The mechanism of the production can at the moment only be surmised. It seems probable that bacterial toxins, or the organisms themselves, are carried by the portal vein to the liver, giving rise to a hepatitis. Ottenberg and Spiegel (1943) stated that jaundice was an occasional symptom in acute septicaemias, and was usually of unfavourable prognostic significance, with the pneumococcus, haemolytic streptococcus, anaerobic staphylococcus, and *Clostridium welchii* as the commoner organisms responsible. Haemolysis and the effect of bacterial toxins were both usually responsible. Those authors thought that the histological changes in the liver bore no relation to the degree of jaundice. On purely physical grounds peritoneal sepsis would be expected to cause jaundice more often than sepsis elsewhere, owing to the direct drainage of the septic area, often of great extent, direct into the liver.

Another possibility is that the associated ileus and circulatory disturbance give rise to abnormal and toxic gut contents able to affect the liver. Other factors which must be considered are a diminished oxygen content in the portal blood due to stasis, because of the associated ileus, anaemia, and shock, all leading to anoxaemia of the liver. Neither anaemia nor shock played any part in these cases.

The incidence of septic jaundice, compared with that of peritonitis, suggests that the resistance of the liver to the effects of sepsis is high. Although in most of the cases in this series the extent of the sepsis was considerable, equally severe cases of similar duration have not developed jaundice. In the absence of proof to the contrary, however, it seems reasonable to assume that, all other factors being equal, the greater the amount of sepsis the more likely is the development of jaundice. The effects of different organisms in causing it remain to be investigated. The duration of sepsis does not seem to be a factor, since most of these cases occurred early in the post-operative period.

The pre-operative state of the liver may be of importance, but in these cases there was no clinical evidence pre-operatively or pathological evidence at necropsy to suggest any predisposing disease.

Summary

Six cases of jaundice occurring after gastro-intestinal operations complicated by peritonitis are described. They form a group for which the name septic jaundice is proposed.

The onset of jaundice was early, usually within the first week of operation, and it persisted until death or until the infection subsided. Signs of hepatic parenchymal failure were not observed. The thymol flocculation and turbidity tests and the serum colloidal gold reaction were consistently negative, and the serum alkaline phosphatase was normal. Treatment and prognosis are those of the peritonitis.

The post-mortem appearances and the liver histology are described; two non-jaundiced cases of fatal peritonitis are included in this section. In some of the cases the liver showed a zone of subcapsular necrosis bounded by a zone of intense congestion of the sinuses, and a layer of pus on the capsule.

The differential diagnosis and the possible factors affecting the liver in this condition are discussed. Severe peritoneal sepsis is thought to be the cause of the condition.

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VITAMIN K IN THE TREATMENT OF ACUTE HEPATITIS

BY

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Since Dam (1935) described vitamin K and Dam and Schönheyder (1936) defined the part it plays in the maintenance of a normal prothrombin level in the blood numerous papers have been published on vitamin K and prothrombin in animals and man. Since 1935 many authors (Hult, 1939–41, among others) have investigated the effect of vitamin K on the prothrombin level in acute and chronic diseases of the liver; and acute hepatitis has also been a subject for research. It was observed that vitamin K administration in these cases exerted a varying influence on the prothrombin level (Butt, Snell, and Osterberg, 1939; Hult, 1940). Begtrup and From Hansen (1942, 1948) found that patients with parenchymal liver damage mostly showed only a slight increase of the prothrombin level after administration of vitamin K. Patients with obstructive jaundice, on the other hand, responded well to the vitamin.

There are several causes of a low prothrombin level. Severe parenchymal liver damage may retard prothrombin formation by the liver, or the absence of bile salts in the intestinal tract in cases with an impeded bile flow may result in a deficient absorption of vitamin K, necessary in some way for the production of prothrombin. The relative extent to which a deficiency in prothrombin formation is due either to liver damage or to defective absorption of vitamin K remains to be determined.

Present Investigation

From January to the beginning of August, 1950, every second case admitted to hospital for suspected hepatitis was on the day after admission put on vitamin K treatment,* one tablet (0.01 g.) three times a day by mouth. The other cases served as controls and were given a placebo.

The material consisted of 247 cases: 178 of acute hepatitis; 5 of subacute hepatitis; 7 of chronic hepatitis or cirrhosis of the liver; 27 of cholecystitis or cholelithiasis (15 stayed two weeks or more in hospital); 13 of carcinoma of the pancreas or the papilla of Vater or neoplasms of the extrahepatic bile ducts; and 17 with various non-hepatic diseases.

