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THE CLINICAL Chemist

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VOL. 5, NO. 6 DECEMBER 1953

THE SECRETARY REPORTS

At the time this issue of THE CLINICAL CHEMIST makes its appearance, the AACC will have completed its first five years. The rapid growth of the Association in this relatively short period is an acknowledgement of the need for such an organization as ours.

Various objectives of the Association set up in the first stages have either already been satisfactorily completed or else well under way. The foremost accomplishment is the evolution of THE CLINICAL CHEMIST from a small newsletter to that of a responsible journal. "Standard Methods" is finding a useful place in the laboratory, with subsequent volumes in preparation. The Ernst Bischoff Award has been established as an outstanding annual event. A Code Of Ethics is now part of our governing principles. Other progress notes have frequently been noted in these columns.

The one phase of Association activity that has not been fully explored is the formation of local sections. The ideal organizational arrangement for us would probably be that of various autonomous local groups consolidated into a cohesive national body. Not the least in importance of local groups is the matter of legislation as it pertains to clinical chemistry, which cannot be properly dealt with from national headquarters. Those people from one State will not benefit from adequate laws governing clinical chemistry in another State. Legislators throughout the country are being made aware of the laboratory problems and they must be alerted to the fact that the public will be better served by high standards in clinical chemistry. Local sections are in the best position to do that.

The membership of the AACC is widely distributed and yet only six local sections are in operation at this time, of which four are along the northeast coast. The other two are Chicago and Southern California. Formation of local sections can only be realized by the initiative of the members in any area.

Max M. Friedman National Secretary

PHILADELPHIA SECTION

The first meeting of the 1953-54 season of the Philadelphia Section, American Association of Clinical Chemists, was held at 8:00 p.m. on Tuesday, October 27, 1953, in the North Lecture Room of the Graduate Hospital of the University of Pennsylvania. Prior to the meeting, there was an informal dinner in honor of the speaker at the Homestead Restaurant.

The President, Mr. A. G. Keller, introduced Dr. David Seligson, Director of the Division of Biochemistry at the Graduate Hospital who spoke on "The Measurement of Chlorides in Biological Fluids and a New Method for the Measurement of Chlorides".

With due consideration to their limitations, as well as to their advantages, Dr. Seligson described the techniques for chloride determination which are now commonly used in clinical laboratories. He then described an electrometric titration for chloride analysis which has been developed at the Graduate Hospital. After the lecture, Dr. Seligson kindly consented to answer questions related to his subject.

At the conclusion of the formal meeting, the group adjourned to the laboratories of the Graduate Hospital for a demonstration of the technique.

BOSTON SECTION

The Boston Section held its first meeting of the current season on October 21st at the New England Center Hospital.

Annual election of officers were held at this time, the following were elected:

Chairman—Arthur DeTore: Sias Laboratories

Chairman ——— Arthur DeTore: Sias Laboratories
Vice-Chairman—Joseph Annino: Massachusetts Memorial Hospital
Sec. Treasurer—Esther Thomas: New England Center Hospital

The speaker of the evening was Joseph Benotti of the Boston Medical Laboratory whose subject was "Protein-Bound Iodine, and its Relation to Total Iodine."

Protein-Bound Iodine (PBI) according to the speaker, and to a rapidly growing literature, constitutes a valuable criterion in evaluating thyroid pathology. According to some, the PBI now is considered a more reliable tool than radio-iodine uptake, and more sensitive an indicator than the serum cholesterol or BMR.

Mr. Benotti reviewed the methods of more recent years for determining PBI; mentioning those of Chaney, Barker, and Zak, all of which depend ultimately on the catalysis by iodine of the yellow ceric ion to the colorless cerous ion. In his own laboratory, the speaker uses the method of Zak.

Basing his opinion on hundreds of PBI's done at his laboratory, the speaker dealt at length with the importance of simultaneous Total Iodine determinations on each specimen. This is done simply by omitting protein precipitation with trichloracetic acid. Greater significance is given to the PBI, he explained, since iodine in varied form is often used in medication. If the iodine contaminant is inorganic (Lugol's, hydriodic acid) it is generally possible to wash it from the protein precipitate during the early stages of the determination.

Foreign iodine administered in organic form, as in such x-ray contrast

(Continued on following page)

QUID NUNCS

ALBERT E. SOBEL, past president of the AACC has just completed a group of lectures on the chemistry of bone and tooth formation before local sections of the ACS. Dr. Sobel was guest speaker at meetings of the Penn.-N.Y. Western Border, Penn.-Ohio Border, Columbus, Wooster, Northeastern Ohio and Akron Sections of the American Chemical Society.

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Dr. Joseph Greenspan announces that his company, Process And Instruments are now situated in larger quarters. The new address is, 15 Stone Avenue, Brooklyn 33, N.Y.

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ALEXANDER T. SHULGIN, formerly a member of the Biochemistry Department of the University of California at Berkeley, becomes director of the Radiochemical Division, BIO-RAD Laboratories, Berkeley, California. Prior to his faculty position at the University, he attended Harvard University and the University of California. His work and publications have been in the field of isotopic organic syntheses for investigation of metabolic processes.

In his new post, Dr. Shulgin will be responsible for technical operations concerned with production of the Laboratories' specialized line of radiochemicals for the biological and medical fields.

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media as lipiodol or diodrast, may cause greater difficulty the speaker explained. In the latter cases, the iodine may be in some way incorporated with protein, so that analytically it may respond as PBI. In these cases, the Total Iodine may be as much as 20 or more micrograms %, with an appreciable difference between it, and the PBI. Normally the speaker stated, the PBI and Total Iodine are either the same, or at most 1 or 2 micrograms % apart from each other. A greater divergence than this, together with a high Total Iodine, should cast some doubt on the diagnostic value of the PBI.

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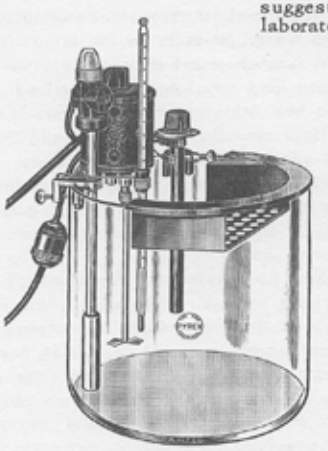

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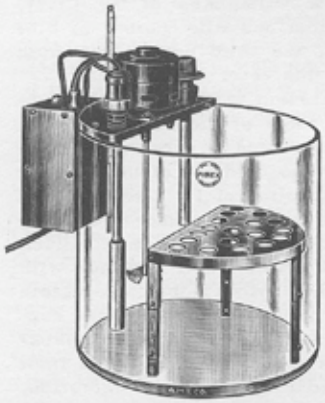

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CHEMICAL EVALUATION OF THE FUNCTIONS OF THE LIVER

by

John G. Reinhold

William Pepper Laboratory of Clinical Medicine, Hospital of the University of Pennsylvania, Philadelphia, Pa.

PART I

(A) The Anatomy Of The Liver
(B) Diseases Of The Liver And Biliary Tract
(C) The Chemical Physiology Of Liver Disease
  (1) Carbohydrate Metabolism
  (2) Cholesterol And Lipid Metabolism
  (3) Nitrogen Metabolism

INTRODUCTION

In discussing tests for liver function an attempt has been made to fit the various practical tests into the larger picture presented by the disturbed chemical physiology of liver disease. The practical tests described are those that the author and his colleagues have found to be useful. Such a selection is conditioned by many factors besides the excellence of a given test and technique, and it should not be inferred that alternative methods are inferior. Furthermore, limitations of time and space, the fact that much of the writing was done while on vacation in Mexico two hundred miles from a library, and the undoubted existence of gaps in the knowledge of the reviewer will account for the failure to mention certain contributions. For further information and a truly comprehensive review, the reader is referred to "Diseases of the Liver, Gall Bladder and Bile Ducts" by S.S. Lichtman, the third edition of which appeared this year.

The present review is to be published in three sections. The first is in this issue, and is concerned with a review of the anatomy of the liver, a brief description of its diseases, and a discussion of disturbances in carbohydrate, fat and protein metabolism. A second section will continue with protein metabolism, the non-protein nitrogen, blood clotting, iron and copper metabolism, detoxification, phosphatase, and bile pigments. The final section will include dye excretion, selection and application of hepatic tests, and the bibliography.

Much of the metabolic activity of the body is centered in the liver. It transforms food into readily usable nutrients, stores such nutrients until needed, releases them into the circulation and regulates their concentrations, and disposes of the waste products of their metabolism. The liver manufactures many substances not available in the food. It detoxifies unwanted materials. It secretes adjuvants to digestive processes in the intestine and excretes waste products in the bile. It releases hormones and enzymes into the circulation and regulates the level of hormones from other sources. It forms lymph. The liver contributes to the production of blood plasma, including substances concerned with clotting. It aids in regulation of the circulating blood volume. It participates in the resistance to infection. It produces heat.

Before discussing the functions of the liver and means for studying them, a resume of the main facts concerning its anatomy seems appropriate. It is necessary also, to describe the major diseases of the liver in order to define more clearly which chemical studies should be made and the response that may be expected.

THE ANATOMY OF THE LIVER

The liver of a healthy adult weighs 1650 grams on the average and it is by far the largest gland in the body. It consists of lobes which vary in size and differ to some extent in the sources of their afferent blood supplies. The liver is encased in a membrane of connective tissue. This extends into the liver at the transverse fissure of the under surface (porta) in the form of a tree with innumerable branches. This framework of connective tissue supports the parenchymal cells, blood vessels, and bile ducts. The portal vein, hepatic artery, hepatic vein and bile ducts enter the liver through the porta also, and undergo equally extensive branching. In its finer structure the liver consists of lobules, one to two millimeters in diameter. The lobules consist of numerous tubular secretory units, each formed by parenchymal cells surrounding a bile canaliculus. Blood reaches the lobules mainly from the branches of the portal vein, but this source is supplemented by arterial blood from the hepatic artery. The blood enters the sinusoids, spaces between cords or sheets of hepatic cells, and passes through them to drain into the central vein of the lobule. The relationship of these structures is shown in Figure 1. (Figure 1 = Figure 352 in Histology, by A.W. Ham, 2nd ed. J.B. Lippincott, 1953) Flow of blood through the liver and the blood supply of the lobules is controlled by an intricate mechanism, much of it located within the lobule itself (Macgraith, B.G., and others, 1951).

Recent studies (Elias, 1953) have altered previously accepted concepts of the liver structure by demonstrating that the parenchymal cells are arranged in sheets. Between these are the sinusoidal spaces which make possible direct access of blood to these cells.

The parenchymal cells, the most abundant and characteristic cells in the liver, have both secretory and metabolic functions. Furthermore they have both exocrine and endocrine secretory functions and are arranged so that each cell faces both a duct and a sinusoid containing blood.

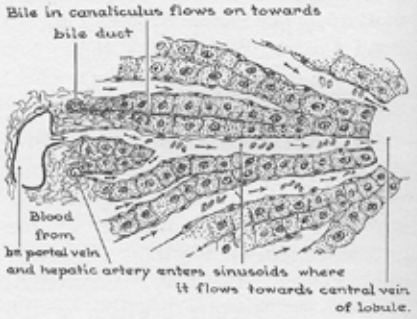

Fig. 1. Drawing (at high-power magnification) to show how blood from the portal vein and the pepatic artery (at left) flows into sinusoids, lined by reticulo-endothelium, that lie between liver cords and empties into the central vein (right). The way that bile travels in the opposite direction in canaliculi to empty into bile ducts in portal areas is also shown.

