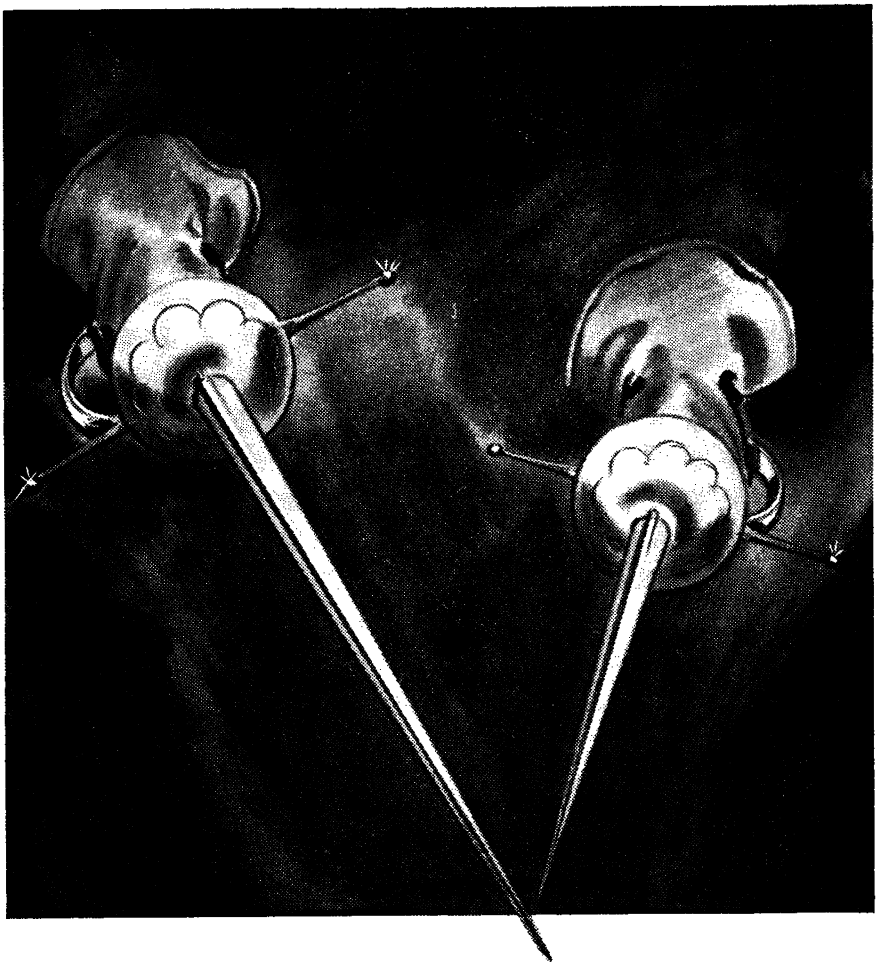
MAY, 1956

THE ULSTER MEDICAL JOURNAL

PUBLISHED BY
THE ULSTER MEDICAL SOCIETY



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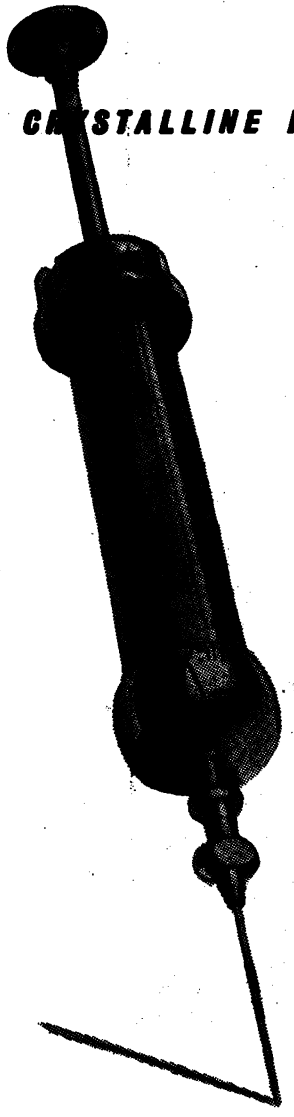

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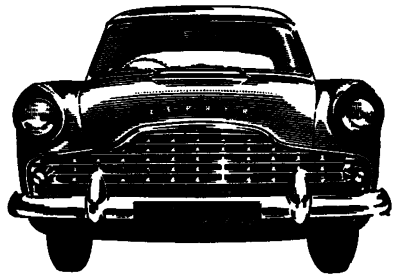

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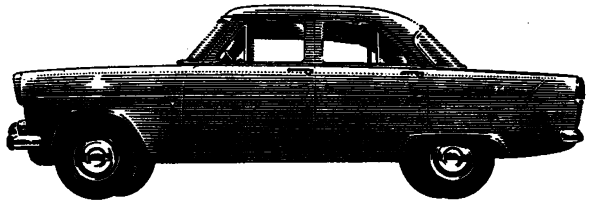

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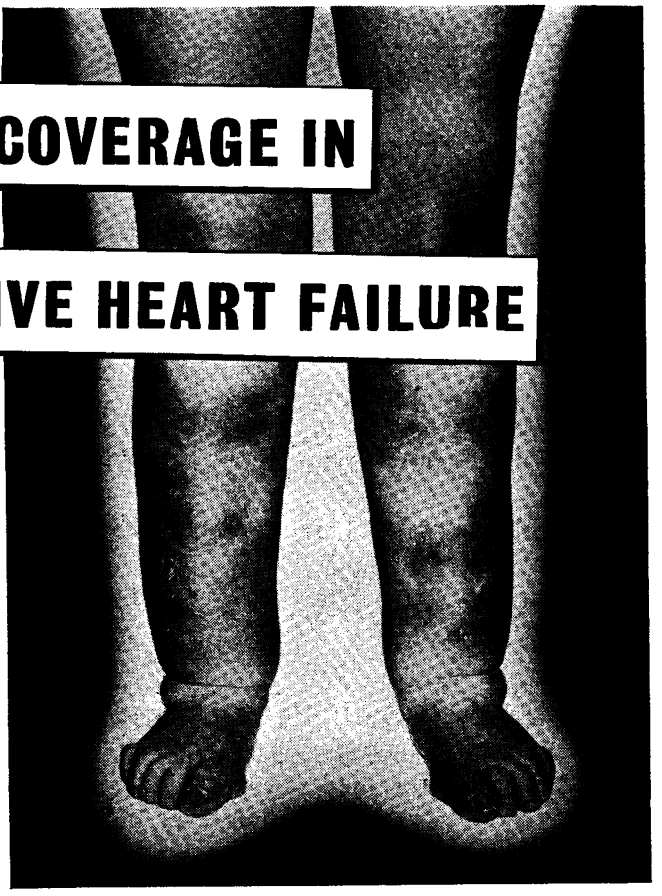

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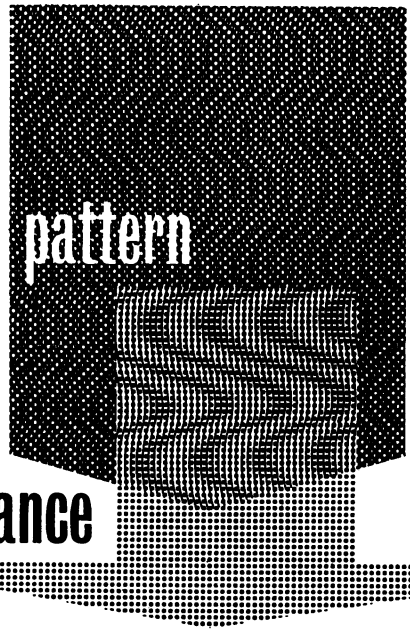

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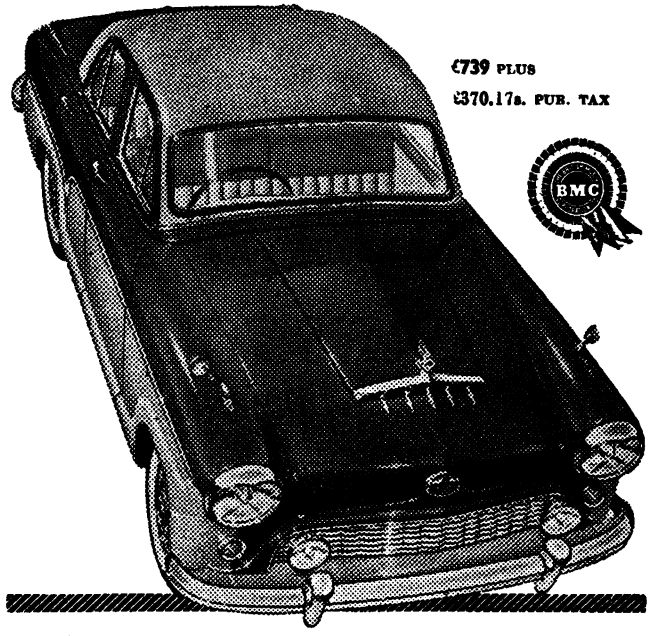

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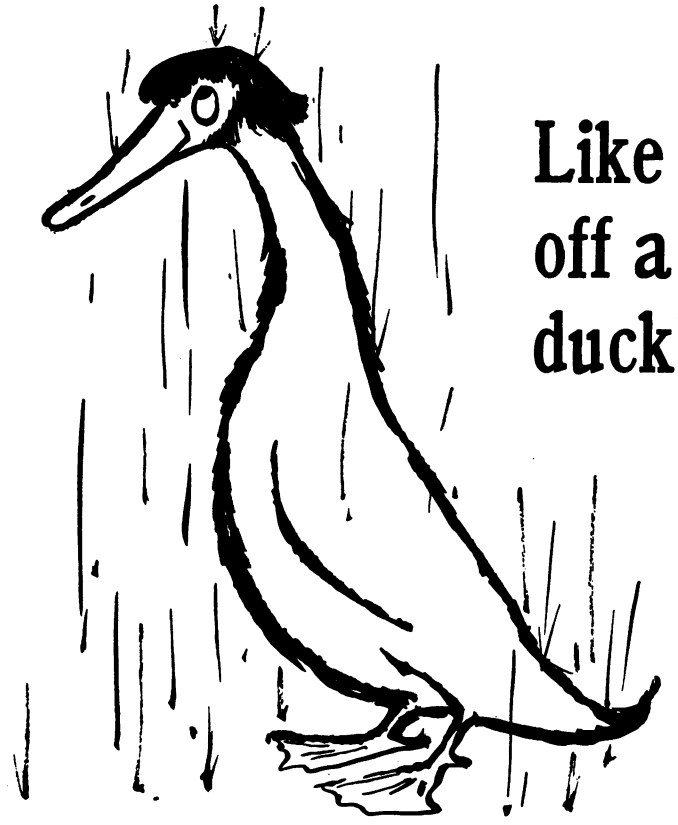

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DATES OF PUBLICATION

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THE
ULSTER MEDICAL JOURNAL

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Vol. XXV 1st MAY, 1956 No. 1

The Clinical Value of Gastric Secretion Tests

By BENGT J. E. IHRE, M.D.(STOCKHOLM)
Physician in Charge, St. Erik's Hospital, Stockholm

A Lecture delivered at the Queen's University of Belfast on October 31st, 1955, under the auspices of the British Council.

YOU all know the story about William Beaumont, the army surgeon, who took care of Alexis St. Martin, the young French Canadian, who was severely wounded by an accidental discharge of a shotgun, which left him for the rest of his life with a hole in his side, through which his stomach could be inspected. William Osler, in an address in 1902, gave a fascinating picture of how the man and the opportunity meet—and match. The opinions on gastric physiology in the beginning of the nineteenth century were so confusing that we understand William Hunter's pithy remark, "Some physiologists will have it, that the stomach is a mill, others that it is a fermenting vat, others, again, that it is a stew-pan: but, in my view of the matter, it is neither a mill, a fermenting vat, nor a stew-pan; but a stomach, gentlemen, a stomach."

Beaumont's observations, published in 1833, anticipated our present knowledge of gastric function. His observations on gastric secretion and on the gastric mucosa were of such fundamental importance that very little was added before the works of Pavlov.

However important Beaumont's observations were, they had very little influence on the opinion concerning gastric diseases of his contemporaries, who were rooted in the anatomical conception of gastritis, as being the most common cause of stomach trouble. This conception had been presented by Broussais, the Frenchman, and was based on post-mortem findings, which later were shown to be due to an erroneous interpretation of post-mortem putrification.

The introduction of the stomach tube as a routine diagnostic aid (Kussmaul, 1867) and the test meal of Ewald-Boas brought on a change of this anatomical view on gastric diseases. The interest during the last decades of the nineteenth century with regard to gastric disorders was mainly focused on the diagnosis of

disorders of gastric secretion. Particularly on the Continent, with the German clinicians en tête, the diagnosis of different forms of secretion disturbances flourished, such as heterochylia, functional achylia, and hyperacidity, which were regarded as responsible for the various symptoms of which the patient complained.