The two groups of acute hepatitis consisted of 87 cases in the K (vitamin-K) series and 91 in the C (control) series. The distribution in age groups was practically the same. No attempt was made to divide the material into epidemic and

* "K-vimin" (water-soluble, 2-methyl-1,4-naphthoquinone tetrasodium diphosphate). Astra, Södertälje, Sweden.

inoculation hepatitis. The blood and urine of all patients were tested once a week: icterus index (Meulengracht), thymol turbidity, and prothrombin level (Quick) of the blood; and bilirubin (Hammarsten), bile acids (Hay), and urobilin (Schlesinger) in the urine.

As a rule, the patients did not leave the hospital before the icterus index had returned to 10 (Meulengracht). Since, however, some were discharged earlier, the value of 15 (Meulengracht) or less was taken as a limit in judging the effect of the treatment. Only 2 of the 178 patients were discharged with a higher value. A note was made of the highest icterus index and the time in weeks for a return to 15 or less. In the same way the highest thymol reaction value was calculated, as well as the value at the discharge (see Table I). For bile pigments, bile acids, and urobilin

TABLE I.—Hospital Stay, Icterus Index (Meulengracht) and Thymol Reaction in the Blood

	K Series (87 Cases)	C Series (91 Cases)
Hospital stay, days	33.4	30.7
Meulengracht:		
Highest value	59.6	52.7
Increase after admission (cases)	21.0	19.0
Time in weeks for decrease to 15 or less	3.2	2.6
Thymol:		
Highest value	13.2	13.7
At discharge	8.0	8.9

in the urine the time in weeks till negative tests were obtained was noted (see Table II). It will be seen that no statistically significant differences were obtained between the K cases and the controls.

TABLE II.—Bilirubin, Bile Acids, and Urobilin in the Urine

	K Series (87 Cases)	C Series (91 Cases)
Bilirubin (Hammarsten):		
Time in weeks pos.→neg. ..	1.2	1.2
Cases pos. at discharge ..	0	2=2.2
Bile acids (Hay):		
Time in weeks pos.→neg. ..	1.4	1.3
Cases pos. at discharge ..	1=1.1	6=6.6±2.6%
Urobilin (Schlesinger):		
Time in weeks pos.→neg. ..	2.4	2.4
Cases pos. at discharge ..	7=8.1±2.9%	12=13.2±3.6%

The prothrombin content is placed in relation to the icterus index. The cases are divided according to the icterus index (Meulengracht less than 40, 40–79, and 80 and over). In cases of a mild or rather fairly severe hepatitis (Meulengracht less than 40 or 40–79) no difference was noted in the prothrombin content of the blood in the C and the K series during the stay in hospital (Fig. 1). However, in cases of severe hepatitis (Meulengracht 80 and over) a difference occurred (Fig. 2). The K series showed an increasing average prothrombin content, in spite of an increasing icterus index, as early as one week after admission. The C series, on the other hand, showed a decreasing average prothrombin con-

FIG. 1.—Mild and fairly severe acute hepatitis.

FIG. 2.—Severe acute hepatitis.

tent and increasing icterus index during the first week. Coincidentally with a subsequent decrease in the icterus index, the prothrombin content during the following weeks slowly rose to the values of the K series. The greatest difference between the series was in the second and third weeks. The differences— 12.6 ± 5.1 and 12.9 ± 5.1 , respectively—are statistically significant.

The urinary urobilin test (Schlesinger's reaction) indicates the degree of excretory disturbance in the liver. If no bile, or only an inconsiderable amount, reaches the intestine the urobilin reaction will be negative. In severe hepatitis in both the K and the C series this was noted in about 65% of cases on admission; therefore the absorption of vitamin K from the intestinal tract must have been considerably impeded. The good response of the blood prothrombin level to water-soluble vitamin K in the K series suggests that, in severe hepatitis, the disturbed absorption of vitamin K is probably the most important cause of the decrease in the prothrombin level. The significance of parenchymal liver damage in these cases is discussed later.

The cases of obstructive and non-acute hepatic jaundice are divided into three groups as follows: Group 1, cases of carcinoma of the pancreas or the papilla of Vater, or neoplasms of the extrahepatic bile ducts without metastases in the liver; Group 2, cases of cholecystitis or cholelithiasis; and Group 3, cases of chronic hepatitis or cirrhosis of the liver.

Fig. 3 shows the good effect of vitamin K on the blood prothrombin level. The icterus index is roughly the same in both series. The prothrombin level in Groups 2 and 3 is shown in Fig. 4. In both groups the average value was normal on admission—high normal in Group 2 (healthy liver) and a low normal in Group 3 (damaged liver). Neither group showed any effect of vitamin-K treatment. The low normal values in the cases of chronic hepatitis and cirrhosis

FIG. 3.—Changes in prothrombin content and icterus index in cases of obstructive jaundice (carcinoma of the pancreas or the extrahepatic bile ducts).