Besides the parenchymal cells, the liver contains large numbers of reticulo-endothelial (Kupffer) cells which form a lining for the sinusoids. Some estimates indicate that up to one-third of the total number of liver cells are of this type. This potentially hemopoietic tissue is similar to that in bone marrow and spleen. The Kupffer cells have phagocytic properties and are important in connection with hemoglobin breakdown and immunity reactions.

DISEASES OF THE LIVER AND BILIARY TRACT

Disturbances of metabolism occurring in liver disease may be sufficiently severe to jeopardize survival. They may be the result of (1) failure of the parenchymal cells to carry out vital functions because of infections or noxious agents (2) decreased mass of functioning parenchymal cells resulting from disease (3) decreased availability of blood to the liver cells because

of (a) distortion of the liver architecture by scar tissue, (b) prolonged disturbance of the mechanisms regulating flow of blood in the liver with consequent shunting of blood around the liver or through it, (c) extrahepatic interference with blood supply, (4) impaired nutrition, (5) reaction of other organs to liver damage, e.g. brain, kidney, pancreas, adrenal, gonads, spleen, (6) injudicious therapy.

Infectious disease of the liver, such as viral hepatitis, is characterized by degeneration and necrosis and regeneration of parenchymal cells as shown by variations in size and staining characteristics. Necrosis and complete disappearance of cells and destruction of the normal architecture of the lobule by scar tissue may follow. Scavenger cells, histiocytes, lymphocytes and plasma cells infiltrate the damaged regions. Edema may occur and together with the disorganisation of structure, contribute to decrease the blood supply. Regeneration of the parenchymal cells can occur rapidly and produce an astonishingly large mass of cells within as little as 24 to 48 hours. Regeneration is favored by a well-maintained blood supply, and lacking this, fails to occur (Grindley and Bollman, 1952).

Viral hepatitus ordinarily runs its course within one to three months with apparently complete recovery. It may however, become chronic in about 1 per cent, persisting for many years, at times in an asymptomatic form. Formerly, viral hepatitus was often mistaken for a disease of the bile ducts and the term "catarrhal jaundice" in the older literature is a result of this confusion.

The term cholangiolitic hepatitis has been applied by Watson and Hoffbauer (1946) to a syndrome in which inflammation of the smaller bile passages predominate. It differs in a striking manner in its effect on the chemical composition of the blood from those diseases that involve primarily the parenchymal cells. Chemical changes are similar to those found in biliary obstruction and all of the resources of medical knowledge may be required to make a differentiation.

Deposition of fat in the liver may occur as part of a general adiposity resulting from overnutrition, as a result of dietary deficiencies, or as an effect of the action of toxic substances. Dible (1951) has recently evaluated the significance of liver fat and has demonstrated experimentally that liver fat tends to be increased in proportion to body fat. Fatty liver resulting from overnutrition has little importance insofar as the function of the liver is concerned. However, deposition of fat caused by toxic substances is evidence of a serious disturbance of hepatocellular function. Among the nutritional deficiencies associated with fatty liver are those of methylation induced by lack of betaine, choline, methionine, or other extrinsic sources of methyl groups. Low protein intakes may also contribute to the production of fatty livers.

Although liver disease of nutritional origin is uncommon in the United States (if the chronic alcoholic is excepted), it is a major problem in many parts of the world, notably the tropical regions of Africa and Asia, and in the Caribbean area. It may be accompanied by infiltration of fat, necrotic changes and cellular infiltration and in certain instances by accumulation of exudate in the liver. In certain parts of Africa, primary carcinoma of the liver may be a major manifestation of nutritional deficiency, in others, cirrhosis has been reported in four of five necropsies.

Liver damage may occur as a result of severe strains on metabolism associated with many diseases. In infectious mononucleosis it may be sufficiently severe to constitute a grave hazard to the patient. Marked jaundice and severe alterations in liver function are seen in such patients. Malaria, lobar pneumonia, typhoid fever, various anemias, syphilis and cholera serve as other examples. Diabetes and thyrotoxicosis also may lead to marked liver damage. Surgical operation and anesthesia cause impairment of liver function in some patients (Fairlie and others, 1951). Liver failure may occur in pregnancy.

A failing heart and the resulting accumulation of blood in the liver and other viscera (chronic passive congestion) may lead to marked impairment of the efficiency with which the liver functions. Hypoxia due to other causes also leads to liver damage.

Proliferation of the connective tissue of the liver may result from liver disease of infectious, nutritional, toxic, hypoxic, or neoplastic etiology, or it may occur spontaneously because of diminished blood supply resulting from circulatory and other factors. The overgrowth of connective tissue in turn leads to disorganization of the liver structure and this again to further interference with the blood supply. The end result is a shrunken liver consisting largely of connective tissue, and with a markedly decreased mass of parenchymal, reticuloendothelial and vascular tissue. The designation of portal cirrhosis, atrophic cirrhosis or Laennec's cirrhosis has been used at various times to denote such scarred livers. Although not all scarred livers fit the pathologist's definition of Laennec's cirrhosis, this descriptive term appears to be preferred at present. Laennec's cirrhosis accounts for a large proportion of the liver disease encountered in American hospitals.

Cirrhosis of a completely different etiology, differing also in its morphologic and chemical characteristics, appears after biliary obstruction has persisted for long periods. Known as biliary cirrhosis, it is relatively uncommon. This condition may occur spontaneously also, its etiology being unclear.

Obstruction of the bile ducts often causes jaundice which may be attributed erroneously to liver disease. It is essential to distinguish jaundice due to biliary obstruction from that caused by liver disease or excessive destruction of blood, because obstruction requires surgical treatment, whereas surgery may not be well tolerated by the patient with liver disease or be needed by the patient who is hemolyzing his erythrocytes. Gall stones entering the common bile duct are the usual cause of biliary obstruction. Other causes include neoplastic disease of the ducts, especially of the ampulla of Vater, or carcinoma of the head of the pancreas. Stricture of the ducts may follow infection, surgical exploration or other trauma. Disease of the gall bladder frequently is complicated by liver damage. The pancreas often shows evidence of being involved. Failure to establish the presence of biliary obstruction and to correct it may lead eventually to biliary cirrhosis.

Carcinoma or other types of neoplastic disease of the liver may or may not be accompanied by jaundice. Often such lesions present few signs to the physician. Obstruction of the bile ducts within the liver by neoplasm presumably brings about the rise in alkaline phosphatase activity in serum which provides one of the few diagnostic aids available.

Damage to the liver may be caused by a large number of chemicals and drugs. Carbon tetrachloride has been extensively used for production of experimental liver damage, and probably has been involved more often than realized as an insidious cause of clinical liver disease. Atophan, various sulfonamides, para aminobenzoate, testosterone, arsenicals, and others have been implicated. The effect on liver function, as measured by laboratory studies, may resemble that observed in biliary obstruction rather than parenchymal liver disease.

Abscesses and cysts of the liver occurring as a result of infections or parasites generally are too localized and involve too little of the liver substance to bring about measurable changes in liver function.

Liver disease may affect the metabolism and functions of other organs. Effects upon the brain are especially noteworthy. Continuing liver disease of maximal severity may eventually lead to loss of consciousness or convulsions, and the characteristic electroencephalographic pattern of the syndrome known as hepatic coma. Hepatic coma is frequently but not necessarily fatal. Recovery may occur spontaneously but also has been attributed to a variety of therapeutic agents. Among treatments reported as successful are: the injection of nicotinic acid, thiamine phosphate, vitamin B_6, multiple B vitamins, glutamic acid and glucose. Exchange transfusion and oxygen therapy also have been used.

Impairment of kidney function commonly accompanies liver disease, and may become a grave problem. The coexistence of hepatic and renal failure is often referred to as the "hepato-renal syndrome". Farquhar (1949) has described the deterioration of kidney function occurring in viral hepatitis.

CHEMICAL EVALUATION OF THE FUNCTION OF THE LIVER

THE CHEMICAL PHYSIOLOGY OF LIVER DISEASE

The list of chemical disturbances observed in liver disease is a long one and will become lengthened as metabolism in liver disease is studied more intensively. Knisely (1951) has listed 18 major asserted chemical functions of the liver with 75 subheadings. Many of these cannot be discussed here for lack of space.

Carbohydrate metabolism.— Studies of hepatectomized animals by Mann (1927) and his collaborators showed conclusively the vital importance of the liver for maintenance of the blood glucose concentration. Although hypoglycemia is not a common complication in patients suffering from acute parenchymal liver disease, it does occur in cirrhosis of the Laennec type with sufficient frequency to require that fasting blood glucose determinations be included in the study of such patients. Blood sugar concentrations as low as 25 mg/100 ml. are not uncommon. A diagnosis of islet cell adenoma of the pancreas occasionally is erroneously made in cirrhotic patients because of recurrent hypoglycemia (Conn et al). Waife (1951) and collaborators showed that insulin resistance occurs in cirrhosis, the fall in blood sugar after a standard insulin injection being delayed as compared with that of healthy individuals. However, hypoglycemia following the insulin persisted for a much longer time than in healthy controls. Thus a markedly impaired ability to mobilize glucose in response to hypoglycemia may be deduced. Injection of epinephrine causes a smaller rise in blood sugar of patients with liver disease than it does in healthy subjects (Geill, 1943; Kinsell and associates, 1949). Hillman (1949) reports this test to have doubtful value in the study of liver function.

Glucose administered to patients with liver disease often causes a greater and more persistent rise in blood glucose than it does in healthy individuals, Conn et al; (Campbell and Tagnon, 1946). Smith, Ettinger, and Seligson (1953) have studied the metabolism of fructose and of glucose in patients with liver disease. They found evidence of impaired ability to utilize both, but not of sufficient consistency to enable application as diagnostic or functional tests.

Decreased utilization of galactose in liver disease has provided the basis for one of the earlier tests of liver function (Bauer, 1906). The measurement of galactose excretion in urine originally used has been replaced by measurement of blood galactose concentrations (Althausen, Lockhart and Soley, 1940), (Althausen, 1949) (Zieve, Hill and Nesbitt, 1950). Colcher, Patek and Kendall (1946) have described an intravenous galactose tolerance test by which the quantity of galactose removed per minute was found to be markedly decreased in liver disease. The cause of the elevated blood galactose and increased output in urine characteristic of severe liver disease has not been established, but presumably it represents interference with the enzymatic conversion of galactose to glucose as described by Caputto, Leloir, Cardini and Paladini (1950). An idiopathic defect in galactose metabolism involving a galactose kinase has been studied by Greenman (1950). The liver is thought to be the source of this enzyme.

Methods for study of disturbances of carbohydrate metabolism in liver disease.— Blood sugar determinations after an overnight fast generally will enable detection of hypoglycemia. A diet very low in carbohydrate accentuates the tendency toward hypoglycemia. (Conn. et al (1938). Glucose tolerance measured by standard three or five hour techniques may be used but, as indicated above offers little useful diagnostic information. Measurement of phosphate concentrations in serum also fails to differentiate clearly between the several causes of impaired glucose tolerance. Methods for galactose tolerance are given in the preceding paragraph. Further studies by means of these improved techniques are needed for appraisal of this procedure. The recent literature gives little reason to believe that fructose tolerance tests would be of value in diagnosis of liver disease.