The development of gastric surgery and the beginning of the rœntgen era gave rise to a critical reaction against these diagnostic subtilities. In 1923 Moynihan, the famous English surgeon, made the following comment regarding the Ewald test meal: "I found that so little information was given, at so great an expense of time and trouble, both to the patient and to ourselves, that I was glad to abandon the procedure altogether."

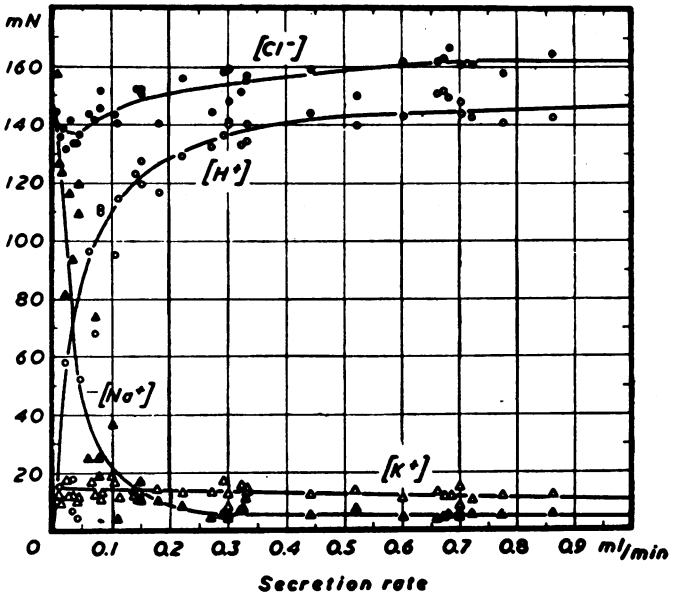

Fig. 1. *The relations of the concentrations of H^+, Na^+, K^+ and Cl^- to the secretion rate.* All the results in this figure are obtained from one dog. Each dot is obtained at a steady state secretion value. The full lines for $[H^+]$, $[Na^+]$ and $[Cl^-]$ are according to the diffusion formulas. — TEORELL (1947).

Since then the clinical value of gastric secretion tests has been discussed many times on various occasions, and the discrepancy of opinion has been marked. On the one hand, it has been emphasized that, apart from the finding of a Congo positive gastric content, the usual gastric secretion tests give very little of diagnostic value. On the other hand, the importance of a careful analysis of the gastric secretion for the understanding of the symptoms has been pointed out as a guide in the treatment of gastro-intestinal disorders.

If you ask what can we get out of a gastric secretion test, the answer depends to a very high degree on how much time and work you are willing to spend on the performance of the test. If you like to get reliable information about gastric secretion, you must realize that the acidity of a gastric sample says very little

about the secretion capacity of the stomach, if you do not know the secretion rate. To get reproduceable values you have to employ, *firstly*, a stimulus that gives a powerful stimulation of the gastric glands, and, *secondly*, a recovery technique which is satisfactory.

Fig. 1 shows the correlation between the dose of histamine, the secretion rate, and the acidity. When the secretion rate has reached a certain level, the acidity is high and does not change, and here you get the same acidity if the secretion rate alters considerably. With low secretion rates the acidity is low, in which case small changes in the secretion rate result in great changes in the acidity. When studying the effects of an inhibitory drug the results may vary from one case to

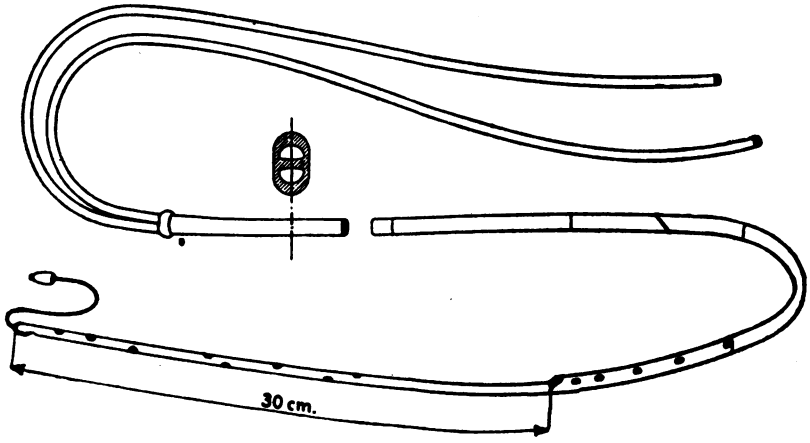

Fig. 2. The Lagerlöf—Ågren double tube.

another. In one case with hypersecretion, for instance, you may get a reduced secretion volume, but the same acidity, and in another case with a low secretion the drop in the acidity values may be much more marked than the small decrease in the secretion volume. Thus, when studying gastric secretion in clinical material you have to use a powerful stimulus where small changes in the secretion rate influence the acidity as little as possible. Histamine is the most powerful stimulant known, and gives you a picture of the chemical phase of gastric secretion under maximal conditions. The cephalic phase of gastric secretion is best studied with insulin, which, by means of insulin hypoglycemia, gives a strong vagal stimulation.

It has been said that gastric secretion tests after parenteral stimulation do not reflect physiologic conditions and that the Ewald test meal and the fractional test meals with alcohol, caffein, and arrowroot are more physiological than a histamine test, and that is perhaps the reason why those unreliable tests are still in use in so many places. It may, however, be doubted if physiologic conditions exist when a patient comes to see a doctor, and I feel very dubious about the ease and the relaxation of the patient when a tube is passed into his stomach. We know that gastric secretion may be inhibited by impulses from the brain through the vagus. It has been shown that such impulses do not only inhibit the vagal and

the spontaneous secretion but that the response to histamine is somewhat reduced at the same time. Some gastroenterologists in the United States find the examination of gastric juice aspirated over successive ten-minute periods under basal conditions, without any special meal or stimulus, the simplest method. In the case of a Congo negative gastric juice, the test is supplemented by a further study after the injection of histamine. I cannot consider this method as being a simplification of clinical work, but only an unsatisfactory way to study the spontaneous secretion, which must often be supplemented by an ordinary histamine test.

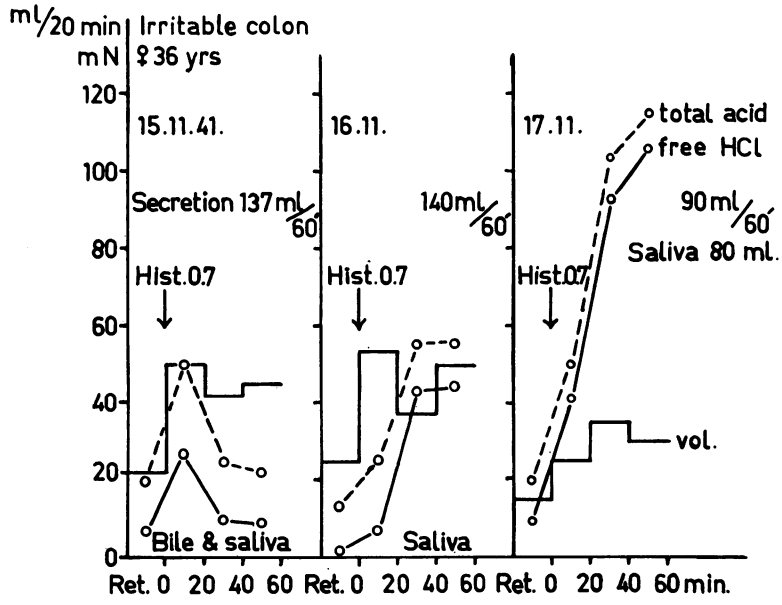

Fig. 3. Gastric secretion tests performed at three subsequent days in the same patient. On the first two days the acidity values were low due to the admixture of bile and saliva. On the third day a continuous suction of the gastric juice as well as of the saliva demonstrated normal acidity values.

As I have already mentioned, the acidity of a gastric sample says very little about the secretion capacity of the stomach, if you do not know the secretion rate. In routine examinations reliable information can only be obtained by a quantitative recovery of the gastric juice by means of a technique, in which the admixture of regurgitated duodenal juice and swallowed saliva is avoided as well as the loss of gastric content through the pylorus. A continuous aspiration of gastric and duodenal juice through a double tube as well as the simultaneous collection of saliva is the ideal (Fig. 2). It is, however, impossible to carry out in routine examinations owing to the difficulty in reachng the duodenum.

The histamine test with a single tube is generally carried out with withdrawals of the stomach content at intervals of 10-15 minutes after the subcutaneous

injection. It is, however, easy to demonstrate that a continuous suction of the gastric juice gives a more satisfactory recovery and higher acidity values. This is true not only in secretion tests after parenteral stimulation, but also when studying spontaneous secretion. In order to avoid admixture with the gastric juice it is necessary to have continuous suction of the saliva. It is not sufficient only to make the patient expectorate the saliva. This is to be seen in Fig. 3.

Fig. 4 shows the distribution of the average corrected number of parietal cells in the areas of eight normal stomachs according to a histo-topographical study by Berger (1934). In the black area the average proportion of parietal cells was

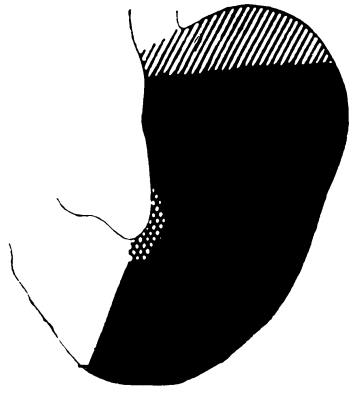

Fig. 4. The distribution of the average corrected number of parietal cells in the areas of eight normal stomachs, according to Berger.

maximal and was designated as 100 per cent. In proportion hereto, in the cross-hatched area, they were 75 per cent., and in the heavily shaded area 50 per cent. Consequently, if the whole acid secretion is to be collected during a test, it is necessary to have the lowest openings of the tube on a level just below the gastric angle.

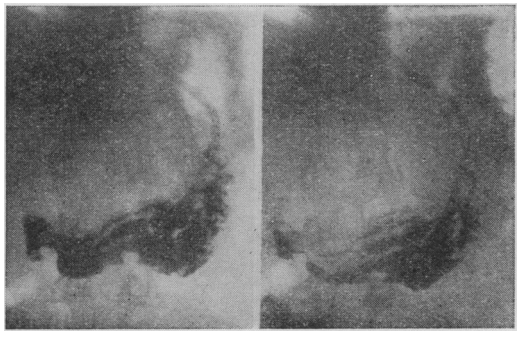

Fig. 5. The right place of the tube with the lowest openings of the tube just below the gastric angle. The left picture shows the stomach before, the right after the suction has been begun.

Fig. 5 shows the tube in the right place with the patient in a sitting posture. The left picture shows the stomach before, the right after the suction has been begun. The suction reduces the gas bubble in the upper part of the stomach, which is sucked together, and this facilitates the recovery of the gastric juice. Roentgen

controls in a series of cases have shown that if the following course is taken the tube will be in the right place.

The patient is examined in a sitting posture. The gastric juice is recovered by continuous suction with a negative pressure of 35-40 mm. Hg. through a somewhat stronger duodenal tube than the one generally used with a metal tip and 3-4 small openings at 1-2 cm. from each other at the lower end. The tube is lowered so that the distance from the lips to the lowest opening of the tube corresponds to the patient's body height, minus 100 cm. The gastric content is recovered with a 20 ml. syringe and the stomach is then blown up by injecting 100-140 ml. air through the tube. This is done to prevent the tube from coiling up within the upper part of the stomach. The gastric content is once more completely recovered. The right position of the tube is now controlled by injecting air through the tube. If the same amount of air can afterwards be recovered in the syringe, this indicates that the openings of the tube are placed at the right level. If this is not the case, the position of the tube has to be changed—usually it has to be raised a little. The tube is now fixed to the cheek with plaster. The suction of saliva and gastric juice is begun and the gastric secretion test can be carried out.

TABLE 1.

Comparison between the Results with Double and Single Tube.