FIG. 4.—Changes in prothrombin content and icterus index in cases of cholecystitis and cholelithiasis (healthy liver) and chronic hepatitis and cirrhosis of the liver (damaged liver).

of the liver may indicate some disturbance in prothrombin formation by the liver. The similar good effects, however, of vitamin K in acute hepatitis and obstructive jaundice show that the parenchymal damage in acute hepatitis affects the prothrombin-producing capacity of the liver only slightly.

Summary

An investigation is made into the effect of oral vitamin K on cases of jaundice, especially those due to acute hepatitis.

Vitamin K did not affect the course of the disease in 178 cases of acute hepatitis. When compared with controls the effect on the blood prothrombin level was obvious only in cases of severe acute hepatitis (Meulengracht 80 and over). In these instances the effect of the oral vitamin was good.

A comparison between treatment of acute hepatitis and of obstructive jaundice failed to show any difference in the effect on the prothrombin value. This indicates that even in cases of severe hepatitis the liver damage does not seriously affect the prothrombin-producing capacity of the liver. Furthermore, it shows above all that decrease of the prothrombin value results from defective vitamin-K absorption in the intestinal tract.

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The annual report for 1951 of the Greenwich and Deptford Hospital Management Committee gives some figures to show the amount of work being done by the typical general hospital. St. Alfege's Hospital, Greenwich, has 504 beds, of which only 366 are staffed and open. In addition, the committee controls the 180 beds of the Miller General Hospital, so there was a total of 684 general beds available, receiving over 10,000 in-patients in the year. The operating-theatres performed nearly 9,000 operations (about 5,000 of them major), and there were 867 live births in the maternity unit. Thus each bed was occupied on the average by 15 patients in the year, for an average stay in hospital of just over three weeks each. Among the work of the special departments may be noted 34,798 x-ray examinations, 52,480 blood and other specimens investigated in the pathological laboratories, and 4,142 E.C.G.s taken. Some of this work was, of course, for out-patients. Just under 26,000 new patients were seen here, and there were over 58,000 attendances in the casualty departments.

STRESS INCONTINENCE

A PRELIMINARY REPORT ON A NEW METHOD OF TREATMENT*

BY

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For many years I have been interested in stress incontinence. I have practised most of the methods of repair: anterior colporrhaphy, "buttressing" of the urethra, sling procedures such as Aldridge (1942), Studdiford (1944), Millin I (Millin, 1947) and II (Millin, 1950). So many times has this subject been debated recently and so many different operations have been advised that I almost hesitate to raise the matter again, but my dimming embers of interest were fanned afresh by still another operation, Mulvany (1951). One must pay close attention when a surgeon summarizes his work as follows: "A simple operation, vesico-urethrololysis, for the cure of stress incontinence in women, is described. In a series of 58 cases extending over a period of three years bladder function was restored to normality in all by the operation, and the improvement has since continued unchanged." My immediate reaction to this summary, not an unreasonable one, was one of distrust: I thought that the sooner confirmation or rejection was made, so much the better.

Mulvany's Operation

Mulvany's operation consists in mobilization of the bladder and urethra through a small transverse suprapubic incision and traversing of the retropubic space. The bladder is freed from the back of the symphysis pubis and its lateral supports are also mobilized. Mulvany thinks it likely that the degree of freedom afforded to the sphincter area by manipulations required to prepare the bladder-neck site for the insertion of urethropexy or bladder-raising sutures permits the sphincter muscles to retract and thereby regain normal function. This mobilization of the bladder-neck is a step common to all sling procedures. It is also reasonable to say, although Mulvany does not say so, that while the baby's head is coming through the pelvis the bladder-neck is pressed against the back of the symphysis pubis, and, because of the rich venous plexus there, haematomata may result, giving rise later to adhesions. These are very real arguments in favour of Mulvany's vesico-urethrololysis.

Personal Results

I have performed this mobilization in 23 cases, but have not always used Mulvany's suprapubic approach. In some cases I used the Millin II incision between the external urinary meatus and clitoris to reach the retropubic space. In other cases I have mobilized the bladder off the back of the symphysis through an anterior vaginal wall incision, and then have passed a finger up either side of the urethra through the pudendal diaphragm. I must stress that mine are immediate results, and not long enough has elapsed to assess late cures. I therefore regard this as very much a preliminary report.

Out of 23 cases 21 have been immediately cured. One of the failures was an early case which was done through the Millin II approach and in which a tight band of fibrous tissue, resulting from a previous colporrhaphy, was not cut through. I know now that if I encountered a similar condi-

*Part of an address to the Birmingham and Midland Obstetrical and Gynaecological Society, March 26, 1952.