Serum cholesterol and lipids in liver disease.— Recent work has supported the belief that the liver is the principal organ concerned with the metabolism and excretion of cholesterol. The serum lipids often show marked changes in diseases of the liver and biliary tract. Although serum cholesterol concentration and the partition between free and esterified cholesterol is most frequently studied, neutral fat and especially phospholipid may also show marked changes. Relationships between free and esterified cholesterol and between free cholesterol and phospholipid which in health are maintained within narrow limits are subject to striking disturbances in severe liver disease. Liver disease in which injury to the parenchymal cells is severe is characterized by lowered lipid concentrations which often fall below the minimal concentrations observed in normal individuals of the same age. The onset of jaundice in viral hepatitis is soon followed by falling serum cholesterol concentrations, the result largely of a decrease of the esterified component. Both concentration of esterified cholesterol and the percentage of the total cholesterol esterified are lowered. If liver damage is very severe, esterified cholesterol may become undetectable. Recovery is accompanied by rising concentrations of the esterified cholesterol in serum. Free cholesterol often is increased at the same time so that moderately elevated concentrations may exist during the stage of remission.

Cirrhosis of the Laennec type, is also characterized by low serum lipid concentrations especially when atrophy of the liver is extensive. Serum cholesterol concentrations of less than 100 mg./100 ml. are quite common. The proportion of esterified cholesterol also is lowered, although exceptions exist. Low phospholipid concentrations also are the rule. Cayer and Comatzer (1950) found decreased rate of phospholipid synthesis in cirrhosis.

Production of fatty livers in animals by nutritional deficiencies or other means tends to lower serum cholesterol and other lipid components of serum. There is some evidence for a similar reciprocal relationship in man.

Biliary obstruction regardless of cause is characterized by elevated concentrations of serum lipids. Extremely high concentrations, among the highest known to occur due to any cause, are encountered in patients with biliary obstruction of long duration or with biliary cirrhosis. Ahrens et al. (1950) in a review dealing with biliary cirrhosis describe a disproportionate increase in serum phospholipid concentration. They have also clarified the relationship of elevated serum lipid concentrations to xanthelasma and xanthomatosis.

Cholangiolitic hepatitis and toxic hepatitis of the type caused by chemicals and drugs also are associated with elevated serum lipid concentrations.

Some use is made of serum cholesterol analyses for differentiation of primarily parenchymal from primarily biliary lesions in jaundiced patients. However, such analyses offer little information that cannot be obtained more easily and dependably by other methods. Not only do some patients with biliary obstruction fail to show a rise in concentration of serum cholesterol exceeding the broad range of concentrations found in healthy individuals, but abnormally elevated serum cholesterol concentrations occur in many patients with infectious hepatitis or other predominantly parenchymal types of liver disease. Such increased levels are observed especially as regeneration of the liver proceeds.

The finding of low concentrations and ratios of esterified to total cholesterol in a jaundiced patient is strong but not conclusive evidence for parenchymal liver involvement of severe degree. Serial studies of the esterified cholesterol concentration provide means for estimating changes in the state of the liver in parenchymal liver disease of maximal severity. They do not however, reliably indicate the prognosis. Esterified cholesterol measurements have their greatest usefulness in study of such patients because more sensitive tests, such as bromsulfalein retention and the turbidity tests become maximally positive when there is still a substantial amount of functioning parenchymal tissue remaining. On the other hand, for study of patients with less severe liver disease, less elaborate and time-consuming methods than esterified cholesterol determinations are more satisfactory. As an indication for de-

termination of esterified cholesterol, the presence of jaundice or of a marked elevation of serum bilirubin serves reasonably well.

The cause of the lowered concentrations of serum lipid characteristic of severe liver disease is not known. Contributing factors include (1) the lowered intake of lipid brought about by prescription of diets high in carbohydrate and low in fat for patients with liver disease. (2) Decreased synthesis of fat and of other lipids because of reduced mass of parenchymal cells, impaired blood supply and other related mechanical factors leading to impaired efficiency of biochemical transformations, (3) a diminished supply of supplementary factors such as labile methyl, choline, etc. because of diminished intake and decreased synthesis, these together with other factors, leading to (4) deposition of fat in the liver.

The decreased esterification of cholesterol occuring in severe liver disease also is unexplained. It may be a manifestation of a general decrease in esterase activity since cholinesterase of serum is also depressed. Bollman (1950) has pointed out that the intestinal mucosa is an active site for esterification of cholesterol and that it adds by way of the lymph each day twice the amount of cholesterol ester found in the plasma at any time. The role of the liver may be to supply some factor needed by the intestine for such synthesis. Its regulatory function is unmistakable.

The cause of the elevated concentration of lipid in serum, so typical of biliary obstruction, is also obscure. The bile does not appear to be an important route of lipid excretion. Much of the cholesterol and probably all of the phospholipid excreted in the bile is reabsorbed. Balfour (1947) found that the rate of production of phospholipid was increased in biliary obstruction and that the rise in concentration was proportional to the increase in rate of its production. Rosenman, Friedman and Byers (1952) have postulated that cholesterol concentration in serum rises as a response to an increase in concentration of bile acid (cholate). They believe that changes in concentration of the latter are accompanied by changes in serum cholesterol which serves perhaps to counteract the toxicity of cholate for tissues. Byers, Friedman and Michaelis (1951) find that the liver itself is the source of the extra cholesterol. The close relationship maintained between free cholesterol and phospholipid might then be expected to lead to increased concentrations of the latter.

Information concerning bile acids in liver disease is scanty and unsatisfactory. It is to be hoped that recent improvements in sensitivity of the methods for bile acid determination in serum and bile will overcome this deficiency. Studies of fistula bile following release of biliary obstruction in patients showed cholate to be absent from the bile for a number of days (Ravdin, et al, 1933). The crude methods available in the past for estimating bile acid concentrations in serum indicate that bile acid enters the blood stream to attain concentrations of 10 to 20 mg. per 100 ml. in the presence of biliary obstruction. Elevated values may occur also in liver disease affecting the parenchyma predominantly, (Sherlock and Walshe, 1948).

Methods for study of disturbances of lipid metabolism in liver disease.— Serum cholesterol and cholesterol ester concentrations may be determined by the methods of Abell, et al (1952) or of Sperry and Webb (1950). For phospholipid concentrations the method of Zilversmit and Davis (1950) is convenient. A rapid and simple method for detecting total lipid of serum, the phenol turbidity, has been described by Kunkel, Ahrens and Eisenmenger (1948).

Phenol Turbidity

Principle.— A reagent containing phenol together with sodium chloride in high concentration reacts with serum lipid to produce turbidity which is proportional to the concentration of lipid.

Reagents.— *Phenol-sodium chloride solution:* Dissolve 60 g. of NaCl in about 450 ml. of water. Add 5 ml. of colorless liquefied phenol (90%). Dilute to 500 ml. with water. Store in the cold and discard when it turns yellow. Liquefied phenol is prepared by adding 1 part of water to 10 parts of phenol crystals.

Sodium chloride solution: 0.85 per cent.

Standard:— Colloidal glass suspension as described for thymol turbidity.

Procedure.— Blood is collected from the fasting subject, preferably before breakfast. Measure 5.4 ml. of phenol-sodium chloride solution into a cuvette and place it in a water bath at 25°C. for 5 minutes. Add 0.3 ml. of serum. Mix and replace in the water bath for 30 minutes. Measure the absorbancy at 660 mu.

For setting the zero, use a blank in which 0.3 ml. of serum are added to 5.4 ml. of 0.85% sodium chloride solution.

Calculation.— Same as described for zinc turbidity. Results are expressed in Shank-Hoagland (1946) units.

Interpretation.— Obstruction of the bile ducts, biliary cirrhosis, hepatitis due to poisons, and other conditions in which there is involvement of the biliary tract, such as cholangiolitic hepatitis, are characterized by elevated phenol turbidity.

The phenol turbidity, measured together with thymol turbidity, enables allowance to be made for elevation of the latter caused by high serum lipid.

Healthy subjects have been found to have phenol turbidities averaging 20 units. Values exceeding 34 units are observed in only five per cent of fasting healthy subjects, those exceeding 40 units may be considered definitely abnormal.

Nitrogen metabolism in liver disease.— Patients suffering from liver disease tend to lose more nitrogen than they take in. Impaired digestion and absorption of food proteins may be an important contributing factor in some forms of liver disease because of a decreased secretion of pancreatic enzymes caused by associated involvement of the pancreas.

The liver, the principal site for transformation of amino acids by synthesis, transamination, etc. into those forms specifically needed, carries out such transformations less effectively when diseased or otherwise injured. Thus, increased concentrations of amino acids are found in blood and urine in liver disease. Analyses of the distribution of amino-acids in the urine of patients suffering from parenchymal liver disease by Dunn, et al. (1950) and Gabuzda, et al. (1952) indicate that certain amino acids are increased substantially, among them methionine, tyrosine, and tryptophane, and that others are lowered, lysine, histidine, and isoleucine. Dent and Walshe (1951) describe six different patterns of aminoaciduria occurring in liver disease.

In Wilson's disease (hepato-lenticular degeneration) amino acid output in urine is substantially increased whereas little or no change can be detected in concentrations of blood amino acids (Dent and Walshe, 1951).

Some of the tests employed for study of liver function have as their basis the impaired metabolism of amino acid. Among such is the tyrosine tolerance test of Bernhart and Schneider (1943). One of the factors causing delayed excretion of hippuric acid in Quick's test (1931) is said to be the decreased ability to mobilize glycine for conjugations.

The elevated amino acid concentration in blood contributes to the elevation of non protein nitrogen characteristic of patients with severe liver involvement. Other substances, creatine, creatinine, uric acid, ammonia, as well as undetermined nitrogen, also may be elevated. Urea concentrations may be increased, within normal limits, or decreased in rare instances. The elevation of non-protein nitrogen is mainly a result of the impairment of kidney function. However, urea nitrogen may not be elevated in proportion to the creatinine and NPN concentrations. In part this is due to impairment of the reactions involved in its synthesis. A second factor is the lowered protein intake often prescribed for patients with liver disease. Conclusions concerning the kidney function of patients with severe disease of the liver therefore are likely to be more reliable if based on creatinine or non-protein nitrogen determinations than if based on blood urea nitrogen.

Creatine concentrations are markedly increased in the serum of many patients in hepatic coma (Reinhold, unpublished observations). The impaired efficiency of the metabolism of muscle in such patients with resulting losses of creatine from phosphocreatine is thought to be the main source. Renal failure alone is not responsible.

The liver is the site of numerous reactions concerned with ammonia metabolism.

These include deaminization of amino acids and of adenylic acid, and synthesis of glutamine. Blood ammonia concentrations in hepatectomized dogs were found to rise rapidly (Gordon, Freeman and Farmer, 1952), confirming earlier work by Bollman and Mann (1930). Interest in blood ammonia in liver disease has been revived recently by the finding that patients suffering from cirrhosis who had received ammonia-containing ion exchange resins often showed signs suggesting those of hepatic coma (Gabuzda, Phillips and Davidson, 1952). Blood ammonia concentrations were substantially increased. Kirk (1936) and others previously have shown that the ammonia content of blood is increased in patients with severe liver disease. Seligson (1953) recently reported that not all patients in hepatic coma had elevated blood ammonia concentrations. A report by Nelson and Seligson (1953) showed that ammonia comes from the portal vein, kidneys, and muscles mainly. In shock the liver fails to remove it completely and the blood ammonia level rises.