No.	Sex and age	Date	Double Tube: Ml. per 60 min.	Double Tube: Free HCl/total acid. in Meq. maximal conc. per 20 min.	Single Tube: Ml. per 60 min.	Single Tube: Free HCl/total acid. in Meq. maximal conc. per 20 min.
1	♀23	15. 2.	–	–	75	105/120
		18. 2.	87	131/140	–	–
			65	141/150	–	–
2	♂47	17. 2.	–	–	160	100/106
		3. 3.	155	116/126	–	–
		12. 3.	148	110/120	–	–
		24. 3.	158	108/117	–	–
		14. 4.	165	128/134	–	–
		29. 10.	–	–	136	112/123
3	♀16	19. 4.	–	–	63	146/152
		21. 4.	63	147/152	–	–
4	♀29	25. 8.	–	–	70	80/90
		27. 8.	91	98/105	–	–
5	♂31	19. 6.	–	–	180	98/106
		27. 8.	140	124/130	–	–
6	♀30	4. 9.	–	–	99	52/67
		7. 9.	71	57/70	–	–

Table 1 shows a comparison between the results with a double and a single tube after histamine stimulation. The gastric as well as the duodenal content was

collected with the double tube, the single one only collecting the gastric content. There is a fairly good agreement between the results, and a statistical calculation of a great number of tests showed no significant difference between the mean errors of the secretion volumes with a double and a single tube. The histamine test, according to the technique described, gives more reliable information as to the secretion volume and acidity than any other clinical secretion test. On the basis of the statistical calculation of a normal material, examined according to the technique described, a secretion volume of more than 180 ml. per 60 minutes after a histamine dose of 0.1 mg. per 10 kilo body weight has been regarded as hypersecretion. High acidity values are not to be identified with hypersecretion, as you will realize from what I have already said. Higher acidity values than in the normal stomach are not to be found. Thus, the term hyperacidity has no clinical support when applying a reliable gastric secretion test (Fig. 6).

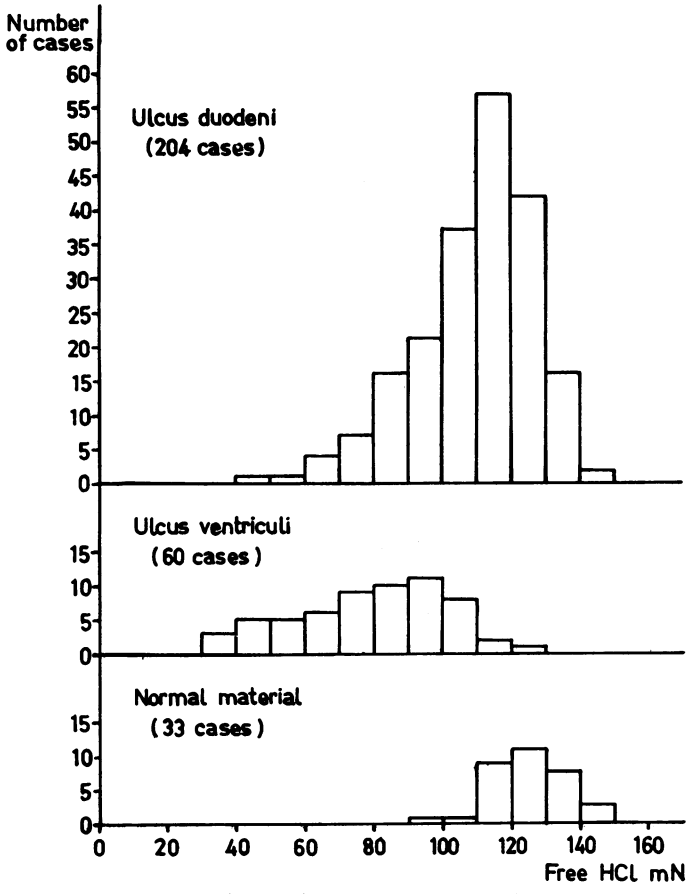

Fig. 6. Frequency distribution of maximal acidity in one of the twenty-minute portions in duodenal and gastric ulcer and a normal material.

The finding of a histamine refractory achlorhydria has a pathognomonic importance in some diseases such as pernicious anæmia and some types of sideropenic anæmia. Here the recovery technique is of fundamental importance. The admixture of bile and saliva may neutralize small amounts of hydrochloric acid and thus give the false impression of an achlorhydria. However, even with a satisfactory recovery technique, histamine refractory achlorhydria is a rather relative conception. With an increased dose of histamine up to 2-2.5 mg., it is in some cases possible to demonstrate hydrochloric acid, where an ordinary dose has failed. This high dose of histamine gives no untoward effects if, as Kay (1953) has proposed, 4 ml. "anthisan" is given 30 minutes before the histamine injection. However, if we wish to distinguish between a true achlorhydria, that is to say where the parietal cells have definitively stopped secreting hydrochloric acid, and a hypochlorhydria we have to use the neutral red test. Neutral red is a dye which is red at pH 6.8 and yellow at pH 8. If given intravenously, it is excreted by the parietal cells and the gastric juice thus turns red. In true achlorhydria, where the parietal cells have stopped the secretion of hydrochloric acid, the dye does not appear in the stomach when histamine is given simultaneously with the neutral red injection. The test is negative in pernicious anæmia as well as in a great number of histamine-refractory achlorhydrias. Hallén has shown that if hydrochloric acid is instilled into the stomach an excretion of neutral red may still be demonstrated in some cases of pernicious anæmia, which shows that the parietal cells lose the capacity to excrete neutral red later than the capacity to secrete hydrochloric acid. The same is true with regard to the secretion of pepsin. In most cases, even in pernicious anæmia, where the secretion of hydrochloric acid has disappeared, the secretion of pepsin remains and can be demonstrated after the instillation of hydrochloric acid into the stomach.

Thus histamine-refractory achlorhydria is not a uniform conception. It comprises different grades of hypochlorhydria down to the neutral red refractory state, when the parietal secretion has completely stopped, as well as conditions with preserved pepsin production down to the complete apepsinia.

In the majority of gastric diseases gastric secretion tests have a rather limited value. This is largely owing to the fact that secretion disturbances are to be found even in healthy subjects. In healthy children achlorhydria is uncommon. The incidence of hypo- and achlorhydria rises with increasing age. According to Polland (1933), achlorhydria was found in 3.5 per cent. between the ages of 20 and 30, whereafter the frequency rose gradually, and was 27 per cent. between the ages of 60 and 70 in healthy subjects without any stomach trouble. In peptic ulcer hypo- as well as normo- and hyper-secretion is found. Hypersecretion is relatively uncommon in gastric ulcer. It is, however, a common finding in duodenal ulcer, particularly in men. There seem, however, to exist some geographical differences. Hypersecretion, for instance, is a more common finding in duodenal ulcer in the southern parts of Sweden than in the north. A hospital material in Stockholm, examined according to the method described, showed a frequency of 34 per cent. hypersecretion in duodenal ulcer. The finding of hypersecretion is

always suspicious of duodenal ulcer. and if this cannot be demonstrated the patient must be regarded as a duodenal ulcer candidate.

If you want to get a complete picture of the gastric secretion you must examine the spontaneous secretion as well as the secretion after histamine and insulin. The spontaneous secretion may either be studied during 1-2 hours in the morning or

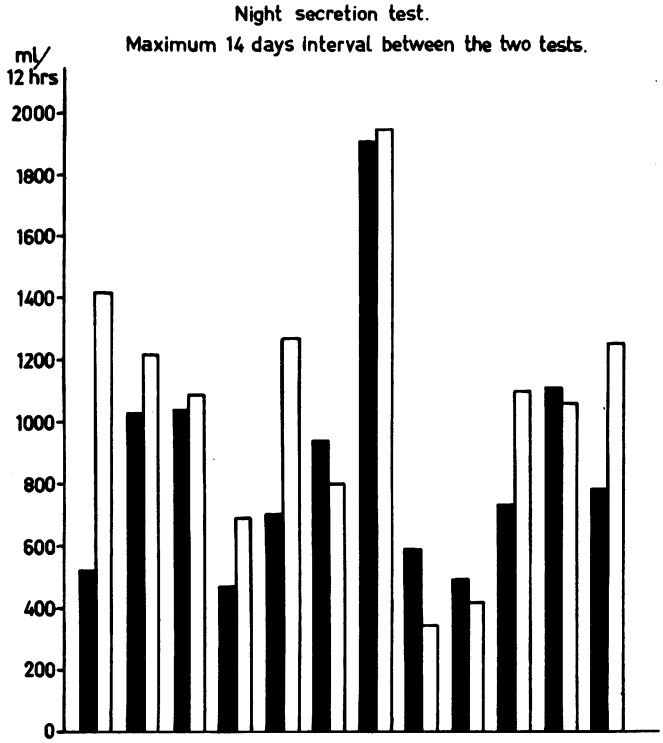

Fig. 7. Night secretion tests performed at two different occasions in twelve patients. The secretion volume was recovered continuously during twelve hours.

as a night secretion test. Even with a careful recovery technique the results of a night secretion test may differ considerably with regard to the secretion volume during 12 hours (Fig. 7). If a high night secretion is found, the secretion after histamine and insulin, as a rule, is also high. A hypersecretion after histamine, however, is not always combined with a high night secretion (Table 2).

The old view that achlorhydria is a common finding in cancer of the stomach is incorrect. Several investigations have shown that 30-40 per cent. of patients with cancer of the stomach have preserved hydrochloric acid secretion. In the differential diagnosis between gastric ulcer and carcinoma of the stomach the finding of a histamine refractory achlorhydria is of decisive importance, total achlorhydria being incompatible with the presence of a benign ulcer. The presence

of total achlorhydria may, however, only be regarded as definite after 2-3 histamine tests, preferably with a dose of 2-2.5 mg., as advised by Kay (1953).

Evidently the secretion of hydrochloric acid takes place at a constant level of about 170 Meq. This primary acidity when recovered—even if the admixture of

TABLE 2.

COMPARISON BETWEEN THE HISTAMINE TEST AND THE NIGHT SECRETION IN EIGHTY PATIENTS.

Diagnosis	No.of Cases	No Hyper-secretion	Hypersecretion		
			Only to ×Histamine	Only at ××Night	Both
Duodenal Ulcer	66	18	24	5	19
Gastric Ulcer	9	8	–	1	–
Prepyloric Ulcer	5	3	2	–	–
Total	80	29	26	6	19

× > 180 ml per 60 minutes
×× > 1025 ml per 12 hours (7 p.m. -7 a.m.)

duodenal juice and saliva is avoided—has dropped due to the nonparietal or mucous secretion, and to an acidity-reducing diffusion process. In a stomach with gastritic changes, both the nonparietal secretion and the acidity-reducing diffusion

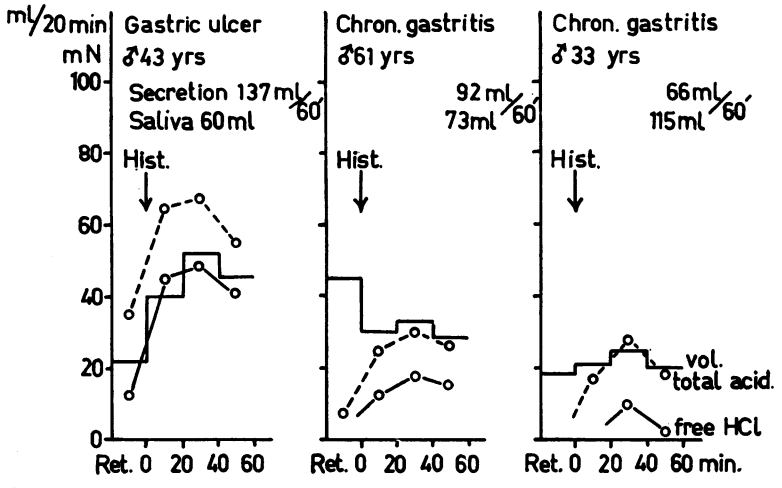

Fig. 8. Histamine tests in chronic gastritis.

process may be increased, resulting in low acidity values, though the secretion volume is considerable. A comparison between the histamine test and the gastroscopic findings confirms this. Acidity values under 90 Meq. at a secretion volume of at least 60 ml. per 60 minutes indicates the existence of chronic gastritis (Fig. 8). Acidity values up to 130-140 Meq. are indicative of a normal mucosa in an endoscopic examination (Fig. 9).

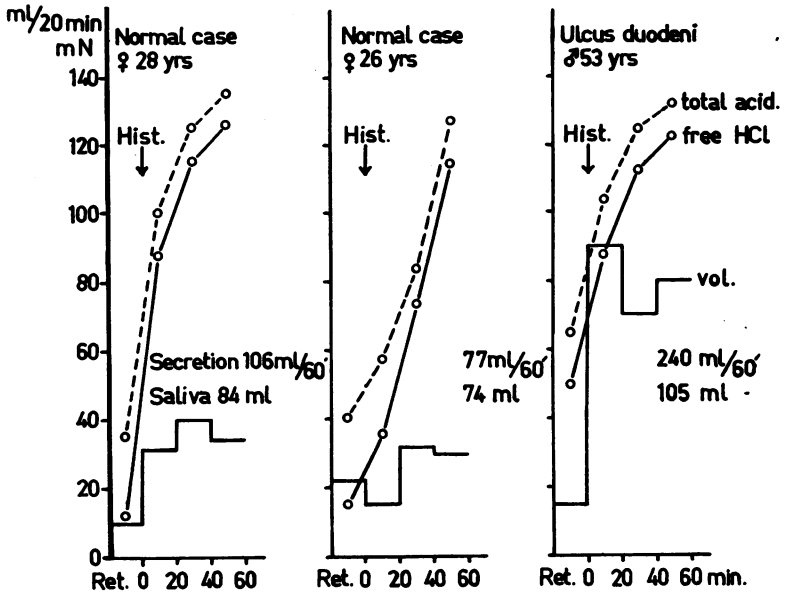

Fig. 9. Acidity values up to 130-140 mN are indicative of a normal mucosa.

