p 897.
- 80. Synnott, M.J.: "Intestinal Toxemia, Its Diagnosis and Treatment," Medical Journal and Record, Vol 136, No 11, 1932, n 441.
- 81. Warwick, R. and Williams, P.L.: Gray's Anotomy, 35th British ed, W.B. Saunders Company, Philadelphia, 1973, p 588.
- 82. Bartle, op cit, p 387.
- 83. Haubold, op cit, p 859.
- Bassler, A.: Intestinal Toxemia Biologically Considered, F.A. 84. Davis Company, Philadelphia, 1930.
- Lintz, W.L.: "Gastrointestinal Allergy," The Review of Gastroenterology, Vol 6, 1939, p 321.
- 86. Lintz, ibid, pp 320-332.
- 87. Eustis, A.: "Further Evidence in Support of the Toxic Pathogenesis of Bronchial Asthma, Based upon Experimental Research," American Journal of Medical Science, Vol 143, 1912, p 863.
- 88. Rochester, D.: "The Treatment of Asthma," Journal of the American Medical Association, Vol 47, No 24, 1906, p 1984.
- 89. Bassler, A.: "The Colon in Connection with Chronic Arthritis (Arthritis Deformans)," American Journal of Medical Science, Vol 160, 1920, p 357.
- 90. Bassler, A.: "Aging, Arteriosclerosis, and Cardiac Conditions," Medical Record, Vol 153, Jan 1, 1941, p 21.
- 91. Lane, W.A.: "Consequences and Treatment From a Surgical Point of View," British Medical Journal, Vol 1, March 15, 1913, p 547.
- 92. Lucas, op cit, p 661.
- 93. Guyton, op cit, p 213.94. Bassler, A.: "Coronary Disease and the Intestine," Medical Record, Vol 155, April 1, 1942, p 249.
- 95. Bainbridge, W.S.: "The Constitutional Effect of Prolonged Intestinal Toxemia," Medical Journal and Record, Vol 122, No 8, 1925, p 438. Barry, D.T.: "Intestinal Toxins and the Circulation," The
- 96. Lancet, Vol 2, July 1, 1916, p 15.
- 97. Hovell, T.M.: "Gastro-intestinal Sepsis, a Cause of Meniere's Symptoms," Proceedings of the Royal Society of Medicine, Vol 11, No 3, 1918, p 16.
- 98. Stucky, J.A.: "Intestinal Autointoxication as a Factor in the Causation of Pathologic Conditions of the Ear, Nose and Throat," Journal of the American Medical Association, Vol 53, No 15, 1909, p 1185.
- 99. Hovell, op cit, pp 15-18.
- Gatewood, W.L.: "Symptoms of Gastrointestinal Origin in the Ear, Nose and Throat," Archives of Otolaryngology, Vol 33, 1941, pp 592-598.
- 101. Hawley, C.W.: "Autointoxication and Eye Diseases," Ophthalmology, Vol 10, No 4, 1914, pp 663-674.
- 102. Reveno, W.S.: "The Cause of Exophthalmic Goiter," Archives of Internal Medicine, Vol 48, Oct 1931, p 597.
- 103. Eustis, A.: "Some Interesting Observations on Goiter," New Orleans Medical and Science Journal, Vol 85, June 1933, pp 892-898.
- 104. Lane, W.A.: "Chronic Intestinal Stasis," Journal of Surgery, Gynecology and Obstetrics, Vol 16, 1913, p 602.
- 105. Von Noorden, C.: "Intoxication Proceeding From the Intestine, Especially Polyneuritis," Journal of the American Medi-cal Association, Vol 60, No 2, 1913, p 104.
- 106. Von Noorden, ibid, p 105.
- 107. Herter, C.A. and Smith, E.E.: "Researches upon the Etiology of Idiopathic Epilepsy," NY Medical Journal, Vol 56, 1892, pp 208-211, 234-239, 260-266.
- 108. Satterlee, G.R. and Eldridge, W.W.: "Symptomatology of the Nervous System in Chronic Intestinal Toxemia," Journal of the American Medical Association, Vol 69, No 17, 1917, pp 1414-1418.