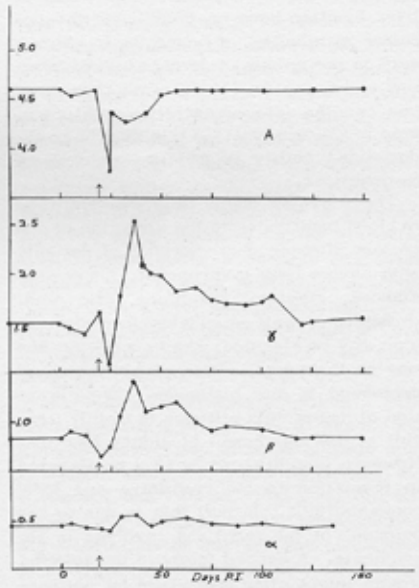

Fig. 2. Changes in serum proteins occurring in a human volunteer during the course of viral hepatitis. At 0 days, this volunteer ingested material containing virus obtained from a patient suffering from infectious hepatitis. The arrow indicates the appearance of clinical symptoms. Note the fall in albumin concentration and the sharp rise in gamma and beta globulin concentrations with gradual return to the original values. Protein measurements were made by the moving boundary method at pH 7.6 in phosphate buffer. (From unpublishes studies by Neefe, Chambers, Gilman, and Reinhold).

Plasma proteins in liver disease.— The liver has a dominant role in plasma protein synthesis. It is the known source of plasma albumin and fibrinogen (Miller, Bly, Watson, Bale, 1951). Probably proteins other than fibrinogen involved in the clotting of blood also originate in the liver. It is the source of important components included in the alpha and beta globulins (Roberts, 1953, Roberts and Brunish, 1953). The liver also is involved in synthesis of gamma globulin although there is much evidence indicating that synthesis of gamma globulin is largely extrahepatic.

Albumin concentrations in serum are lowered in patients suffering from cirrhosis, in viral hepatitis during its clinically active stages, in nutritional liver disease, and in neoplastic disease involving the liver. Many authorities consider it to be among the most dependable measurements available for establishing the presence of liver disease (numerous other causes of low albumin concentration usually can be excluded without difficulty) and for following its clinical course. For this purpose it is superior to total serum protein concentration because changes in albumin are commonly masked by an equal and simultaneous rise in globulin so that total protein remains unchanged. The changes occurring in serum proteins of a volunteer in whom viral hepatitis was induced is shown in Figure 2.

The causes of the decreased albumin concentration include (1) impaired synthesis because of decreased mass of parenchymal tissue (2) losses by transudation into ascitic and edema fluid (3) losses by hemorrhage, a frequent complication of cirrhosis (4) increase in plasma volume (in cirrhosis) (5) low protein intake.

The lowered concentration of serum albumin is one of the factors responsible for the occurrence of flocculation in the cephalin-cholesterol flocculation and related tests. A change in constitution of albumin and alpha globulin probably contributes. A second major factor is a rise in concentration of gamma globulin. Yet another variable is a rise in stabilizing action associated with biliary obstruction (Ducci, 1950). The physicochemical basis for the flocculation and turbidity tests has been discussed recently by Saifer (1952).

An increase in gamma globulin accounts for much of the increase in total globulin of serum. Whereas in healthy individuals gamma globulin reaches a maximum of 1.6 g./100 ml., in liver disease concentrations double this are common, and concentrations five or more times the maximal normal occur in some patients with hepatitis. Zimmerman, Heller and Hill (1951) observed a globulin concentration of 9.9 g./100 ml. in a patient with subacute hepatic necrosis. Values as high as this have been encountered on several occasions in the writer's laboratory.

The marked rise in gamma globulin occurring in liver disease is similar to that occurring in many other diseases. Robertson (1950) has described extremely high gamma globulin concentrations as a manifestation of sensitization to drugs, and Carter (1949) described equally high gamma globulin concentrations as a result of trichinosis. Teilum (1948) considers it to be part of a general response to sensitizing agents. The rise is associated with a marked proliferation of plasmacytes in bone marrow to as much as 25 per cent or more of the total count. Plasmacytes and lymphocytes also accumulate in the liver and other affected areas. These and other observations have led Fagreus (1948), Bjorneboe and associates (1947), and Ehrich (1953) and others to attribute to the plasma cells the formation of gamma globulin. According to their hypothesis the elevation in gamma globulin occurring in response to stressful stimuli, among them liver disease, is a result of the proliferation of this gamma globulin producing tissue. However, Popper (1951), Franklin (1951) and their coworkers have observed changes in the Kupffer and mesenchymal cells of the liver that correlate with increased gamma globulin concentrations in serum. They believe that these cells are the source of the extra gamma globulin. Yet another explanation has been offered recently by Miller (1953), who believes that the diminished synthesis of albumin and other plasma proteins leaves a surplus of amino acids which are converted into gamma globulin outside the liver.

The rise in gamma globulin in many patients suffering from liver disease is accompanied by elevation of beta globulin. Extra protein components not normally observed may appear when electrophoretic separation of serum protein is made at pH 8.6, (Martin, 1946). That most frequently encountered is the H protein of Viollier (1950) observed in infectious hepatitis. Increased gamma$_1$ concentrations were observed by Franklin and associates (1951) frequently in liver disease and rarely in other conditions. These components have mobilities close to that of fibrinogen. Other abnormal components may appear within the gamma and beta fractions.

Fibrinogen appears to be within normal limits or low in the plasma of patients suffering from liver disease (Stefanini, 1949).

Alpha globulins, particularly alpha$_1$, show a tendency toward lower concentrations than those existing in health. Serum mucoprotein has been found by Greenspan et al (1952) to be decreased by hepatitis but increased by metastatic invasion of the liver. Much of the mucoprotein is included within the alpha globulin fraction of the serum protein.

The concentrations of some of the specific proteins included in the alpha globulin fraction also are decreased. Thus serum pseudo-cholinesterase is markedly decreased in many patients with liver disease. Gray, Probstein and Heifetz (1941)

showed also that serum amylase activities are decreased (exceptions occur if there is an associated pancreatitis or impaired renal function). Others have reported lowered lipase and esterase activities.

Methods for evaluating changes in serum protein.— Practical methods for serum albumin and globulin determinations have been described in a recent article (Reinhold, 1953). Measurements of albumin and globulin concentrations are among the most useful available for the study of liver disease. Zone electrophoresis on paper shows great promise as a method for study of serum proteins of patients with liver disease.

Some patients, especially those with liver damage of moderate degree, do not show significant changes in serum albumin and total globulin concentrations. The quantitative measurement of gamma globulin concentrations may offer some advantage in this connection and the salting-out methods of Wolfson, et al (1948) or of Jager and Nickerson (1948) are available for this purpose. De la Huerga and Popper (1949) have described a turbidimetric method for measurement of gamma globulin which is rapid although less accurate than the preceding. However, the most widely used methods for detecting changes of the type occurring in serum proteins in liver disease are the semi-empirical flocculation and turbidity tests. More than a dozen of these have been proposed but techniques for only three, the zinc turbidity, thymol test, and cephalin-cholesterol flocculation will be described in the present paper. For information concerning others and an analysis of their mechanisms, Saifer's (1952) review may be consulted.

Zinc turbidity.

(Kunkel (1947) test for gamma globulin)

Principle.— Gamma globulins are precipitated by zinc ions in buffered solutions of low ionic strength. The concentration of gamma globulin may be estimated by measuring the absorbency of the suspended precipitate.

Reagents.— Zinc sulfate solution: Weigh accurately 0.480 g. of zinc sulfate hydrate ($ZnSO_4.7H_2O$). Transfer it to a volumetric flask of 100 ml. capacity and dilute to volume with freshly distilled water. *Note:* Zinc sulfate hydrate may lose water on standing. Use only well formed crystals.

Zinc barbiturate reagent: Weigh 0.280 g. of barbital and 0.210 g. of sodium barbital. Transfer to a volumetric flask of 1 liter capacity with the aid of sufficient water to dissolve the buffer. Add 5 ml. of the zinc sulfate solution and dilute to the mark with freshly distilled water of low carbon dioxide content. Protect the reagent against uptake of carbon dioxide. Test the pH, which should be 7.60. If it differs by more than 0.05, adjust by addition of dilute sulfuric acid or sodium hydroxide. This reagent keeps a month or more, but should be tested periodically for change in pH.

Standard colloidal glass suspension: Same as described for thymol turbidity.

Procedure:— Measure 6 ml. of zinc-barbiturate reagent into a test tube or cuvette. Place in a water bath at 25°C. for 5 minutes. Add 0.1 ml. of serum using a pipette accurately calibrated to deliver between marks. Do not blow out the pipette. Mix. Stopper and allow to stand at 25°C. for 30 minutes. Measure the absorbency at 660 mu; using the reagent for a zero setting. If the result exceeds 20 units, repeat with 0.05 ml. of serum.

Calculation.— Zinc sulfate turbidity $= U \times \frac{C}{S}$ where U is the absorbency of the unknown, S of the standardized glass suspension, and C the concentration of the standard in terms of Shank-Hoagland (1946) units.

Interpretation.— This method demonstrates markedly elevated concentrations of gamma globulin. It is not sufficiently sensitive to do more than grossly indicate variations occurring within the normal range. Elevated concentrations are the rule in viral hepatitis, cirrhosis of the Laennec type and certain other types of liver disease. However, comparable elevations of gamma globulin concentrations occur in many other diseases and this lack of specificity must be considered in interpreting the results. The zinc turbidity is elevated in a few patients, particularly those suffering from chronic liver disease when the thymol and cephalin-cholesterol flocculation tests are negative. Thus it finds some use as a supplement to these more sensitive tests. The zinc turbidity often is within normal limits when the thymol and cephalin-cholesterol tests are abnormal, whereas the reverse occurs infrequently.

Healthy individuals tested by this method give readings ranging from 3 to 10.5 units (95% limits). Only 1 per cent exceed 12.6 units.

Thymol Turbidity and Flocculation.

Principle.— Maclagan (1944) discovered that thymol in barbital buffer of low ionic strength added to serum produced marked turbidity in the presence of parenchymatous liver disease. In addition, flocculation often appeared on longer standing.

The thymol Barbital reagent is saturated with thymol. Temperature and other factors influencing solubility must be considered in preparing the solution. The procedure described below has been developed recently in the William Pepper Laboratory of Clinical Medicine of the Hospital of the University of Pennsylvania by the writer and Miss Virginia Yonan. It enables better control of thymol concentration and pH and yields a more uniform reagent of improved stability.

Reagents. - Thymol barbital reagent: Transfer 6.0 g. of thymol crystals (colorless) to an Erlenmeyer flask of borosilicate glass with a capacity of 2000 ml. Weigh on an analytical balance 3.09 g. of barbital and 1.69 g. of sodium barbital. Heat 1000 ml. of distilled water to boiling in another flask and boil it for about five minutes to remove carbon dioxide. Allow the water to cool to about 95°, and pour about 300 ml. into the flask on the thymol, which will melt and partially dissolve. Add the barbital and sodium barbital and the remainder of the hot (above 75°) water to the flask containing the thymol. Without delay, stopper the flask and mix by rotating vigorously for about five minutes. Allow the solution to cool gradually to room temperature. Transfer the reagent to a volumetric flask of 1000 ml. capacity and dilute to the mark with distilled water. Return the solution to the original flask. Add about 1 g. of thymol crystals. Shake vigorously until the solution becomes clear. Allow the flask to stand at 25° plus or minus 1° overnight preferably in a constant temperature bath. Mix again and filter through Whatman No. 1 paper, maintaining the temperature of the solution at or near 25° C.