Bloomfield, in the textbook edited by Portis (1953), draws the following conclusions in a chapter on clinical aspects of gastric secretion. "Analysis of the gastric secretions usually gives little or no information of clinical use. In certain special situations—for instance, when duodenal ulcer or pernicious anæmia is suspected—the determination of gastric acidity may be helpful. 'Routine' gastric analysis is a waste of time; the procedure should be reserved for the occasional case in which useful information may be obtained." I am of an opposite opinion. Not only in pernicious anæmia and certain types of sideropenic anæmias, but also in the treatment of peptic ulcer, regurgitation œsophagitis, gastritis, and diarrhœa it is of importance to know the gastric secretion. The choice of the operative procedure in peptic ulcer has to be accommodated according to the results of the gastric secretion tests. A Billroth I operation is not advisable in a hypersecretory stomach. Here we prefer a Billroth II operation possibly combined with a vagotomy in order to depress the secretion and avoid the occurrence of a marginal ulcer. In diseases of the digestive system the clinical examination is not complete if a gastric secretion test has not been carried out.

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REVIEW

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These are only a few selections from the thirty-seven subjects considered, and each author presents his material in a masterly manner. This is a volume well worth careful perusal by all family doctors.

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Retrolental Fibroplasia :
A mysterious type of blindness affecting premature infants

by O. D. FISHER, M.D., M.R.C.P.(LOND.), D.C.H.
Department of Child Health, Queen's University, Belfast

THIS condition was first reported in Boston in 1942 when Terry (1942), an ophthalmologist, detected a form of blindness in children which had not hitherto been described. It occurred only in those children born prematurely and of low birth weight. Examination of the eyes showed a white fibrous mass behind the lens which obliterated the retina—a condition he named "retrolental fibroplasia."

In the following few years, the incidence of this disease increased alarmingly and many cases were reported from all parts of the United States. The first case in England was detected in 1946, and by 1950 this disorder had become the commonest cause of blindness in the pre-school child. For example, in one maternity hospital three out of every four premature infants below 4 lbs. birth weight at one period were noted to show evidence of the disease. In Belfast, by 1951, thirteen children blind from this disease had already been seen at the Royal Belfast Hospital for Sick Children. The full extent of this catastrophe in Ulster, as elsewhere, is difficult to assess, as additional cases may possibly have been seen at other eye and children's hospitals or departments.

RECOGNITION.

How can the disease be detected clinically? It is confined to premature infants, especially those under 4 lbs. weight at birth. The affected infant's eyes appear normal at birth, but within a few days or weeks the iris becomes injected and the vitreous slightly hazy. The retinal veins are full and dilated, the arteries tortuous. (See Fig. 1.) This change is best seen peripherally where new vessels develop, and hæmorrhages may occur. The retina becomes pale and œdematous and later folded or detached. (See Fig. 2.) Finally, the retina becomes obliterated by a mass of white fibrous tissue so that, on looking into the eye, only a white reflex is seen and little or none of the detail of the retina can be appreciated. Up to the stage of retinal detachment the changes are reversible and in about half the cases they regress partially or completely. In the former, pigmentation and scarring follow, but some useful sight remains. (See Fig. 3.) At the stage of retinal detachment the changes are permanent and vision is grossly or completely impaired. The lesion is almost always bilateral; but the severity of the disease is not necessarily equal in both eyes, for the condition may regress in one eye and progress to blindness in the other eye. Later, the anterior chamber appears shallow and the iris grey and atrophic. Microphthalmos, glaucoma, and cataract may ensue.

The pathological changes found in the eye occur principally in the nerve fibre layer of the retina where there is excessive proliferation of vasoformative tissue.

This may burst through the retinal layer and invade the vitreous; it results in œdema and hæmorrhage leading to retinal detachment and later fibrosis which results in the formation of the dense retrolental membrane.

A New Disease.

Retrospective surveys indicate that retrolental fibroplasia is, in fact, a new disease, since it was not observed in premature infants born before 1940, nor has it been seen in children of a similar age group in schools for the blind.

Ætiology.

Since it is a disease which occurs only in the premature infant, the search for the ætiology was limited to factors such as prematurity and its causes, or the management and environment of the infant in the early weeks of its life.

Investigation had shown that no factor such as parity, maternal age, maternal

TABLE 1.

Relation of Birth Weight and Incidence of Retrolental Fibroplasia. M.R.C. (1955).

No. of Infants		Birth Weight		Incidence of Retrolental Fibroplasia (per cent.)
61	...	Under 2½ lbs.	...	16.4
152	...	2½–3 ,,	...	15.8
298	...	3 –3½ ,,	...	11.7
580	...	3½–4 ,,	...	2.6
Total, 1,091		4 omitted with congenital cataracts.		

disease or anæsthetic agent could be incriminated. Likewise, there was no evidence that a toxic element in the diet, whether breast or artificial feeding, or in the dietary supplements such as iron, vitamins A and D, or C, given to the infant was responsible. There was no association found between retrolental fibroplasia and other angiomatous conditions such as skin nævi, or with exposure to light or with ultra violet irradiation.

The Rôle of Oxygen.

By 1950 most of these theories had been investigated and excluded and a most promising clue was being followed up. Was retrolental fibroplasia due to the increasing use of oxygen in the management of the premature infant? For example, Crosse (1952) had observed that reducing the duration of oxygen treatment had lowered the incidence of retrolental fibroplasia. She also noted that the disease occurred particularly in those premature baby units in modern hospitals in the United States where oxygen was used extensively. Campbell (1951), in Melbourne, had also noted that more cases of retrolental fibroplasia occurred in premature infants in one maternity unit where high concentrations of oxygen were used, but fewer cases occurred in two other hospitals where little oxygen and lower concentrations were available.

If oxygen is the causative agent, how could this be safely proved?

Clinical Evidence.

In 1951 the Medical Research Council organised an extensive clinical investigation in seventeen maternity hospitals in the United Kingdom, including the Royal Maternity Hospital, Belfast. One thousand nine hundred and ninety-nine premature infants of less than 4 lbs. birth weight were observed and of one thousand and ninety-five who survived for at least two months, eighty-four (7.7 per cent.) developed retrolental fibroplasia, forty-five (4.2 per cent.) progressing to blindness. The Medical Research Council's report (1955) showed that retrolental fibroplasia occurred almost exclusively in premature infants below 4 lbs. in weight and that the incidence of retrolental fibroplasia rose progressivly as the birth weight fell (Table 1). The condition only occurred when oxygen was given and that the longer oxygen was administered the more likely was retrolental fibroplasia to develop (Table 2).

TABLE 2.

Incidence of Retrolental Fibroplasia in Relation to Duration of Oxygen Treatment.

M.R.C. (1955).

No. of Infants	No. of Days in Oxygen	Cases of Retrolental Fibroplasia		Total per cent. Incidence
		Blind	Regressed	
344	–	–	–	–
237	> 5	1	1	0.8
230	5–10	5	8	5.7
214	11–30	25	16	19.2
40	< 31	11	13	60.0

Between October, 1951, and May, 1953, of one hundred and nine premature infants who survived at the Royal Maternity Hospital, eleven showed evidence of retrolental fibroplasia; in four of these the changes regressed, but in seven blindness developed in one or both eyes.

This 10 per cent. incidence of retrolental fibroplasia at the Royal Maternity Hospital represented the fifth highest in the seventeen hospitals in the Medical Research Council investigation. There are at least three reasons for this apparently high figure. Firstly, every surviving infant was successfully followed up, and it is most unlikely that a single case of retrolental fibroplasia in the Royal Maternity Hospital survey escaped detection. Secondly, and most important, the overall survival rate—67 per cent. of premature infants below 4 lbs. birth weight—was the highest of the seventeen hospitals in the trial. Thirdly, a few of the English maternity units were aware of the possible association of oxygen and retrolental fibroplasia somewhat earlier in the trial. Since 1952, when lower concentrations of oxygen have been in use at the Royal Maternity Hospital, the incidence of retrolental fibroplasia fell immediately, and from June, 1953, there have only been two definite and one doubtful cases of retrolental fibroplasia, all of which regressed before the stage of retinal detachment, and so blindness has not developed. In 1955 there have not been any examples of the disease.

The importance of oxygen has also been shown by Boyd and Hunt (1955) in a survey of all premature infants born in England and Wales in 1951. This revealed an incidence of 2.1 per cent. of retrolental fibroplasia in premature infants born in, or transferred to hospital, whereas no case of retrolental fiboplasia occurred in premature infants reared at home, presumably without oxygen therapy.

Experimental Evidence.

That oxygen is an important, if not the sole agent causing this form of blindness in premature infants has been confirmed by the experimental work of Ashton and his colleagues at the Institute of Ophthalmology (1953 and 1954).

These workers noted that kittens' eyes at birth are at a similar stage of development as those of the small premature infant and became mature about three weeks after birth. They showed histologically that a marked constriction of the retinal vessels resulted when the newborn kittens were reared in high concentrations of oxygen. This constriction affected the arteries especially, beginning in less than twelve hours and going on to complete obliteration of the vessels after about thirty-six hours in a concentration of 70-80 per cent. of oxygen. The degree of vasoconstriction varied with the duration and concentration of oxygen used and the immaturity of the kittens' eyes, i.e., a 35 per cent. concentration of oxygen did not produce any constrictive effect regardless of the duration of treatment. After three weeks of age no change could be induced in the kittens' eyes with high concentrations of oxygen.

After a high oxygen concentration had produced a vasoconstriction of the retinal vessels the exposure of the kittens to air again resulted in a new development. A revascularisation of the retina began, the constricted vessels reopened, filled with blood, the pattern being now grossly abnormal and irregular. Hæmorrhage and retinal detachment followed rupture of the vessels, exactly the pattern that is seen in the early stages of retrolental fibroplasia in the premature infant.

Using the limbal window technique, Ashton and Cooke (1954) have observed and recorded cinematographically the actual changes in the retinal vessels, how, when the newborn kitten is placed in 80 per cent. concentration of oxygen, the retinal vessels become constricted and even obliterated in as short a time as eight hours. These vascular changes do not occur, however, in the vessels of the brain or other parts of the body.

There is, thus, experimental and clinical evidence to show that retrolental fibroplasia is liable to occur in premature infants, particularly in those below 4 lbs. in birth weight when kept in a high concentration of oxygen for a prolonged time.

Treatment and Prevention.

Knowing now how the blindness develops, what can be done for the victim?

In a condition in which spontaneous remission may occur the effect of therapy is difficult to assess. Adrenocorticotrophic hormone (A.C.T.H.) and adrenocortical steroid, e.g., cortisone, have been advocated, but there is no definite evidence to confirm their efficacy, and death has resulted in the premature infant as a result of such therapy. Likewise, vasodilators, such as tolazoline hydrochloride (Priscol),

have been tried but without effect. So far there is no effective therapy in the reversible stage and once retinal detachment has occurred the blindness which results is permanent.

How, therefore, can premature infants be protected from this tragic form of blindness?

Prevention of this disease is possible, urgent, and imperative.

At the Royal Maternity Hospital the following precautions are taken. Oxygen is not given to premature infants as a routine but only when indicated for feeble respiratory movements, cyanosis, apnœa, etc. Oxygen is used for as short a period as possible, usually one to four days, the concentration of oxygen is checked daily for individual incubators or boxes, using the Oxygenaire pyrogallic acid oxygen analyser, and the level is recorded for each premature infant receiving oxygen. For everyday use, a concentration of 40 per cent. of oxygen is not exceeded, this being obtained by a flow of not more than 2 litres per minute, as recorded by the flowmeter for the Oxygenaire type incubator, or 1½ litres per minute for the Queen Charlotte's Hospital canopy-type incubator. The oxygen concentration is reduced slowly by opening progressively the air vent on the Oxygenaire incubator or by propping open the lid of the Queen Charlotte's Hospital box with wooden pegs increasing in size from ½ in. to 4½ ins. There is a piped oxygen supply to the premature infant nursery which is controlled by float-type flowmeters permanently fixed to the walls which prevents the possibility of tilting or damage when attached to or detached from the oxygen cylinder.