- 109. Haubold, op cit, pp 857-861.

- 110. Satterlee, op cit, p 1414.
- 111. Satterlee, ibid, p 1417.
- 112. Bainbridge, op cit, pp 437-443.
- 113. Nascher, I.L.: "Lane's Autointoxication Complex and the Manifestations of Senility," NY Medical Journal, Vol 100, No 6, 1914, p 256.
- 114. Osgood, R.B.: "Etiologic Factors in Certain Cases of So-Called Sciatic Scoliosis," Journal of Bone and Joint Surgery, Vol 9, Oct 1927, pp 667-676.
- 115. Von Noorden, op cit, p 103.
- 116. Schwartz, H.J.: "Association of Intestinal Indigestion with Various Dermatoses," Archives of Dermatology and Syphilology, Vol 13, 1926, p 674.
- 117. Burgess, J.F.: "Endogenous Irritants as Factors in Eczema and in Other Dermatoses," Archives of Dermatology and Syphilology, Vol 16, No 2, 1927, p 139.
- 118. Galloway, J.: "Cutaneous Indications of Alimentary Toxaemia," British Medical Journal, Vol 1, April 19, 1913, pp 815-817
- 119. Bartle, op cit, p 28.
- 120. Lane, W.A.: "Consequences and Treatment from a Surgical Point of View," British Medical Journal, Vol 1, March 15, 1913. p 547
- 121. Lane, W.A.: "Chronic Intestinal Stasis," Journal of Surgery, Gynecology and Obstetrics, Vol 16, 1913, p 601.
- Bainbridge, op cit, p 443.
  Prehn, R.T.: "A Clonal Selection Theory of Chemical Carcinogenesis," Journal of National Cancer Institute, Vol 32, No 1, Jan 1964, p 1.
- 124. Satterlee, G.R.: "Autogenous Colon Vaccines in the Study, Diagnosis and Therapy of Chronic Intestinal Toxemia." Journol of American Medical Association, Vol 67, No 24, 1916, p 1731.
- 125. Bartle, op cit, p 448.
- 126. Goodhart, R.S. and Shils, M.E.: Modern Nutrition in Health and Disease, 5th ed, Lea and Febiger, Philadelphia, 1973, р 30.
- 127. Hegsted, D.M.: "Minimum Protein Requirements of Adults," American Journal of Clinical Nutrition, Vol 21, No 5, May 1968, pp 352-357.
- 128. Turck, F.B.: "Intestinal Venous Stasis: Diffusion of Bacteria and Other Colloids," Boston Medical and Surgical Journal, Vol 176, No 19, 1917, p 665.
- 129. Soper, H.W.: "Autointoxication in Chronic Constipation," Journal of the American Medical Association, Vol 69, No 18, 1917, p 1512.
- 130. Bartle, op cit, p 447.
- 131. Stucky, op cit, p 1186.
- 132. Synnott, op cit, p 444. 133. Saundby, R.: "Alimentary Toxemia: Its Symptoms and Treatment," British Medical Journal, Vol 1, March 15, 1913, p 545.
- 134. Dubin, op cit, p 91.
  135. Gerard, R.W.: "The Lethal Agent in Acute Intestinal Obstruction," Journal of American Medical Association, Vol 79, No 19, 1922, p 1583.
- 136. Sherwin, C.P. and Hawk, P.B.: "Fasting Studies: VII. The Putrefaction Processes in the Intestine of a Man During Fasting and During Subsequent Periods of Low and High Protein Ingestion," Journal of Biological Chemistry, Vol 11, No 3, 1912, p 177,
- 137. Fitch, W.E.: "Putrefactive Intestinal Toxemia," Medical Journal and Record, Vol 132, Aug 20, 1930, p 186.
- 138. Bainbridge, op cit, p 440.
- 139. Soper, H.W.: "Autointoxication in Chronic Constipation," Journal of the American Medical Association, Vol 69, No 18, 1917, p 1512.

- 140. Bassler, A.: "Chronic Intestinal Toxemia," Medical Record. Vol 145, 1937, p 159.