Test the pH which should be 7.55 plus or minus 0.03. The pH may be adjusted by adding 0.1 N NaOH if the reagent is too acid, or by shaking in the presence of a little CO_2 if too alkaline. Expired air may be used as a convenient source of CO_2.

The optimal temperature at which to keep the reagent appears to be 15°, however at a temperature of 25°, the reagent remains unchanged for at least 2 weeks. At temperatures lower than 15° crystals may separate thus causing lower values for thymol turbidity.

Remove sufficient solution for one day's use and place it in a bath at 25°C. at least 30 minutes before using. Exposure to carbon dioxide should be kept to a minimum. The reagent should be renewed when it becomes opalescent.

Standards: Turbidity can be measured either visually or photometrically. The latter is recommended. Colloidal glass suspensions recently have been proposed (Jones, 1951) for use as standards for thymol turbidity measurements. These may be obtained commercially or be prepared according to the following procedure: A one liter borosilicate (pyrex) reagent bottle with glass stopper is filled to about one-fourth its capacity with fragments of clean borosilicate glass. The glass fragments are covered with distilled water and agitated vigorously on a mechanical shaker until a milky suspension is produced. The time varies with the type of shaker, rate and force of oscillation, whether the glass had been shaken previously, and other factors. 8 to 20 hours have been sufficient on an ordinary reciprocal shaker. The suspended glass is decanted into a 1000 ml.

cylinder and diluted to the mark with distilled water. It is mixed and allowed to stand 14 days. The upper 500 ml. is decanted for use as a stock standard.

Standardization.— For this operation a Beckman Model DU spectrophotometer has been used. Probably other instruments of comparable quality will be found to serve as well. A trial reading of stock standard is made in the spectrophotometer at a wavelength of 660 mu.; cuvette depth, 10 mm. Water is used as a blank. Sufficient water then is added to an aliquot of the stock standard to make the absorbency of the diluted suspension approximately 0.100.

The exact absorbency of the diluted suspension then is measured in the spectrophotometer using the conditions specified in the preceding paragraph. A suspension having an absorbency of 0.100, tested in an Evelyn photoelectric colorimeter with a filter of 660 mu. maximal transmission, 6 ml. aperture, and standard reflector was found to be equivalent to 3.3 Maclagan (1944) units and 6.5 Shank-Hoagland (1946) units. The glass suspension may then be used as a semipermanent standard.

Colloidal glass standards deteriorate slowly but may be used for many months if protected against contamination with soluble organic matter.

For visual comparison, the egg albumin-gelatin-formazine standards of Kingsbury, Clark, Williams and Post (1923) were originally used by Maclagan. Satisfactory turbidity standards can be purchased either as colloidal glass suspensions, egg-albumin-gelatin-formazine, or as plastic substitutes for the latter.

Procedure.— Measure 6 ml. of thymol-barbital reagent into a photometer cuvette stopper and place the tubes in a water bath maintained at 25°C. plus or minus 1°C. Add 0.1 ml. of serum from a pipette that will deliver accurately between marks. (Do not blow out). Stopper the cuvette and mix well. In 30 minutes measure the absorbency in a photometer at 660 mu. Use a cuvette containing 6 ml. of thymol-barbital solution for the zero setting. If the thymol turbidity exceeds 20 units, repeat using 0.05 ml. of serum. Multiply the result by 2 when this is done.

Calculation.— Thymol turbidity units $= U \times \frac{C}{S}$ where U is the absorbency of the unknown, S of the glass standard, and C is the thymol turbidity equivalent of the standard.

The thymol turbidity is consistently within the limits of normal in patients suffering from biliary obstruction, if this is of recent origin. Occurrence of elevated thymol turbidity in a patient diagnosed as having biliary obstruction should cause the clinician to re-evaluate carefully the evidence for the diagnosis. On the other hand, lesions of the biliary tract after a month or more may produce sufficient damage to the liver to cause the thymol turbidity to become positive. Even in these circumstances, however, it is unusual for it to be abnormal.

The thymol reagent reacts also with serum lipid and significant elevations of thymol turbidity may be caused by serum lipid (Shay, et al, 1947; Popper, et al, 1948). Such changes probably do not have the same meaning insofar as parenchymal liver disease is concerned as does the "true" turbidity due to the changes in protein. Specimens of blood for thymol turbidity tests collected after a meal rich in fat usually give higher readings than fasting specimens, and blood should be obtained, if possible, with the patient in post-absorptive state. Estimation of serum lipid concentrations by means of the phenol turbidity method of Kunkel has been used to evaluate the degree of lipidemia and thereby to provide some estimate of the extent of the effect of lipid. However, this reagent fails to discriminate between the various lipid components of serum and their inevitably different reactivity with the thymol reagent.

Thymol turbidity may be increased in any disease characterized by marked elevation of gamma globulin. This may occur quite independently of liver involvement. Among conditions which may show elevated thymol turbidity are multiple myeloma, lymphogranuloma, sarcoidosis, parasitic infections, and others. Although liver damage may occur in these and other disease states either as an intrinsic part of the disease or as a complication, it is necessary to obtain confirmation of its presence in these circumstances by recourse to other liver function tests not directly dependent on change in serum protein.

Thymol flocculation.

Flocculation occurs more frequently when the thymol turbidity readings are elevated but it may occur when there is little or no rise in turbidity. Clinical experience also suggests that the occurence of flocculation depends upon some additional or different factors than does the production of turbidity (Neefe, 1946). In general, the significance of a positive flocculation test is the same as for abnormal turbidity. The flocculation test is less frequently positive than is the turbidity test, however, false positive tests are uncommon and the occurence of thymol flocculation thus may be accepted with a higher degree of confidence as evidence of liver damage.

The serum of healthy individuals shows no flocculation. Flocculation graded one plus or more therefore, is abnormal.

Visual measurement.— The comparison is made with standards with light coming from behind the observer. Readings may be made directly from the standards. If egg albumin-gelatin-formazine standards are used, the protein equivalent is divided by 60 for Maclagan units or 30 for Shank-Hoagland units.

Thymol flocculation.— Decant the turbid solution into a conical centrifuge tube of 15 ml. capacity. Stopper and replace in the water bath at 25°C. until the following day. Examine the solution for the presence of a flocculum and note also the extent to which the supernatant has cleared. If flocculation has occurred, it is graded as 4 plus if the supernatant is water clear, or as 1 plus if the flocculation and clearing of the supernatant are minimal. Intermediate degrees of flocculation are designated 2 plus or 3 plus.

The readings may be made quantitative if, after centrifuging, the supernatants are measured in a photometer or comparator.

Interpretation.— Results may be expressed either as Maclagan units or as Shank-Hoagland units. One Maclagan unit equals two Shank-Hoagland units. Ninety five per cent of a group of healthy subjects tested by the writer and his colleagues were found to have thymol turbidities not exceeding 5.5 Shank-Hoagland units; and 99 per cent were less than 6.6 units when a thymol-barbital reagent buffered at pH 7.55 was used. The corresponding values for a reagent buffered at pH 7.80 are 4.4 and 5.5 units. The Commission on Liver Disease of the Armed Forces Epidemiological Board, has recommended that Shank-Hoagland units be adopted in preference to Maclagan units.

The thymol turbidity test ranks high among liver function tests in its ability to reveal presence of liver disease. It is among the tests frequently positive in viral hepatitis, and is particularly useful during the recovery period for evaluation of progress and for detection of carriers of viral hepatitis. Although patients with cirrhosis may show elevated thymol turbidity, often it may fail to become positive in cirrhosis either of the Laennec or biliary type. Mateer and associates (1947) introduced the use of the reagent buffered at pH 7.55, claiming improved sensitivity. This has been confirmed by Neefe and associates (1950). See also Latner and Pendleton (1949).

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A NOTE ON QUANTITATIVE UROBILINOGEN DETERMINATIONS

BY

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The Watson method for the quantitative estimation of urobilinogen in urine and feces in the evaluation of hepatic and hemolytic diseases (1-4) has become widely accepted as a useful tool in clinical practice. The optimum acidity for extraction of urobilinogen from the ferrous hydroxide filtrate, however, has been dealt with vaguely in the literature. Studies on this step of the procedure show it to have a marked influence upon the result.

EXPERIMENTAL

The urine specimens were selected from patients under observation for various forms of liver disease; the stool specimens, from those received in the laboratory for routine parasitologic or bacteriologic examinations.

The method of Schwartz, Sborov and Watson (1) was employed for the urobilinogen determinations except for the establishment of the standard curve using pontacyl dyes which is described by Watson and Hawkinson (4). Readings were made at 565 millimicrons on the Coleman Junior Spectrophotometer Model 6 A.

The critical aspect of this study is concerned with the concentration of acetic acid in urine or fecal samples after the addition of fresh ferrous sulfate and 10% sodium hydroxide which yields final concentrations of 2.5% sodium hydroxide in urine samples and 2.0% in fecal specimens. At this point varying amounts of water and glacial acetic acid were added to the ferrous hydroxide filtrate to make final acid concentrations ranging from 0.1% to 50%. Extraction with petroleum benzine was then carried out in the usual fashion.

RESULTS

Urine: Identical aliquots of ferrous hydroxide filtrate were pipetted into each of several separatory funnels. Water and glacial acetic acid were added to make the final volume 50 ml. and extraction with petroleum benzine, etc., was carried out in the usual fashion. Results of a series of experiments are graphed in Fig. 1. It is apparent that the most complete extraction is realized with a glacial acetic acid concentration of 2%. That this extraction actually is complete was checked by a second extraction made immediately after the first, in which a negligible amount of urobilinogen was recovered (99% transmittance). Similar results were obtained using either 5 or 10 ml. of filtrate for extraction, indicating complete extraction of at least 10 ml. of filtrate.

Determinations of pH over the critical range of 0.5 to 4.0% glacial acetic acid are shown in Table I. A Beckman pH Meter Model G was used for all pH measurements.

Feces: Identical experiments were carried out on feces as described for urine, above. Glacial acetic acid concentrations ranged from 0.1 to 10%. From Fig. 2, it can be seen that the optimum concentration of glacial acetic acid is between 0.5 and approximately 1.5%. Here also similar results were obtained using 5 or 10 ml. of filtrate for extraction.

Determination of pH over the critical range of 0.1 to 3.0% glacial acetic acid are shown in Table I.

The experiments were repeated on five separate samples of both urine and feces yielding identical results in each instance.