The eyes of all premature infants who weigh less than 4½ lbs. at birth are examined as soon as the individual infant is fit enough. One drop of 1 per cent. homatropine solution is put in each eye one and a half hours and half an hour before the examination which is made at weekly or fortnightly intervals or more frequently if indicated. Special miniature lid retractors, made by Weiss Ltd., facilitate the examination.

The Influence of Oxygen Therapy on Survival Rate.

Is retrolental fibroplasia, which is a man-made disease with the premature infant as the victim and oxygen as the weapon, the price to be paid to lower the mortality from prematurity? Since oxygen has been used as a valuable ally in the fight to save the premature infant, but is now regarded as a dangerous drug in high concentrations and for prolonged use, is there any evidence to show that these higher concentrations and longer duration of therapy do, in fact, increase the survival rate of the premature infant? In the Medical Research Council report it was concluded that, though there were many other variables to be taken into account, the nurseries in which little oxygen was given and which were free from cases of retrolental fibroplasia did not, on the average, experience survival rates inferior to nurseries using more oxygen. It is interesting to compare the survival rates of premature infants over the past twenty years at the Royal Maternity Hospital, Belfast. In the four years, 1932-1935, every premature infant under 3 lbs. birth weight died, whereas the overall survival rate up to 5½ lbs. birth weight was 64 per cent. By 1947, 21 per cent. of infants under 3 lbs. birth weight survived

and the overall survival rate had risen to 74 per cent. In 1952, during the early part of the Medical Research Council investigation, when the use of oxygen was unrestricted, 55 per cent. of the infants under 3 lbs. survived, while the overall survival rate was 84 per cent. But in 1954 only 23 per cent. of infants under 3 lbs. survived, while the overall survival rate had also fallen to 80 per cent. (Table 3).

TABLE 3.

PER CENT. SURVIVAL OF PREMATURE INFANTS AT ROYAL MATERNITY HOSPITAL, BELFAST.

	Birth Weight in Lbs. and Ozs.						Average Survival
	> 2.15	3–3.7	3.8–3.15	4–4.7	4.8–4.15	5–5.8	(per cent.)
1932-5	0	56	76	72	83	82	64
1947	21	63	80	72	79	96	74
1952	55	68	91	80	95	98	84
1954	23	59	88	87	93	98	80

The only major change made in the management of the premature infant at this time was a reduction in the duration of oxygen administration and careful control of its concentration.

Can the decrease in the survival rate in the smaller premature infants in 1954 be due to the use of lower concentrations of oxygen, as the survival rate in the higher birth weight groups of premature infants in 1954 was the same as the 1952 level?

TABLE 4.

FATE OF PREMATURE INFANT BELOW 3 LBS. BIRTH WEIGHT AT THE ROYAL MATERNITY HOSPITAL, BELFAST, IN 1952 AND 1954.

		Number of Infants Birth Weight				
		lbs. ozs.	lbs. lbs. ozs.	Total		Per cent.
		> 1 15	2 – 2 15			Surviving
1952—Period of unlimited oxygen therapy :	Alive	- –	22	22		55
	Dead	- 7	*11	18		–
1954—Period of restricted oxygen therapy :	Alive	- –	7	7		23
	Dead	- 6	*17	23		–

*Includes one infant born with congenital malformations incompatible with prolonged survival.

To eliminate three possible variables in these groups the following analysis shows that the number of cases and birth weight distribution are roughly the same for premature infants under 3 lbs. in 1952 and 1954, and also that there is the same number of congenital abnormalities incompatible with prolonged survival (Table 4). This suggests that at the Royal Maternity Hospital the use of higher

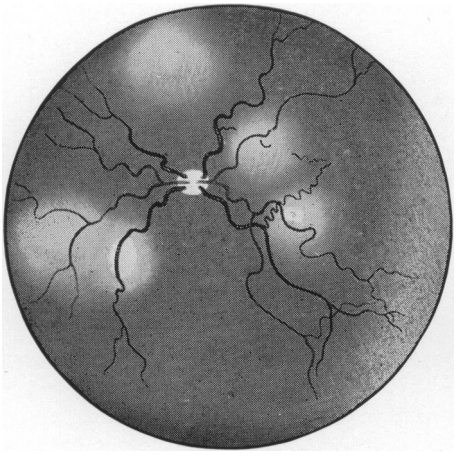

Fig. 1—Fundus at 3½ weeks.

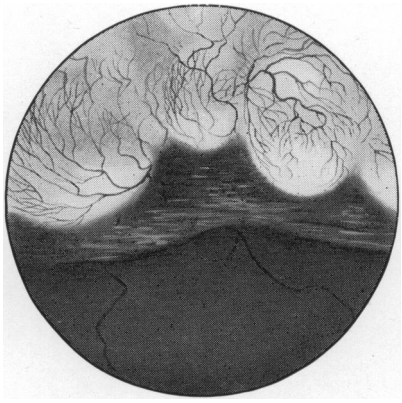

Fig. 2—Fundus at 13 weeks.

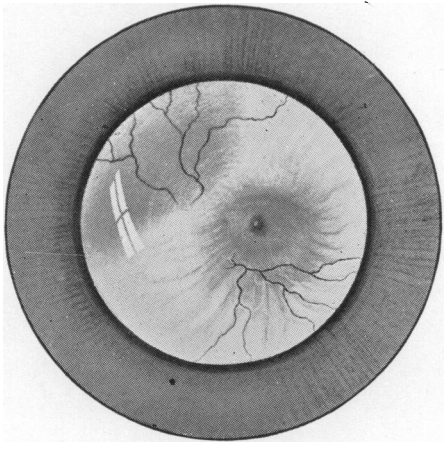

Fig. 3—Fundus at 21 weeks.

Reproduced from "British Journal of Ophthalmology," Vol. 56, page 281, 1952.

concentrations of oxygen over prolonged periods in 1952 may have been a factor improving the survival rate for the smaller premature infant. However, the definite risk of causing blindness in a premature infant when using high oxygen concentrations far outweighs the possibility of a slightly higher survival rate in the small premature infants, and the use and concentration of oxygen must continue to be carefully controlled in the treatment of the premature infant.

ACKNOWLEDGMENTS.

I wish to thank my medical and nursing colleagues at the Royal Maternity Hospital, Belfast, for their advice and assistance in the study of this problem. I am most grateful to Doctors C. A. Brown and Beryl Corner, and to the Editor of the "British Journal of Ophthalmology" for permission to reproduce the illustrations of retrolental fibroplasia.

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Orf
(Contagious Pustular Dermatitis of Sheep)

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DURING the spring and early summer of 1955 an unusually large number of cases of orf were seen in patients attending the various skin clinics in Northern Ireland. The condition aroused considerable interest, and it is hoped that these notes may prove helpful to local practitioners.

Orf is an infective disease, caused by a filtrable virus of the pox group, affecting commonly sheep and goats. The lesions which occur in animals are often secondarily infected with pathogenic bacteria and appear as vesicles and pustules on the skin of the lips, legs, and genitalia, frequently followed by ulceration, suppuration, and necrosis. Orf occurs throughout the world, and though the mortality from the uncomplicated disease is negligible, the rapid loss of weight and condition of the young animal is serious. The infestation of the lesions of orf with the screw-worm fly in Texas causes enormous animal losses (Boughton and Hardy, 1935). The condition in humans is, of course, usually seen amongst those who work with sheep, and was reported for the first time by Peterkin (1937).

VETERINARY ASPECTS.

There are three main clinical types in sheep (Král and Novak, 1953):

(a) *Labial*, which is the most common with small vesicles occurring on the mouth, lip, and nose. These vesicles rupture, are secondarily infected with resulting ulceration, so that eating and swallowing becomes painful and the young animals frequently develop ulcerative gastro-enteritis and broncho-pneumonia. The mortality rate is about 90 per cent.

(b) *Podalic* form or foot rot. The lesions are present between the hoof clefts or in advanced cases up to the knee joint. Ulceration leads to necrosis of ligaments, tendons, and bones. This type is almost always fatal.

(c) *Genital* form affecting the vulva and skin of mucous membrane underneath the tail, and in males the prepuce.

The prognosis in young animals is poor, especially if more than one clinical form is present.

Prophylactic measures include the maintenance of strict hygiene, periods of observation before putting newly purchased sheep in with old flocks. Vaccination has occasionally been carried out successfully.

The treatment is mainly local since the sulphonamides and antibiotics have not proved of any value.

Clinical Features.

The incubation period is a short one of 3-7 days. The initial lesion is a firm, painless, dusky-red papule. These lesions are generally on the fingers where the skin might come in contact with a lesion on the mouth or elsewhere in a young animal. A nodule about 10 mm. diameter is present within 14 days, and later may grow to as much as 2-3 centimetres (Fig. 1). The centre is depressed, covered by a white membrane. In an early lesion, when this membrane is broken, a fluid material can be seen, but in older lesions the contents are semi-cheesy. The lesions may be single or multiple. There may be associated lymphadenitis, possibly from secondary infection. The disease is self-limiting, lasting about 8-10 weeks (Goldsmith and Hellier, 1954). Occasionally an erythema multiforme-like eruption with general constitutional symptoms is associated (Blakemore, Abdussalam, and Goldsmith, 1948). This was seen in one of our cases (Fig. 2).

Etiology.

The virus was discovered in 1928 and has been adapted to many animals, in some of which a solid immunity can be achieved by vaccination with active virus. Blakemore *et al.* (1948) inoculated material into several lambs producing lesions of orf. The disease was further transmitted to a human volunteer. Agglutinins were detected in the serum of the patient as late as 132 days after infection. Lyell and Miles (1950) cultivated the virus from man on living chick embryo and reproduced the disease from this in a lamb.

Differential Diagnosis.

The condition, once seen, is easily recognised again, particularly if the history of contact with infected lambs or sheep is available. Molluscum contagiosum is an uncommon condition in adults and the lesions are never as big as are those of orf, but, of course, orf can and does occur in young children who may play with and feed young lambs. Molluscum sebaceum is a benign tumour which does not often affect the fingers or hand (Fig. 3). The central part of the molluscum sebaceum lesion is a mass of hard keratin (Beare, 1953). Squamous carcinoma may give rise to difficulty, but the short history in orf and the inflammatory nature of the lesion are distinguishing features. Granuloma pyogenicum is more vascular and friable (Fig. 4).

Treatment.

The treatment is entirely local since the sulphonamides and antibiotics have no effect on the condition, though these may be necessary to control secondary infection. Destructive measures are occasionally advocated, but our recent cases have been treated with potassium permanganate soaks, and the lesions seem to have settled down quite satisfactorily.

The patient shown in Fig. 2 was particularly interesting because of the erythema multiforme eruption limited to the light-exposed areas (face, hands, wrists, and knees), but this eruption settled within twenty-four hours on admission to hospital, presumably because he was no longer exposed to direct sunlight.

Acknowledgments.

I wish to thank Dr. Ivan H. McCaw for permission to publish a photograph of one of his cases (Fig. 2).

Mr. D. H. Mehaffey was responsible for Figs. 3 and 4.

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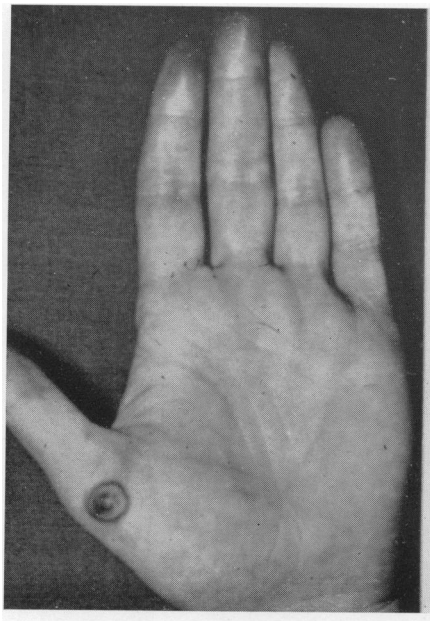


Fig. 1
Typical example of orf.

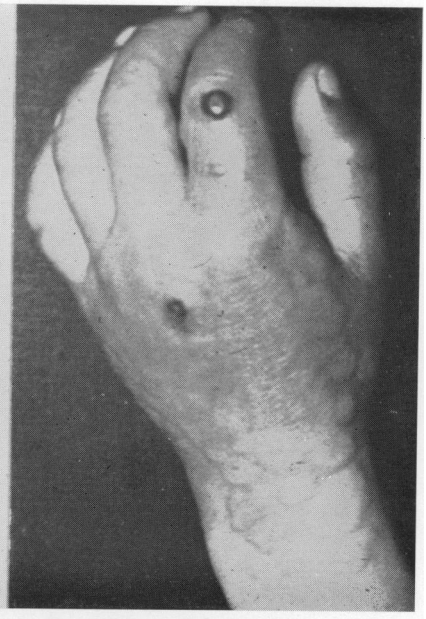


Fig. 2
Orf on the back of forefinger and hand of a young boy, also showing erythema multiforme reaction on the parts exposed to light.