- 141. Fordtran, J.S., Scroggie, W.B., and Polter, D.E.: "Colonic absorption of tryptophan metabolites in man," J Lab and Clin Med, Vol 64, 1964, p 125-132.
- 142. Bakke, O.M.: "Urinary simple phenols in rats fed purified and nonpurified diets," J Nutr, Vol 98, 1969, p 209.
- 143. Folin, op cit, p 309.
- 144. Bakke, O.M.: "Urinary simple phenols in rats fed diets containing different amounts of casein and 10% tyrosine," J Nutr, Vol 98, 1969, pp 217-221.
- 145. Aarbakke, J. and Schjönsby, H.: "Value of urinary simple phenol and indican determinations in the diagnosis of the, stagnant loop syndrome," Scand J Gastroent, Vol 11, 1976, pp 409-414.
- 146. Tomkin, G.H. and Weir, D.G.: Quart J Med, Vol 41, 1972, pp 191-203.
- 147. Fordtran, et al, op cit.
- 148. Neale, G., Lambert, R.A., and Gorbach, S.L.: "The production of indole by bacteria in vitro," Gut, Vol 10, 1969, pp 1056-1057
- 149. Happold, F.C.: "Tryptophanase-tryptophan reaction," Advances in Enzymology, Vol 10, 1950, p 51.
- 150. Donaldson, R.M., Jr : "Malabsorption of Co60-Labeled Cvanocobalamin in Rats with Intestinal Diverticula. I. Evaluation of Possible Mechanisms," Gastroenterology, Vol 43, 1962, p 271.
- 151. Robbins, op cit, p 55.
- 152. Thorn, G.W., Adams, R.D., Bruanwald, E., Isselbacher, K.J., Petersdorf, R.G.: Harrison's Principles of Internal Medicine. 8th ed, McGraw-Hill Book Co, New York, NY, 1977, p 715. 153. Thorn, ibid, p 1606.
- 154. Thorn, ibid, p 1909.
- 155. Tabaqchali, S., Scand J Gastroent. 1970 Suppl, Vol 6, pp 139-163.
- 156. Bakke, O.M., Scand J Gastroent, Vol 4, 1969, pp 603-608.
- 157. Alam, S.Q., Boctor, A.M., Rogers, Q.R., and Harper, A.E.: "Some effects of amino acids and cortisol on tyrosine toxicity in the rat," J Nutr, Vol 93, 1967, p 317.
- 158. Bakke, O.M.: "Urinary simple phenols in rats fed diets containing different amounts of casein and 10% tyrosine," J Nutr, Vol 98, 1969, pp 217-221.
- 159. Horning, E.C., and Dalgliesh, C.E.: "The association of skatole-forming bacteria in the small intestine with the malabsorption syndrome and certain anemias," Biochem J, Vol 70, 1958, p 13.
- 160. Izquierdo, J.A., and Stoppani, A.D.M.: "Inhibition of smooth muscle contractility by indole and some indole compounds," Brit J Pharmacol, Vol 8, 1953, pp 389-394.
- 161. Proctor, M.H.: "Bacterial dissimilation of indoleacetic acid: a new route of breakdown of the indole nucleus," Nature, Vol 181, 1958, p 1345.
- 162. Horning, E.C. and Dalgliesh, C.E.: "The association of skatole-forming bacteria in the small intestine with the malabsorption syndrome and certain anemias," Biochemical I. Vol 70, 1958, p 13.
- 163. Sprince, H.: "Biochemical aspects of indole metabolism in normal and schizophrenic subjects," Annals New York Academy of Sciences, Vol 96, 1962, pp 399-418.
- 164. Dalgliesch, C.E., Kelly, W., and Horning, E.C.: "Excretion of a sulphatoxy derivative of skatole in pathological states in man," Biochemical J, Vol 70, 1958, p 13.
- 165. Horning, E.C., Sweeley, C.C., and Kelly, W.: "Mammalian hydroxylation in the 6-position of the indole ring," Biochem Biophys Acta, Vol 32, 1959, pp 566-567.