TABLE I

Relation between Concentration of Acetic Acid and pH of Ferrous Hydroxide Filtrates

Glacial acetic acid concentration	pH	
	5 ml. of filtrate	10 ml. of filtrate
Urine		
per cent		
0.5	4.4	4.9
1.0	4.0	4.3
2.0	3.7	3.9
3.0	3.5	3.8
4.0	3.3	3.6
Feces		
0.1	6.1	11.6
0.5	4.0	4.4
1.0	3.7	3.9
2.0	3.3	3.6
3.0	3.2	3.4

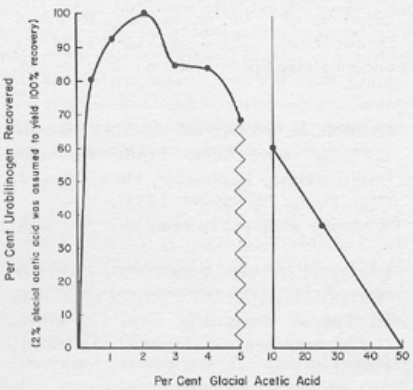

Fig. 1. — Recovery of urine urobilinogen from varying glacial acetic acid concentrations.

DISCUSSION

Various references (2, 3) to the methods for extraction of urobilinogen from urine and feces are somewhat vague as to the specific degree of acidification optimum in the procedure. The importance of this aspect of the method has been a matter of some speculation in this laboratory. We were gratified, therefore, to note that optimum extractions were obtained uniformly when acidification of urine filtrates were held rigidly at the end concentrations of 2.0% glacial acetic acid and fecal filtrates, at 1%. We have, therefore, instituted the following modifications of the standard Watson method referred to above.

Urine: Either 5 or 10 ml. of ferrous hydroxide filtrate is transferred to a spearatory funnel. Water is added to bring the volume to 49 ml. One ml. of C.P.

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QUANTITATIVE UROBILINOGEN DETERMINATIONS

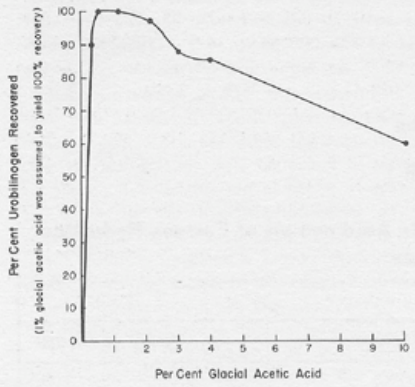

Fig. 2. – Recovery of fecal urobilinogen from varying glacial acetic acid concentrations.

glacial acetic acid is added and the solution is mixed. Petroleum benzine is added and the analysis is continued as outlined by Watson and co-workers (1, 4).

Feces: The ferrous hydroxide filtrate is treated precisely as is urine except that water is added to bring the volume to 49.5 ml. and 0.5 ml. of C.P. glacial acetic acid is added.

The above findings have suggested that additional study of urobilinogen at various pH levels and its possible solubility in organic acids would be of value. Such will be undertaken shortly.

It is not surprising that the optimum acetic acid concentration is less for the fecal ferrous hydroxide filtrate than for the urine since the former is less alkaline than the latter.

The significance of the plateaus in Figs. 1 and 2 between acid concentrations of 3 and 4% is not known.

SUMMARY

A procedure is discussed whereby the optimum extraction of urobilinogen from feces and urine can be accomplished by standardization of acidulation of the ferrous hydroxide filtrates in the Watson method.

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EUROPEAN MEETING

The French Society of Clinical Biology was host to an international group at a meeting at Monaco on May 28-30. The meeting was held in conjunction with the 30th Anniversary of the discovery of anaphylaxis by Charles Richet and Paul Portier. This discovery took place during a voyage on the yacht of Prince Albert of Monaco, patron of the Oceanic Institute of Monaco and Paris.

Twenty-five papers were presented under the following three headings:

1—Anaphylaxis And Allergy. Dr. Kallos (Swedish): Immuno-chimie In Allergy; Prof. Serafini (Rome) and Dr. Halpern (Paris). "The Laboratory Need in Allergic Affections".

2—Laboratories and Diseases Of The Lungs: Prof. King (London): "The Part of the Laboratory in the Diagnosis of Silicosis": Prof. Delarue (Paris): "Anatopathology of Silicosis".

3—Endocrinology: Dr. Azerad and Prof. Agr. Fauvert (Paris): "Comparison of Different Tests of Evaluating Thyroid Function".

REVIEW OF CURRENT LITERATURE

ELLENMAE VIERGIVER – EDITOR
CECILIA RIEGEL, C. VON FRIJTAG DRABBE, HARRY G. ANRODE

CLINICAL EXPERIENCE WITH SERUM-IRON DETERMINATIONS IN THE DIFFERENTIAL DIAGNOSIS OF JAUNDICE. Paul Eckey (Krankenhaus Forst, Lausitz, Ger.) Z. ges. inn. Med. *8*, 21-36 (1953).

Of 52 cases of hepatitis 42 showed an increased serum Fe. In 53 cases of obstructive jaundice only 3 showed an increased Fe. H.A.

DETERMINATION OF IODINE IN BLOOD SERUM. H.F.W. Kirkpatrick (London Clinic) Analyst *78*, 348-53 (1953).

A modification of the method of Chaney is described. Reproducibility and accuracy are demonstrated. The normal range was found to be 3.5 - 8.5 gamma per 100 ml. H.A.

ELECTROPHORETIC PROTEIN FRACTIONATION ON FILTER PAPER. M. Knedel (Univ. Marburg/Lahn, Ger.) Plasma (Milan) *1*, 87-100 (1953) (in German)

A lecture on the method of Grassman and Hannig, practically applied by Knedel. H.A.

DETERMINATION OF TOTAL CHOLESTEROL IN SERUM. Sidney Pearson, Sidney Stern, and Thomas McGavack (New York Med. Coll., New York) Anal. Chem. *25*, 813-14 (1953).

A rapid and simple method for the determination of total cholesterol in serum is described. Close agreement with the Shoenheimer-Sperry method is claimed. H.A.

CHEMICAL ASSAY OF BLOOD COAGULATION FACTORS. Frank D. Mann (Mayo Clinic, Rochester, Minn.) Am. J. Clin. Path. *23*, 623-37 (1953).

A review with 170 references. H.A.

QUANTITATIVE ASSAY OF TRYPSIN WITH THE AGAR DIFFUSION METHOD. E. Speyer (Penicillin Ges. Gottingen, Ger.) Arzneimittel Forsch. *3*, 309-10 (1953).

A method for the assay of trypsin is described which is similar to antibiotic tests on agar plates and is based on the diffusion of trypsin on agar-casein gels. The method is simple and exact and allows detns. of 0.002 units/ml. H.A.

SIGNIFICANCE OF COPPER IN BIOLOGY AND PATHOLOGY. Walter Brenner (Univ. Bonn, Ger.) Med. Monatsschr. *7*, 409-13 (1953).

The relation of Cu and Fe in the serum to certain diseases is discussed. H.A.

PAPER CHROMATOGRAPHY OF PORPHYRIN PIGMENTS. L. Erikson (Univ. Oslo, Norway) Scan. J. Clin. and Lab. Invest. *5*, 155-7 (1953).

An ascending paper chromatographic system is described for the separation of porphyrin pigments using 2,6 lutidine/H_2O (5:3) in the presence of NH_3 vapor. H.A.

BUIRET REACTION IN THE DETERMINATION OF SERUM PROTEINS. Augustin D. Marenzi and Carlos Jorge Gomez. Pubs. centro invest. tisiol. (Buenos Aires) *15*, 245-52 (1951).

It is suggested that fibrinogen or fibrin be used as a reference standard when determining plasma proteins by the buiret method. H.A.

ASSAY OF VITAMIN A AND CAROTENE IN BLOOD SERUM. G. Karmarkar and K. Rajagopol (All-India Inst. Hyg. Pub. Health, Calcutta) Current Sci. (India) *21*, 193 (1952).

The method of Bessey uses a kerosene-xylene solvent for extracting vitamin A and carotene form serum. This solvent often contains impurities which affect the spectral characteristics. Refluxing the solvent over Na then distilling the xylene and kerosene at appropriate temperatures removes the impurities. H.A.

NEW STAINING METHOD FOR SERUM-PROTEIN FRACTIONS IN PAPER ELECTROPHORESIS. Heinz Rottger (Staatsbad, Elster, Ger.) Naturwissenschaften *39*, 451 (1952).

The stain is saturated Ponceau 2R in 50% methanol containing 10% AcOH. Time 10 minutes. The extinction coefficient is proportional to the protein concentration. H.A.

DETERMINATION OF THIAMINE BY THE THIOCHROME REACTION. APPLICATION OF CYANOGEN BROMIDE IN PLACE OF POTASSIUM FERRICYANIDE. Motonori Fujiwari and Kiyoo Matsui (Kyoto Univ.,) Anal Chem. *25*, 810-812 (1953).

3-5 of thiamine in biologic materials can be analyzed by conversion to thiochrome with CNBr and taka-diatase. The solution is passed through a zeolite column and the fluorescence is measured after elution with iso-butanol. H.A.

THE DETECTION OF GALACTOSE IN URINE. F.S. Fowweather. (University of Leeds, England). Biochem. J., *55*: 718-720 (1953).

The Tollens test for galactose is generally unsatisfactory in the presence of xylose or glucose, which produce similar color changes. According to van der Haar [Rec. Trav. chim., *37*:108 (1917)] galactose is the only sugar to form an insoluble o-tolylhydrazone (m.p. 176°).

Fowweather recommends the following procedure. Pipette into a test tube one ml. of urine, previously evaporated if necessary to a sugar concentration of one percent, as determined by analysis. Add 0.2 ml. of a freshly prepared 5 percent solution of o-tolylhydrazine hydrochloride. Dissolve approximately 20-30 mg. of sodium acetate in the mixture and add 10 ml. ethanol. Heat for a few minutes at 90-100° and filter off the precipitate. Evaporate the filtrate at 90° to remove all alcohol. Cool, add one drop of distilled water, mix and examine the drop on a microscope slide. Typical radiating needles of the hydrazone are observed within a few minutes if galactose is present. Glucose may yield osazone crystals after 2-3 hours. Fructose also produces osazone crystals, usually more rapidly than glucose. These may be readily distinguished from galactose o-tolylhydrazone. J.J.C.

URINARY AND FECAL COPROPORPHYRIN EXCRETION IN RATS. III. EXCRETION OF INJECTED COPROPORPHYRIN. F.W. Hoffbauer, C.J. Watson and S. Schwartz (Dept. of Medicine, Minneapolis). Proc. Soc. Exp. Biol. & Med. 83: 238-242, 1953.

Coproporphyrins I and III were administered intravenously to rats. In the normal animal, no increase in urinary excretion occurred; approximately one-half of the injected material was excreted in the feces. In acute liver injury due to carbon tetrachloride, there was an increase in urinary coproporphyrin excretion with a relative reduction in the amount excreted in the feces. In rats with chronic liver injury due to a choline-deficient diet, virtually none of the injected porphyrin could be recovered in either urine or feces. E.V.

ELECTROPHORETIC PLASMA PROTEIN PATTERNS IN FAMILIES WITH HEMOPHILIA. P. Bernfeld, M. Stefanini, R.D. Berkowitz and F.B. Hennessey (Cancer Research and Cancer Control Unit, Depts. of Surgery, Biochemistry, and Medicine, Tufts College Medical School, Boston). Proc. Soc. Exp. Biol. & Med. 83: 311-315, 1953.

A total of 14 individuals with active hemophilia were studied. An electrophoretic anomaly, called α_x-globulin, was found in the plasma of all 14. The same anomaly was also observed in 8 members of hemophilic families who were not active bleeders. The electrophoretic anomaly was seen in females as well as in males. E.V.