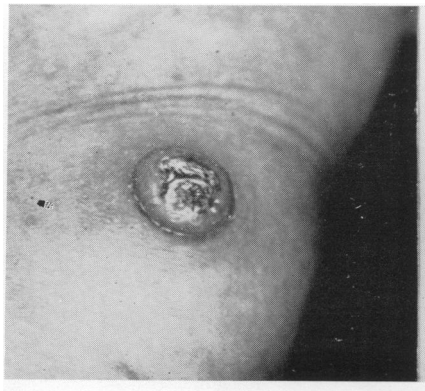


Fig. 3
Typical molluscum sebaceum on knee.

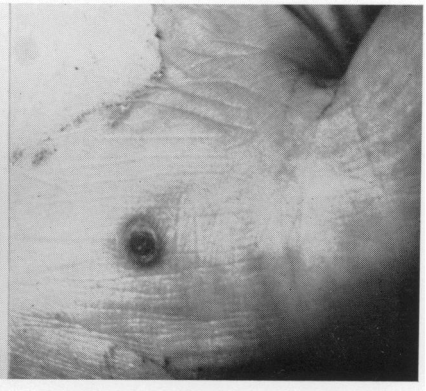


Fig. 4
Pyogenic granuloma of palm.

Cecil Armstrong Calvert

M.B., F.R.C.S.I.

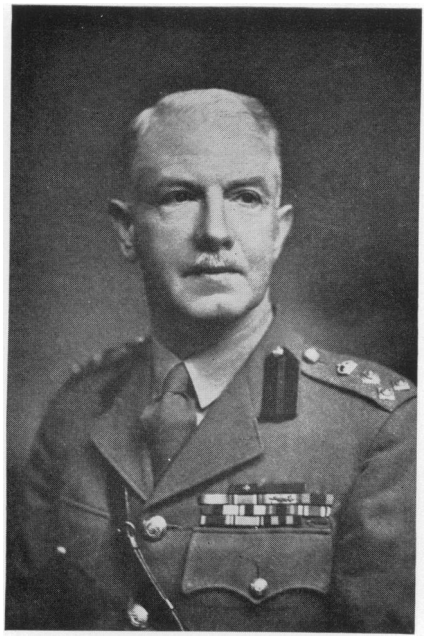
Vice-President Ulster Medical Society, 1955-1956

9th MAY, 1894 — 4th APRIL, 1956

Educated at Lurgan College and The Queen's University, Belfast, graduating in medicine with first-class honours in 1922. Formerly Visiting Surgeon, Royal Victoria Hospital and Ulster Hospital for Children and Women. Lt.-Col. R.A.M.C., Military Hospital for Head Injuries, Oxford, 1940-45. Director of the Department of Neurosurgery, Royal Victoria Hospital. Author (jointly) 8th Edition 'Whitla's' Dictionary of Treatment, 1938, and of contributions to the surgery of head injuries published in War Supplement of the 'British Journal of Surgery,' 1947.

One of the original members of the Society of British Neurosurgeons and a pioneer in this field, 'whose daring led him to open up new horizons in brain surgery'; of his work at St. Hugh's, Oxford, it has been said he was 'the staff on which Sir Hugh Cairns leaned.'

Cecil Calvert had an international reputation of the first order as a clinician and surgeon, but posterity will honour and revere his memory more for his gentle and unassuming nature and his lifelong practice of teaching by example rather than by precept. This latter characteristic was an inspiration to all those who had the good fortune to work with him, and the full significance of its influence cannot yet be measured. He was undoubtedly one of the most outstanding men the Belfast School has produced.



John Alexander Sinton

V.C., O.B.E., M.D. (honoris causa), D.Sc., F.R.S., D.T.M.

Brigadier Indian Medical Service

2nd DECEMBER, 1884 — 25th March, 1956

Educated at the Royal Belfast Academical Institution and Queen's College, Belfast, he obtained, with first-class honours, the degrees of M.B., B.Ch., B.A.O., of the Royal University of Ireland in 1908.

As a Captain of the Indian Medical Service he was awarded the Victoria Cross "for conspicuous bravery and devotion to duty. Although severely wounded in both arms and through the side, he refused to go to hospital and remained as long as daylight lasted attending to his duties under heavy fire. . . ."

His researches, particularly in malaria, earned for him wide fame and the Fellowship of the Royal Society. Among many other distinctions he treasured the Honorary Fellowship and the Robert Campbell Medallion of our Society.

He was a Pro-Chancellor of the Queen's University and Honorary Colonel of its Officers Training Corps. Elsewhere it has been written of him that "his greatness sprang not so much from his unusual intellectual gifts as from the simple qualities of absolute integrity and tremendous industry. . . . He was by nature the kindest, most generous, and gentlest of people."

He bore his last, long painful illness with smiling courage. Truly "he remained as long as daylight lasted attending to his duties. . . ."

Blood Loss in the Third Stage of Labour

By T. WILSON RODDIE, M.B., B.CH., B.A.O., M.R.C.O.G.

Senior Lecturer in Obstetrics and Gynæcology, The University of Malaya, Singapore;
Formerly Principal Registrar and Tutor in Obstetrics
The Royal Maternity Hospital, Belfast

THE third stage of labour has often been the subject of much discussion and dissension, yet it is the least understood, the most ill-managed and the most tampered-with stage. Control of post-partum bleeding continues to be one of the highly important problems which are encountered by obstetricians throughout the world. Post-partum hæmorrhage is always a dangerous and alarming accident, requiring prompt and vigorous treatment. Dr. Gooch (quoted by Swayne, 1884) said: "In these cases . . . the life of the patient depends on the man who is upon the spot; he must stand to his gun, and trust to his own resources. A practitioner who is not fully competent to undertake these cases of hæmorrhage can never conscientiously cross the threshold of a lying-in chamber."

There has been a striking decrease in the maternal death rate during the past fifteen to twenty years, caused by a sharp reduction in two of the leading causes of maternal deaths, namely, puerperal sepsis and the toxæmias of pregnancy. However, maternal mortality caused by obstetric hæmorrhage has remained practically constant except for minor fluctuations. Although obstetricians are all seriously concerned when a woman bleeds to death from a post-partum hæmorrhage, it is suggested that frequently there is not sufficient concern shown for the woman who does not die but who sustains a greater than normal blood loss which invites infection, delays convalescence, and interferes with the nursing of the baby. The patient also is robbed of the high level of health and happiness she deserves, and is burdened with several months of ill health.

Many women lose comparatively large amounts of blood during labour without noticeable effect, while others may be seriously affected by a relatively small blood loss. A small third-stage hæmorrhage, plus a low hæmoglobin level, can be a very grave matter. The combination of profound anæmia, with even a moderate hæmorrhage in labour, is probably, the world over, the most common cause of death in child-bearing.

Calkins *et al.* (1931) analysed a series of approximately 5,600 deliveries and found the average blood loss in the third stage to be about 5 fl. ounces (150 ml.). This figure is considerably less than the amount given as an average by the majority of other investigators. In most hospitals the recognised upper limit of normal blood loss is 20 fl. ounces (570 ml.), though most standard obstetric textbooks state that a third-stage loss greater than 10 fl. ounces (285 ml.) is abnormal. A study of obstetric reports from British hospitals, where the upper limit of normal blood loss is taken as 20 fl. ounces, indicates that the incidence

of post-partum hæmorrhage is somewhere between 2 and 6 per cent., and even in well-regulated institutions deaths from post-partum hæmorrhage, although rare, do occur. Figures from American sources are similar in showing that the overall incidence of third-stage and post-partum hæmorrhage is not less than 5 to 6 per cent. (Davis, 1940; Davis and Geady, 1946), and, if accurate measurements of blood loss were always made, the figure might approach 10 per cent. It is evident that the incidence of post-partum hæmorrhage shows no appreciable decrease, but is, in fact, increasing. One reason for this increase is undoubtedly the adoption of more rigid criteria as to what constitutes abnormal bleeding.

In an attempt to get an accurate idea of the third-stage blood loss an analysis of the amounts lost following 1841 consecutive vaginal deliveries, which occurred in the Royal Maternity Hospital, Belfast, during 1953, was made. In this hospital care is taken to collect as much as possible of the post-partum blood loss and the amount is measured. Where this has not been possible the loss is recorded as normal or moderate. Although, no doubt, far from being accurate, a moderate loss is assumed to be anything from 10 to 15 fl. ounces (285 to 435 ml.). It is important that the blood loss should be measured when at all possible, as the measured loss nearly always exceeds, and at times greatly so, the estimated loss.

In this series of 1841 patients there were 263 (14.3 per cent.) who had a third-stage loss greater than 10 fl. ounces. The percentage with a blood loss greater than 20 fl. ounces, the usual criterion of a post-partum hæmorrhage, was 2.4 per cent. This latter figure compares favourably with other hospitals. For patients delivered with forceps, under anæsthesia, 16.3 per cent. had a loss greater than 10 fl. ounces, but only 1.7 per cent. had a loss of more than 20 fl. ounces. In the series there were 50 cases of twins, of which 12 per cent. had a loss greater than 10 fl. ounces and only 2 per cent. a loss greater than 20 fl. ounces. The complete results are shown in the following table:—

	No. of Patients.	No. with loss over 10 fl. oz.	No. with loss over 20 fl. oz.
Whole Series	1,841	263 (14.3%)	44 (2.4%)
Normal delivery	1,332	189 (14.1%)	35 (2.6%)
Forcep delivery	342	56 (16.3%)	6 (1.7%)
Breech delivery	117	12 (10.2%)	2 (1.6%)
Twin delivery	50	6 (12%)	1 (2%)

If it is accepted that a third-stage loss of 10 fl. ounces is the upper limit of the normal, then the cases analysed revealed that post-partum hæmorrhage occurred nearly as often after normal as after abnormal deliveries. Taking a figure of 20 fl. ounces as the upper limit, as is usual, then the incidence of post-partum hæmorrhage was greater after normal than after abnormal deliveries. Contrary

to the usual teaching, there was no evidence that twins, prolonged labour, or multiparity were predominantly associated with excessive third-stage bleeding. It was interesting to note that the average duration of normal labours which had a third-stage loss greater than 10 fl. ounces was 13.3 hours for primigravidæ and 8.2 hours for multiparæ. The normally delivered patients who suffered a loss greater than 20 fl. ounces had actually a shorter average duration of labour—10.8 hours for the primigravidæ and 7.1 hours for the multiparæ.

It is interesting to compare the results in this present series with figures published by de Boer (1955). In an analysis of 2,444 successive vaginal deliveries at the London Hospital in the years 1950 to 1952 the overall incidence of a post-partum hæmorrhage of 20 fl. ounces or more was 16.5 per cent. From his figures he noted that prolonged labour, forcep delivery, and anæsthesia are important ætiological features in the production of post-partum hæmorrhage. The figure in this series seems high when compared with that from the Royal Maternity Hospital, Belfast. The incidence of manual removal of the placenta in the Belfast series was 1.07 per cent. and in de Boer's series 2.04 per cent.

What can be done to prevent excessive third-stage blood loss? In the first place, prevention must begin with the proper conduct of the terminal phase of the second stage of labour. For the placenta to separate properly it is of the utmost importance that the baby be delivered slowly. The time taken to effect delivery should be at least three minutes, taking a half to one minute pause after the delivery of each shoulder.

Secondly, knowledge of the mechanism of placental separation must be kept in mind if undue hæmorrhage is to be avoided. It matters little whether one accepts the older view of separation by means of a retroplacental hæmatoma, or the more modern view of a decrease in size of the uterine cavity and area of placental attachment leading to a partial and finally complete separation. In this latter view, as the uterus contracts and retracts, its walls become thicker and thicker and the area of placental attachment smaller, so that eventually the compact placenta can no longer follow the changes and is consequently separated from the uterine wall. It is important that no attempt should be made to hasten this process. The practice of massaging and squeezing the uterus, after the birth of the baby, to separate the placenta, as well as the less traumatic plan of holding the fundus, are mentioned only to be condemned. By such procedures nothing is gained and much may be lost by unnecessary traumatism. The ecchymotic and contused abdominal walls, the sensitive and bruised uterus, and the relaxed and tender uterine ligaments are still occasionally seen to-day.

Thirdly, the signs of placental separation and descent must be appreciated. In short, the fundus rises and becomes smaller, harder, and rounder. There may be a small gush of blood from the vagina and the cord outside the vulva lengthens. When these have occurred, the placenta is expelled by grasping the fundus uteri in the hollow of the hand, and as soon as it is felt to harden, strong and firm pressure is made upon it, downwards and backwards, in the axis of the pelvic brim.