PREPARATION OF STABLE HUMAN PROSTATIC PHOSPHOMONESTERASE. M. Davison, I. Asimov, and H.M. Lemon. (Boston Univ. Sch. of Med.) Am. J. Clin. Path. 23, 833-5, 1953.

Human prostatic tissue is extd. with 0.85% NaCl. The supernatant obtd. after centrifugation is dialyzed against water. The supernatant from this is frozen and dried. The lyophilized crude material is fractionated with ETOH and the final ppt. again dialyzed and lyophilized. The preps. varied in activity, the poorest hydrolyzing the equivalent of 26 mg P under the conditions used, and the best prepn. hydrolyzing the equiv. of 75 mg P. Assay for alkaline phosphatase was negative. NaF caused 95% inhibition. C.R.

USE OF PERCHLORIC ACID FILTRATE AND STABILIZED ANTHRONE FOR DETERMINATION OF SERUM GLUCOSE. V. Kapuscinski and B. Zak. (Wayne Univ., Detroit). Am. J. Clin. Path. 23, 784-8, 1953.

Anthrone dissolved in glacial acetic forms a stable solution which may be used either in conjunction with, or without, sulfuric acid to determine glucose in a perchloric acid filtrate of serum, a blue green color being produced which conforms to Beer's Law. C.R.

DETERMINATION OF Hg IN BIOLOGICAL MATERIALS. D.G. Simonsen. Sch. of Med., U. of S. Calif., Los Angeles. Am. J. Clin. Path. 23, 789-97, 1953.

Organic material is removed from blood, urine or gastric contents by digestion with HNO_3 and H_2SO_4 while, at the same time, Hg is converted to the divalent form. In this acid solution, Hg combines with dithizone to form a yellow-orange complex that is soluble in $CHCl_3$. The Hg-dithizone complex, plus any uncombined dithizone, is estimated colorimetrically; the Hg complex is destroyed by an acid-potassium iodide reagent and then the total uncombined dithizone is determined. The difference between the 2 readings is a measure of the Hg dithizonate and hence a quantitative measurement of the Hg present in the sample. The advantages of the method are: as little as 0.2 micrograms of Hg can be detected; can be completed in one hr.; the samples required may be as little as 2 ml of blood, 2 gm of tissue, or 5 ml of urine or gastric contents; the use of acid potassium iodide gives a sharp end point for the reaction and eliminates interference by practically all other metals that react with dithizone at a pH of less than 1. C.R.

A REVIEW OF QUANTITATIVE DETERMINATIONS OF LACTIC ACID IN SERUM AND A NEW PHOTOELECTRIC METHOD. Fritz Kubowitz (Med. Univ. Klin., Berlin) Z. ges. inn. Med. u. Grenzgebiete *7*, 865-82 (1952).

A modified colorimetric method is presented in which $FeCl_3$ is added to the the H_2SO_4 prior to the oxidation of lactic acid by AcH. After the addition of veratrol a stable green color results. H.A.

NEW CLOTTING FACTORS IN PLASMA. S. van Creveld. Maandschr. Kindergeneesk. *21*, 145-53 (1953).

The modern concept of blood clotting is reviewed. H.A.

DETERMINATION OF BARBITURATES. ULTRAVIOLET SPECTROPHOTOMETRIC METHOD WITH DIFFERENTIATION OF SEVERAL BARBITURATES. Leo R. Goldbaum (Army Med. Service Graduate School, Washington, D.C.)

A simple, specific and rapid ultraviolet spectrophotometric method for the determination and identification of various barbiturates is described. The method may be applied to blood, urine, and tissues. H.A.

COMPARATIVE STUDY OF THE DETERMINATION OF URIC ACID IN THE BLOOD SERUM. C. Alvarez Herrero (Inst. espanol med. colonial, Madrid) Med. colonial, Madrid *21*, 524-33 (1953).

The method of Heilmeyer and Krebs (C.A. *24*, 4802) is said to be quicker, more sensitive, and requires less specimen than that of Folin and Wu. H.A.

URINARY AND FECAL COPROPORPHYRIN EXCRETION IN RATS: I. RESULTS IN NORMALS AND CASTRATES. F.W. Hoffbauer, C.J. Watson, and S. Schwartz (Dept. of Medicine, Univ. of Minnesota School of Medicine, Minneapolis). Proc. Soc. Exp. Biol. and Med. 83: 228-232, 1953.

Increased excretion of urinary coproporphyrin has been used as a sensitive index of liver functional impairment. Because of the extensive use of white rats in the study of liver damage, the excretion of coproporphyrin by this animal was investigated. The data obtained using normal animals are presented. E.V.

TUBELESS GASTRIC ANALYSIS BY USE OF INSOLUBLE SALTS WITH EXOGENOUS IONS. H.L. Segal and L.L. Miller (Depts. of Medicine and Biochemistry, the Strong Memorial Hospital, Rochester Municipal and Genesee Hospitals, Univ. of Rochester School of Medicine and Dentistry, Rochester, N.Y.). Proc. Soc. Exp. Biol. & Med. 83: 483-487, 1953.

A method for the determination of gastric acidity without intubation is described. The amount of quinine in the first 2-hour urine excretion after the oral administration of quinine carbonate is used to determine the presence of gastric hydrochloric acid secretion. E.V.

SAFETY OF IMMUNE SERUM GLOBULIN WITH RESPECT TO HOMOLOGOUS SERUM HEPATITIS. R. Murray and F. Ratner (Laboratory of Biologics Control, National Microbiological Institute, National Institutes of Health, Public Health Service, U.S. Dept. of Health, Welfare and Education, Bethesda, Md.). Proc. Soc. Exp. Biol. & Med. 83: 554-555, 1953.

Immune serum globulin produced by the cold-ethanol method from proved infectious plasma failed to produce hepatitis in 10 volunteer subjects inoculated with 2.0 ml. each. E.V.

URINARY AND FECAL COPROPHORPHYRIN EXCRETION IN RATS.: II. RESULTS IN EXPREIMENTAL LIVER DAMADE. F.W. Hoffbauer, C.J. Watson, and S. Schwarz (Dept. of Medicine, Univ. of Minnesota School of Medicine, Minneapolis). Proc. Soc. Exp. Biol. and Med. 83: 232-237, 1953.

Despite the presence of severe liver involvement, no consistently significant increases were observed in the excretion of urinary or fecal coproporphyrin in rats fed a diet or thioacetamide. Average 3- to 4- fold increases, however, were observed in the ratio of urinary to fecal coproporphyrin. Transitory increases in urinary coproporphyrin fecal coproporphyrin ratio following acute exposure to carbon tetrachloride vapor were observed. The injection of lead in rats produced a striking increase in urinary coproporphyrin excretion. E.V.

CHROMATOGRAPHIC SEPARATION OF THE PLASMA LIPIDS. D. Fillerup and J. F. Mead (Atomic Energy Project, School of Medicine, Univ. of California, Los Angeles). Proc. Soc. Exp. Biol. & Med. 83: 574-577, 1953. E.V.

EFFECT OF ACUTE COLD EXPOSURE ON SERUM POTASSIUM, AND MAGNESIUM AND THE ELECTROCARDIOGRAM IN MAN. M. Quinn, D.E. Bass, and C.R. Kleeman (Biochemistry Branch, Quartermaster Climatic Research Laboratory, Lawrence, Mass.). Proc. Soc. Exp. Biol. & Med. 83: 660, 1953.

Clad only in underwear, twenty healthy young men were exposed to a temperature of 45-50°F for a period of 90 minutes. There was a significant elevation in the mean concentration of serum potassium but no significant changes in the mean serum concentrations of serum magnesium, either total or ionized. No correlation was observed between the changes in the electrocardiogram (increase in amplitue of T waves in lead V_4) and the changes in the concentration of serum potassium. E.V.

METABOLIC PRECURSORS OF DEHYDROISOANDROSTERONE. S. Lieberman and S. Teich (Depts. of Biochemistry and of Obstetrics and Gynecology, College of Physicians and Surgeons, Columbia University, New York). J. Clin. Endocrin. 13: 1140-1148, 1953.
Review article. E.V.

BLOOD VOLUME MAINTENANCE AND REGULATION. I.S. Ravdin, James M. Walker and Jonathan E. Rhoads (Univ. of Penna., Philadelphia) Ann. Rev. Physiol. *15*, 165-94 (1953)

A review of methods for measuring blood, plasma, and red cell volumes and the interpretations of results obtained. 189 references. H.A.

THE CARR-PRICE REACTION AND THE REACTION WITH GLYCEROL DICHLOROHYDRIN (For Vitamin A) H. Tastaldi (Univ. Sao Paulo, Brazil) Anals Fac. farm. e ondontol., Univ. Sao Paulo, *8*, 163-77 (1950).

The colors obtained by the Sobel-Werbin method (glycerol dichlorohydrin) or $SbCl_3$ in $CHCl_3$ are less reliable and less intense than the color obtained in the original Carr-Price procedure. H.A.

A SIMPLIFIED METHOD FOR THE PREPARATION OF CORTICOTROPIN. R.J. Bartholomew (Ziskind Research Laboratories, New England Center Hospital, and Dept. of Medicine, Tufts College Medical School, Boston). Proc. Soc. Exp. Biol. & Med. 83: 334-335, 1953.

A simple method is described for the preparation of highly active corticotropin by direct adsorption from a crude extract of sheep or pig pituitaries. E.V.

CHARACTERISTIC INDIVIDUAL ELECTROPHORETIC PATTERNS IN HUMAN. P. Bernfeld, V.M. Donahue and F. Homburger (Cancer Research and Cancer Control Unit, Depts. of Surgery, Biochemistry, and Medicine, Tufts College Medical School, Boston). Proc. Soc. Exp. Biol. & Med. 83: 429-434, 1953.

The detailed examination of the contours and fine architecture of the electrophoretic patterns of human plasma in different individuals may reveal considerable dissimilarities between persons, even in those cases where the relative amounts of plasma components are similar. These dissimilarities are discussed in detail. E.V.

A COLORIMETRIC METHOD FOR HYALURONIC ACID ESTIMATION. W.A. Pierce, Jr., R.H. Steele, and A.G.C. White (Depts. of Microbiology and Biochemistry, Tulane Univ. School of Medicine, New Orleans). Proc. Soc. Exp. Biol. & Med. 83: 373-375, 1953.

A new colorimetric method for the determination of hyaluronic acid is described, based upon precipitation of acid polysaccharide with hemoglobin at pH 3.8 and conversion of the prosthetic group of the precipitated protein to ferriheme cyanide. E.V.

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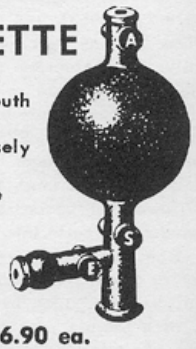

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Volume 1, Number 1 of this bimonthly publication is scheduled for release in February 1954. Among the papers to be published in the early issues are a group devoted to radiation chemistry (with A. O. Allen, E. S. G. Barron, H. A. Dewhurst, W. M. Garrison, J. L. Magee, H. R. Raymond, A. H. Samuel, and B. M. Weeks as authors), radiation biology and medical research (with L. H. Gray and Raymond E. Zirkle as authors) a Symposium on Physical Measurements in Radiobiology (with papers by G. Failla, U. Fano, Payne S. Harris, L. D. Marinelli, and Burton J. Moyer).