The management of the third stage of labour may vary from place to place,

but the fundamental principles do not change. If these are remembered, good results should be obtained. The value of giving oxytocic drugs after the child's head is born and before delivery of the placenta still evokes much discussion, but whether these drugs are given or not seems to be mostly a matter of individual experience. Good management of the third stage of labour is vital, and all those practising obstetrics should realise that the prevention of post-partum hæmorrhage is much easier than the treatment. In the year 1767 John Harvie stated that "nature is to be assisted, to be followed and supported, but seldom or never forced." These words are worth remembering.

Summary.

1. The third-stage blood loss following 1841 consecutive vaginal deliveries at the Royal Maternity Hospital, Belfast, is analysed.
2. A blood loss greater than 10 fl. ounces (285 ml.) occurred in 14.3 per cent. of cases and 2.4 per cent. sustained a loss greater than 20 fl. ounces (570 ml.).
3. Post-partum hæmorrhage occurred as often after normal as after abnormal deliveries.
4. There was no evidence that prolonged labour, forcep delivery or anæsthesia, twins or multiparity were predominantly associated with excessive third-stage bleeding.
5. Prevention of excessive third-stage loss depends on the proper conduct of the terminal phase of the second stage of labour, knowledge of the mechanism of placental separation, and the ability to recognise the signs of placental separation and descent.
6. An endeavour to hasten placental separation is a basic factor in many cases of excessive third-stage bleeding.

I wish to thank Professor C. H. G. Macafee for his advice in the preparation of this paper and the members of the Consulting Staff of the Royal Maternity Hospital, Belfast, for permission to use the records of patients who had been admitted under their supervision.

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The Inheritance of Tylosis Palmaris et Plantaris in Two Families in Northern Ireland

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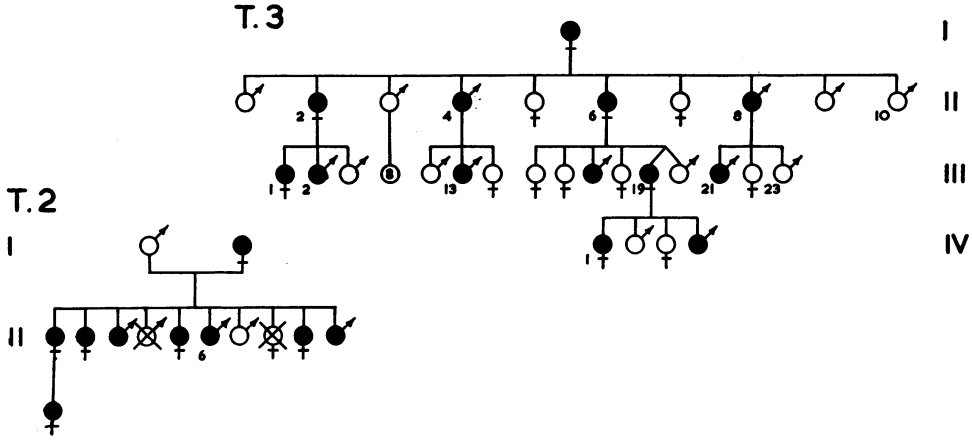
STEVENSON and Pearson (1953) recorded the inheritance of tylosis palmaris et plantaris in one large family in Northern Ireland, and an isolated case which they regarded as an example of a new mutation. It seems worth recording two additional families for a number of reasons. The condition is an example of a trait carried by a regular dominant gene, which appears always to be expressed. It seems likely from the writer's experience, from that of the Department of Social and Preventive Medicine at Queen's University, Belfast, and from the records of the Dermatological Clinics in Northern Ireland over the last four years that all, or a very high proportion of all, the subjects affected by this trait in Northern



Ireland have been identified. It is therefore possible to estimate the prevalence of this trait with some confidence, a matter on which relatively little information is available concerning single gene determined traits. Finally, linkage data have been collected which are complementary to those of Stevenson and Pearson (1953).

The condition of affected individuals in the families here reported shows a very marked uniformity. The skin on the palms of the hands is, in every case, thicker and harder than normal, with the flexure lines appearing as deep fissures or cracks in a smooth expanse of thickened skin extending from wrist to finger tips. The appearance of the hands is so characteristic that it could be immediately determined by a single glance whether or not an individual was affected. The appearance of the

feet is not so immediately conclusive, for the skin on those parts of the soles which are not normally in contact with the ground is relatively soft and tends to have a more normal appearance, although it is abnormally thick. The skin covering the plantar surfaces of the toes and the parts of the soles normally in contact with the ground is thick and horny and deeply fissured at flexure lines.

In a number of cases the hard, thickened skin spreads on to the dorsal surfaces of the digits at their tips and reaches the nail beds, causing deformity of the nails and occasional discomfort, but many affected individuals have normal nails. In a few cases there is also some thickening of skin over the dorsal aspects of the distal interphalangeal joints. No other encroachment of abnormal skin on to the dorsal surfaces of hands or feet was observed in any of the affected individuals interviewed.

All affected individuals suffered discomfort as a result of the fissuring and peeling of the horny skin of the palms and soles. Discomfort was not continuous, but was almost always present during very warm or very cold weather; it was also exacerbated by manual labour especially if the work involved exposure of the hands to moisture and cold—e.g., cement mixing in winter was mentioned by one affected individual as being a particularly unpleasant occupation. Peeling of horny skin took place from time to time in all cases, but varied in frequency from once to three or four times a year in different individuals.

No affected individual showed any thickening or other defect of the skin elsewhere on the body, or any other malformation or characteristic which could be related to the condition.

Most affected individuals had experimented with various methods of treatment. There was fairly general agreement that applications intended to soften the skin made the condition less comfortable, and that rubbing the horny layers away with pumice improved the appearance but gave little, if any, relief from discomfort. At the time of interview no affected individual was carrying out any treatment intended to cure or alleviate the condition.

Examination of individuals showed that the condition was virtually indistinguishable in members of all three families. The only point of difference appears to be that a minority of members of the family previously described and of Family T3 here reported had a thickening of the skin over the distal interphalangeal joints on their dorsal aspects, while none of the individuals in Family 1 showed such a condition.

Genetical Considerations.

For a number of reasons it was impossible to interview all the individuals who appear in the pedigree of Family 1—some are dead, some live in England and some in the U.S.A., while the members of the family resident in Ireland have lost touch with others—consequently, the accuracy depends to some extent on memory. However, as there is no clinical doubt as to whether a particular individual is affected or not, and as the information about dead or absent members was received independently from several members of the family interviewed, and in every case information from these different sources tallied exactly, it may be reasonably presumed to have a high degree of accuracy.

In Family 2 the affected mother was a foundling, who was deposited on the steps of an orphanage, and nothing is known of her parents.

The pooled sibships of the offspring of affected individuals in the two families show twenty affected and eighteen unaffected. (Ten affected males and ten unaffected; ten affected females and eight unaffected.) This suggests the full manifestation of a single dominant gene transmitted entirely in accordance with theoretical expectation.

Incidence.

In Northern Ireland at the present time there are thirty-six known living persons showing this trait, as detailed in Table 1. As the population of Northern Ireland is about 1,371,000, this gives an incidence of about 1 in 40,000 in the population.

TABLE 1.

	Males	Females	Total
Stevenson and Pearson family and sporadic case (T1)	11	11	22
Family T2 here reported	3	6	9
Family T3 here reported	2	3	5
Total	16	20	36

It is not possible to calculate a mutation rate because any fertility reduction would be too small to be detectable. Unfortunately, the linkage data are too incomplete in Family T3 to demonstrate linkage, but they are reproduced as they may be of value taken together with data collected subsequently elsewhere.

Summary.

Two families are described where tylosis palmaris et plantaris has been transmitted by a dominant gene mechanism.

An incidence rate of 1 in 40,000 for this condition in the population of Northern Ireland has been calculated with some confidence.

Reference.

Penrose, L. S. (1949). *The Biology of Mental Defects.* London: Sidgewick & Jackson.

I wish to express my thanks to Professor A. C. Stevenson of the Department of Social and Preventive Medicine of the Queen's University of Belfast, without whose assistance and criticism the preparation of this report would not have been possible. My thanks are also due to Miss C. Crozier, who did the blood typing, and to Dr. A. E. Mourant, who supplied the sera. Finally, some of the expenses for this survey were met from a grant to the Department of Social and Preventive Medicine by the Nuffield Foundation.

Appendix overleaf (page 30).

APPENDIX.

Family T_2. All living sibs were seen; I (2) was a foundling who knew nothing of her parents and all efforts at tracing them through an orphanage failed.

LINKAGE DATA.

No.	Ref.	A	Sex	CV	Taste	ABO	Rh	Lea	Leb	Lua	K	Fya	P	MNS	Sec.
T_2	I 1	U	M	+	+	0	R_1R_2	−	..	−	−	..	+	MNS	..
	2	A	F	+	+	0	rr	−	..	−	−	..	+	MNS	..
	II 1	A	F	+	−	0	R_1r	−	..	−	−	..	+	MNS	..
	2	A	F	+	+	0	R_1r	−	..	−	−	..	+	MNS	..
	3	A	M	..	..	..	..	..	..	..	..	..	..	..	..
	5	A	F	−	+	0	R_2r	−	..	−	−	..	+	MNS	..
	6	A	M	+	−	0	R_2r	−	..	−	−	..	+	NNS	..
	7	U	M	+	+	0	R_2r	−	..	−	−	..	+	NNS	..
	9	A	F	+	+	0	R_1r	−	..	−	−	..	+	NNS	..
	10	A	M	+	+	0	R_1r	−	..	−	−	..	−	MNS	..

Family T_3. I (1) had some sibs and stepsibs, their number, condition and that of their parents could not be discovered. Those seen were II (3), (4), (6), (8), and (9); III (12), (19), (21), and (23); IV (1), (2), (3), and (4).

(Some of these individuals reside in the Irish Republic, hence the apparent discrepancy in numbers recorded in Table 1.)

I (1), II (1), and (10) and III (15), (16), (18), and (21) are dead.
II (2), (5), and (7) and III (1), (2), and (3) reside in England.
III (4-11) are in U.S.A. and, although their father was seen, he was rather elderly and confused, and only knew that he had eight children, all unaffected.

LINKAGE DATA.

No.	Ref.	A	Sex	CV	Taste	ABO	Rh	Lea	Leb	Lua	K	Fya	P	MNS	Sec.
T_3	II 3	U	M	+	−	A_1	rr	..	..	−	−	..	+	MMs	..
	4	A	M	+	−	0	rr	−	+	−	−	+	+	MMs	..
	6	A	F	+	+	A_1	rr	..	..	+	−	..	+	MMs	..
	8	A	M	+	−	A_1	rr	..	..	−	−	..	+	MMs	..
	9	U	M	+	−	A_1	rr	..	..	+	−	..	+	MMs	..
	III 12	U	M	+	−	0	rr	−	+	−	−	+	+	MMs	..
	19	A	F	+	+	0	R_1r	..	..	+	−	..	−	MMs	..
	21	A	M	+	−	A_1	rr	..	..	−	−	..	−	MMS	..
	23	U	M	+	−	0	R_1r	..	..	−	−	..	+	MMs	..
	IV 1	A	F	+	+	0	R_1R_1	..	..	−	−	..	+	MMs	..
	2	U	M	+	+	0	R_1R_1	..	..	−	−	..	+	MNs	..
	3	U	F	+	+	0	rr	..	..	+	−	..	+	MMs	..
	4	A	M	+	?	0	R_1r	..	..	−	−	..	+	MMS	..

Subcutaneous Emphysema in Bronchial Asthma

By R. J. KERNOHAN, M.D., M.R.C.P., D.P.H., D.C.H.

Waveney Hospital, Ballymena

THE following case is reported to draw attention to the occurrence of subcutaneous emphysema in bronchial asthma.

CASE REPORT.

A man, aged 28 years, developed an attack of asthma on 8th October, 1955. He had been subject to asthmatic attacks from early infancy. The attacks had been recurring with decreasing frequency, and the previous severe attack was five years previously. Treatment at home failed to relieve his symptoms, and he was admitted to hospital on 12th October, 1955.

On examination, he was in acute respiratory distress. He was sitting upright, sweating profusely, cyanosed, and gasping for breath. The left side of the face was swollen. There was marked subcutaneous emphysema about the neck, and over the anterior chest wall. The air entry was poor, with marked prolongation of expiration. There were no crackling rales along the mediastinal border or synchronous with the heart beat. The blood pressure was 130/80. The pulse rate was 120 per minute and regular.

X-ray chest revealed subcutaneous emphysema in the supraclavicular regions. There was no evidence of pneumothorax or mediastinal emphysema.

He was treated with intravenous aminophylline, adrenaline hydrochloride (1 : 1,000) subcutaneously, and prednisolone. Within forty-eight hours he had improved considerably. The emphysema gradually subsided, and he was discharged home after two weeks in hospital.