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The third volume in the "Biochemical Preparations" series was published this month by John Wiley & Sons, 440 Fourth Avenue, New York 16, N.Y.

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Volume 33 in the well-known "Organic Syntheses" series was published in September by John Wiley & Sons.

This latest addition to the annual publication supplies the most convenient methods for the preparation of 40 reactions, contributed by 64 chemists. In accordance with established procedure, each reaction has been thoroughly checked in the laboratory before publication.

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" "Organic Syntheses," Volume 33, contains 115 pages and is priced at $3.50.

SOUTHERN CALIFORNIA SECTION

Robert J. Foster, Ph.D., Research Fellow, Kerckhoff Biological Laboratories, California Institute of Technology, discussed "Enzyme Kinetics", October 6-meeting at the Cedars of Lebanon Hospital, Los Angeles.

M. E. Morton, M.D., Ph.D., Chief, Radioisotope Unit, Long Beach Veterans Administration Hospital, spoke on his work on the "Determination and Interpretation of Serum Thyroxin Levels", November 3 at the Hollywood Presbyterian Hospital, Los Angeles.

Robert M. Fink, Ph.D., Research Biochemist, Department of Investigative Medicine, Long Beach Veterans Administration Hospital, discussed "Chromatographic Techniques", at the December meeting held at the Los Angeles County Hospital.

In his September 1 address at the Los Angeles County Hospital, Joseph R. Goodman, Ph.D., Biochemist, Long Beach Veterans Administration Hospital, spoke on the fundamental principles in the operation of the analytical centrifuge. The derived equations of Svedberg were given, and general theories were discussed. Considerable time was devoted to the description of the operation of the Spinco models, and some of the problems and techniques of evaluation were discussed. The analytical spinning head and the cells were exhibited and their functions described. The methods and techniques of Gofman's lipoprotein analysis were presented, and the limitations and implications of this kind of data were mentioned. The talk was followed by a discussion period which was led by Kenneth Johnson who has been working on ultracentrifugal problems at the California Institute of Technology.

GUIDE TO ETHICS GOVERNING THE CONDUCT OF CLINICAL CHEMISTS

A tentative Code Of Ethics was published last year (C.C. 4 July 1952) so that the AACC membership would study the articles and contribute suggestions. After reviewing all suggestions and revisions, the articles and sections were approved by The National Executive Committee, September 1953. The following is now the official Code Of Ethics of the American Association Of Clinical Chemists, Inc.

Article I

DEFINITIONS AND GENERAL CONSIDERATIONS

WHAT CONSTITUTES CLINICAL CHEMISTRY

Section 1. Clinical chemistry is that branch of chemistry which deals with the composition of the secretions, excretions, concretions and fluids of the human body in health and disease, and the chemical composition and metabolism of cells and tissues. Also the search for the presence of substances (or their derivatives) given for diagnostic or therapeutic reasons and the search for poisons (or their derivatives) are properly included in the field of clinical chemistry.

WHAT CONSTITUTES A CLINICAL CHEMIST

Section 2. Any individual equipped by education and experience to engage in the practice of clinical chemistry as defined above shall be considered a clinical chemist.

RESPONSIBILITY OF THE CLINICAL CHEMIST

Section 3. The profession of clinical chemistry, as an adjunct to the profession of medicine, has as its ultimate responsibility the welfare of the public. The clinical chemist shall use to the best of his ability his scientific skills and knowledge to the benefit of all men without regard for racial or religious origin.

EDUCATION AND EXPERIENCE

Section 4. The clinical chemist shall have as his goal the acquisition of the best available education and experience in chemistry. He shall strive to constantly enlarge and improve his knowledge.

RELATIONSHIP TO THE MEDICAL PROFESSION

Section 5. The clinical chemist shall deal with the medical profession at all times at the highest professional level. The compensation by the patient for chemical services shall include no rebates or commissions to any persons for solicitation or referral of analyses.

RELATIONSHIP TO THE PATIENT

Section 6. The clinical chemist shall perform no services to the patient except on advice or prescription from any licensed practitioner of the medical arts. All reports and discussion of chemical findings shall be only between the chemist and the physician in charge.

Article II

PUBLICATION, PATENTS, AND ADVERTISING

DISSEMINATION OF SCIENTIFIC INFORMATION

Section 1. The clinical chemist shall freely discuss with his fellow chemists and with scientists in related fields, advances in the science of clinical chemistry. To withhold information for personal gain shall be considered unethical. This Section shall not apply to information classified by a government agency for reasons of national security.

PUBLICATION OF RESEARCH FINDINGS

Section 2. An obligation to publish, after critical evaluations, new knowledge pertaining to the science of clinical chemistry obtained through research or other observations, shall be acknowledged.

ADVERTISING AND PUBLICITY

Section 3. The clinical chemist shall not use, or allow his name to be used, in advertising directed to the public. Professional announcements shall be brief, dignified and consistent with accepted customs in medical and allied fields. The clinical chemist shall not seek publicity, yet he shall recognize the right of the public to have access to information concerning the public health and welfare. Publication of a scientific article or book shall precede the release of such material to the lay press. Because of the danger of misinterpretation, he must use restraint and great caution in releasing information having diagnostic or therapeutic implications.

PATENTS AND COMMISSIONS

Section 4. The application of discoveries and developments in Clinical Chemistry, directly effecting public health and welfare, should not be limited by unreasonable restrictions for personal gains to the Clinical Chemist.

Article III

OBLIGATIONS AS A CHEMIST

ACCURACY OF CHEMICAL ANALYSES

Section 1. The clinical chemist shall have as his goal the attainment of the highest precision and specificity that existing procedures permit.

REPORTING OF SIGNIFICANT FIGURES

Section 2. The analyst shall not report figures or decimal places that lack significance.

CRITICAL SURVEY OF METHODS

Section 3. It shall be considered inadequate practice for a clinical chemist to use any procedure that has not been adequately studied in his own laboratory.

REPLICATES AND RECOVERIES

Section 4. The systematic use of controlled procedures, such as replicates and recoveries, shall be considered indispensable to good practice.

RESULTS OF UNCERTAIN MAGNITUDE

Section 5. The clinical chemist shall not report any result of uncertain magnitude of error, unless this uncertainty is clearly made known to the recipient of the report.

ARTICLE IV

INTERPRETATION OF RESULTS
RELATIONSHIP TO THE PHYSICIAN

Section 1. The clinical chemist shall, at the request of the physician in charge of the patient, outline to the physician the significance of any chemical findings, and suggest further determinations that would aid the physician in making a diagnosis or prognosis. The clinical chemist shall under no circumstance transmit to the patient either the results or the interpretation of the results. The clinical chemist shall receive no compensation from the patient for interpretation of results to the physician.

Article V

THE CLINICAL CHEMIST AS AN INDIVIDUAL

A SCIENTIST AT ALL TIMES

Section 1. The clinical chemist shall conduct himself as a scientist at all times.

HIGH REGARD FOR MEDICAL PROFESSION

Section 2. The clinical chemist shall hold in high esteem the profession of medicine, to which he is an adjunct.

RELATIONSHIP WITH ANALYSTS

Section 3. The clinical chemist shall carefully supervise the analysts working in his laboratory. He shall train these workers to the best of his ability, encourage them to attain the highest professional competence, and teach them by word and example to adhere to the ethical standards herein outlined.

PUBLICATIONS AND COLLABORATORS

Section 4. The clinical chemist shall contribute as much as possible to research and advancement of his specialty. He shall encourage those working in his laboratory to do likewise. He shall accept as collaborators whenever possible the junior members of his staff and encourage these members to contribute to the science of clinical chemistry. He shall to the best of his ability assist physicians and other scientists by fully collaborating in their efforts to advance medical science.

Article VI

CONCLUSION

The ethics of the clinical chemist shall at no time be inferior to the standards long prevailing in the medical profession. The outline here presented can act only as a general guide, and shall be periodically reviewed and revised. It is for the individual to judge his professional conduct in the light of his obligation as a scientist to serve mankind.

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Joseph I. Routh, Ph.D.
Professor, Biochemistry
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At the recent Annual Meeting, Professor Routh consented to initiate the Association Employment Clearing House, and will keep an active file of all employment opportunities.

BOOK REVIEWS

ORGANIC ANALYSIS, VOL. I. Edited by J. Mitchell, I. M. Kolthoff, E. S. Proskauer, and A. Weissberger. 473 Pages. Interscience Publishers, Inc. 250 Fifth Ave., New York 1, N.Y. $8.50—*Reviewed Norman P. Salzman. Section on Chemical Pharmacology. National Heart Institute.*

This book is the first in a series that has as its goal the consolidation of information in organic quantitative non-elemental analysis. In this first book, sections are devoted to methods for the quantitative determination of hydroxyl, alkoxyl, alpha-epoxy, carbonyl, acetal and organic sulfur groups. In addition, discussions are included for the determination of active hydrogen by organometallic compounds and diazomethane and the use of spectroscopic functional group analysis in the petroleum industry. Each section includes a brief discussion of the various procedures available for the particular determination. This is followed by detailed discussion of the more important methods, the laboratory procedure and a description of the apparatus. Micro as well as macro techniques are discussed.

The best feature of this book is the critical evaluation of the various procedures showing in detail the limitations of the methods and the modifications that the authors found necessary in the application of the procedures in their own laboratories.

This book should prove of value as a critical source of information on quantitative procedures for both the organic and analytical chemist.

CORRECTION

In reporting the lecture "The Chemical Detection Of Barbiturates And Physiological Antagonists To Barbiturates" by Dr. Theodore Koppanyi (C.C. *5*, 75 (1953)), attention has been called to the following addenda.

Under *Procedure:* the third sentence should read *"The cobaltous acetate and lithium hydroxide reagents are added to each of the three test tubes: 0.05 cc to A, 0.1 cc to B and 0.15 cc to C."*

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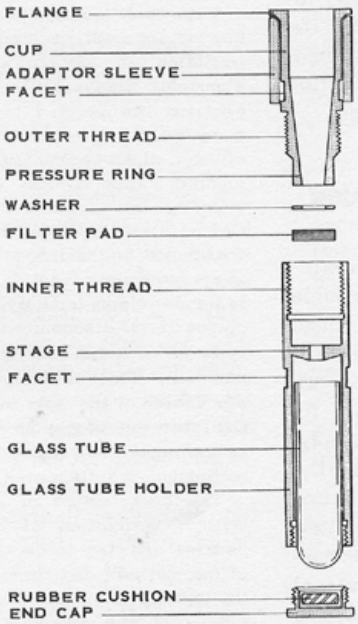

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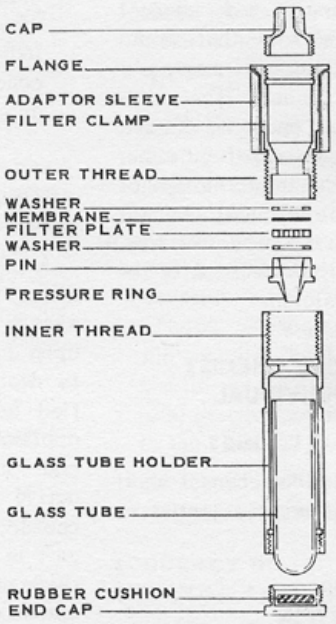

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