COMMENT.

Subcutaneous emphysema is due to air escaping into the subcutaneous tissues. The Macklins have shown that air in the extra-pulmonary spaces originates from an initial pulmonary interstitial emphysema. They have demonstrated that pulmonary interstitial emphysema results from rupture of the "marginal" type of alveoli. The bases of "marginal" alveoli rest against structures such as bronchi, bronchioles, blood vessels or pleura, in contrast to "partitional" alveoli whose bases rest against other alveoli with which there is intercommunication by means of pores. Over-expansion of alveoli, without a corresponding widening of the lumen of the blood vessels, results in the formation of a pressure gradient which may cause leakage of air. The air seeps through multiple small ruptures in the alveolar bases, and enters the pulmonary interstitial tissue. The blood vessels underlying the alveolar bases become compressed, and the pulmonary circulation is impeded. The air then travels along the vascular sheaths to the mediastinum.

This may cause circulatory embarrassment due to pressure on the heart and mediastinal vessels. From the mediastinum the air may escape into the neck and subcutaneous tissues. Rupture may occur into the pleural cavity, causing pneumothorax. Occasionally the air may pass downward along the aorta into the retroperitoneal space.

The clinical findings depend upon the amount and position of the ectopic air. The symptoms and signs of mediastinal emphysema may be obscured by the co-existence of subcutaneous emphysema. Mediastinal emphysema is diagnosed by the combination of substernal pain, a peculiar crunching sound synchronous with the heart beat (Hamman's sign), radiological evidence of air in the mediastinum, and sometimes subcutaneous emphysema. When mediastinal emphysema is marked, subcutaneous emphysema tends to be slight, and vice versa. The escape of air into the subcutaneous tissues of the neck may lead to sudden improvement in the clinical condition, such escape having a decompression effect upon the mediastinal structures.

Spontaneous mediastinal and subcutaneous emphysema have occurred in influenza, pneumonia, pulmonary tuberculosis, diphtheria, after trauma to the chest, by lung blast from explosions, and following excessive coughing. This complication is very rarely encountered in status asthmaticus. There is no indication for surgical interference. Air infiltration into the tissues ceases, and within two to three weeks the air is completely absorbed.

Summary.

A case of subcutaneous emphysema complicating status asthmaticus is described. The causation of this complication is briefly discussed.

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Unrecognised Rib Fracture Simulating Pleurisy

By W. T. WARMINGTON, M.D.
Enniskillen Chest Clinic, Cornagrade, Enniskillen

WITHIN fifteen months three patients with unsuspected rib fracture were referred to Enniskillen Chest Clinic because of chest symptoms. The first rib was fractured in two cases, and the tenth rib in the remaining one. All fractures were on the right side.

Two case histories are given :—

(1) Miss M. McA., aged 32, consulted her doctor because of violent cough and acute chest pain, diagnosed as right-sided dry pleurisy, followed after one week by swelling and joint pains in the lower limbs. Two weeks later, on 3rd February, 1956, an X-ray at the Chest Clinic showed fracture of the tenth rib on the right side, with some displacement of fragments. Callus formation was well marked on a film taken 17th March, 1954. No pulmonary abnormality was present on either film. On questioning, she recalled that she had slipped while pushing a car about the time of the onset of her chest pain. Although she "wrenched herself," no impact was sustained. On 3rd February, 1956, the B.S.R. was normal (7.0 Cutler 1 hour) and the Mantoux test negative to 1 mgm. old tuberculin. The fracture was considered to have been the cause of her symptoms.

(2) John L., aged 19, took influenza on 14th March, 1955, for which he spent one week in bed. He had slight cough and sputum. Two weeks after onset he had pain in the right scapular region, at first worse on moving, but later on coughing. This persisted until he was seen at the Chest Clinic on 10th October, 1956, having complained of right infra clavicular pain three-four days previously. On examination, no swelling or tenderness was found, but chest X-ray showed a fracture of the first rib on the right side. B.S.R. was normal (5.0 Cutler 1 hour) and Mantoux positive to 0.1 mgm. old tuberculin. The infra clavicular pain for which he was referred was due to this fracture. Both instances of fracture of the first rib occurred without known cause in right-handed muscular young men.

These cases show that unsuspected rib fracture must be borne in mind as a possible, though rare, cause for chest pain associated with cough.

REVIEWS

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FOR this new edition the work has been substantially rewritten and new subjects discussed include Genetical Aspects of Child Health, Child Guidance, Vocational Guidance, Health Education, Punishment, and Child Health in the Tropics. Many of these, and the other activities discussed in Part II—Social Aspects of Child Health, are now also the concern of a large number of trained lay workers employed by the State and Local Authorities. If medical men are to continue to interest themselves and advise on all that concerns the health and well-being of children they must also be informed on these activities. Nowhere else will they find so much useful information on these topics collected and discussed within a reasonable space.

About half of the book is devoted to development, and this includes chapters on prenatal development, the newborn, digestion, nutrition and feeding, feeding habits, postnatal growth, puberty and adolescence, intellectual, emotional and instinctive development and immunity. Throughout the book is concerned with the healthy child, his physical and mental growth and his place in society. It is not directly concerned with the diseased child.

The editor is responsible for five of the twenty-three chapters and for a broad-based and stimulating introduction. He has had the assistance of sixteen other contributors. The outcome is an up-to-date book, well edited, and providing the practitioner with information both for everyday application and for reference.

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Medical men, as well as those engaged in research on vitamins and nutrition, will welcome this number. Some of the contributions are highly technical, but much information of value to those concerned with applied nutrition is also provided. A valuable article, "Vitamins in Nutrition: Orientations and Perspectives," by B. S. Platt, and an article on the B vitamins in the blood and intestinal tract by L. J. Witts are among those of greatest interest to medical men. A useful review of the effect of processing on the vitamin content of food is provided by L. W. Mapson, and the more specialised reader will be interested in the report on the chemistry and mode of action of vitamin B_{12}.

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F. A. MacL.

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Whilst agreeing that chloramphenicol is often effective in the treatment of "gastro-enteritis," the reviewer feels that some mention should have been made of its potential toxicity to the bone-marrow and liver.

The discussion on wasting in infancy is unfortunate in that it fails to make any reference to important and well-recognised metabolic disorders such as hypercalcæmia and renal acidosis which often masquerade as "feeding problems" with failure to thrive, for which the authors suggest "improving the hygiene and nursing care." In such cases an estimation of the serum calcium and alkali reserve might provide the diagnosis and indicate the correct treatment.

With the above reservations, this book can be recommended as a helpful guide to all those concerned with the feeding of babies and young children. The type and illustrations are good, there is a complete and accurate index, and by present standards the price is reasonable.

R. D. G. C.

PSYCHOLOGICAL MEDICINE.

By Desmond Curran, M.B., F.R.C.P., D.P.M., and Maurice Partridge, M.A., D.M., D.P.M. Fourth Edition. (Pp. viii + 407; figs. 20. 21s.) Edinburgh: E. & S. Livingstone, 1955.

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To the preparation of the fourth edition Dr. Curran brings the authority and experience of those intervening twelve years and his collaborator is a worthy successor to the late Dr. E. Guttmann. The aim, as in earlier editions, is to present the elements of psychiatry to medical students and to the practitioner requiring a concise and up-to-date account of psychological medicine. Wisely, a plethora of technical terms has been avoided and, although the authors do not hesitate to express personal opinions, the subject matter is presented in a balanced and objective manner, the clinical approach predominating.

The book has been extensively revised, the treatment of alcoholism and schizophrenia receiving more attention and the section on psychiatry in war time being omitted.

Most of the illustrations, including examples of schizophrenic drawings, to the uninitiated so very like certain forms of contemporary art, have appeared in earlier editions, but are none the less worth retaining.

This book is what it claims to be, namely, a short Introduction to Psychiatry. Those who require more than that will be disappointed, but no medical student or busy general practitioner should be without it.

J. B. N.

BRITISH JOURNAL OF TUBERCULOSIS AND DISEASE OF THE CHEST.

Vol. 50, No. 1. (Pp. 1-104. 12s. 6d.) London: Baillière, Tindall & Cox, 1956.

WE welcome this Golden Jubilee number. It is a proud record for a specialist journal and the successive editors and the publishers are to be congratulated. The enlarging interest of the journal and the decision in 1943 to include all diseases of the chest was timely, and we hope the journal will continue to survive the hazards of medical publication.

The present number is mainly occupied with "Fifty Years On—Then and Now," and includes a series of articles reviewing progress in these islands and throughout the Commonwealth.

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Vol. 50, No. 1. (Pp. 1-104. 12s. 6d.) London: Baillière, Tindall & Cox, 1956.

WE welcome this Golden Jubilee number. It is a proud record for a specialist journal and the successive editors and the publishers are to be congratulated. The enlarging interest of the journal and the decision in 1943 to include all diseases of the chest was timely, and we hope the journal will continue to survive the hazards of medical publication.

The present number is mainly occupied with "Fifty Years On—Then and Now," and includes a series of articles reviewing progress in these islands and throughout the Commonwealth.

MODERN METHODS OF FEEDING IN INFANCY AND CHILDHOOD.

By Donald Paterson, B.A., M.D., F.R.C.P., and George Newns, M.D., F.R.C.P. Tenth Edition. (Pp. ix + 188; figs. 15. 15s.) London: Constable, 1956.

THE most recent edition of this book presents a clear picture of orthodox British practice in the feeding of young children. The section on breast feeding is excellent and incorporates several recent advances in the physiology and management of lactation. Infants who have the good fortune to be artificially fed on the lines put forward by the authors should never go hungry, for Drs. Paterson and Newns recommend three ounces of milk per pound body-weight daily against the traditional "two and a half ounces per pound per day" generally thought to be sufficient.

Whilst agreeing that chloramphenicol is often effective in the treatment of "gastro-enteritis," the reviewer feels that some mention should have been made of its potential toxicity to the bone-marrow and liver.

The discussion on wasting in infancy is unfortunate in that it fails to make any reference to important and well-recognised metabolic disorders such as hypercalcæmia and renal acidosis which often masquerade as "feeding problems" with failure to thrive, for which the authors suggest "improving the hygiene and nursing care." In such cases an estimation of the serum calcium and alkali reserve might provide the diagnosis and indicate the correct treatment.

With the above reservations, this book can be recommended as a helpful guide to all those concerned with the feeding of babies and young children. The type and illustrations are good, there is a complete and accurate index, and by present standards the price is reasonable.

R. D. G. C.

PSYCHOLOGICAL MEDICINE.

By Desmond Curran, M.B., F.R.C.P., D.P.M., and Maurice Partridge, M.A., D.M., D.P.M. Fourth Edition. (Pp. viii + 407; figs. 20. 21s.) Edinburgh: E. & S. Livingstone, 1955.

THE first edition of this book was published in 1943 at a time when physical methods in the treatment of mental illness were coming to the fore and long-established concepts were being re-examined in the light of knowledge gained in the midst of total war.

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FRACTURES OF THE FACIAL SKELETON. By N. L. Rowe, F.D.S.R.C.S., L.R.C.P., M.R.C.S., L.M.S.S.A., H.D.D.R.C.S., and H. C. Killey, F.D.S.R.C.S., L.R.C.P., M.R.C.S., L.M.S.S.A., H.D.D.R.C.S. (Pp. xxxvi + 923; 1,231 illustrations, 14 in colour. £6.) Edinburgh: E. & S. Livingstone, 1955.

SIR REGINALD WATSON-JONES, in a foreword, describes this book as monumental and likely to become one of the classics of surgery. Your reviewer entirely agrees. Its scope is encyclopædic and, in addition to a consideration of the fractures of the mandible and maxilla, there are sections on associated injuries of the scalp, skull, brain, and eye; a section on the special problems of anæsthesia in maxillo-facial injuries, one on radiography, another on gunshot wounds of the face and jaws, one on the treatment of the residual traumatic deformities, one on facial lacerations, and a final chapter on the historical background. The joint authors are the consultants in oral surgery at the Plastic and Maxillo-Facial Surgery Unit at Basingstoke, but contributions have been made by, amongst others, Patrick Clarkson, Sir Harold Gillies, and D. T. Gilchrist of Belfast. Considerable reference is made in the section on head injuries to the work of C. A. Calvert.

Although large, the book is not verbose, and is obviously based on a wide and extensive experience of facial injuries. The style is lucid and the English so good that an occasional lapse (page 226, last sentence) is the more noticeable. The authors' attention to detail and their descriptions of the practical procedures reveal the care they have taken to perfect their operative technique—as, for example, in the paragraphs on the preparation of eyelet wires (page 68). The text is dogmatic, at times too much so, and the definitive statement of the date of the death of Homer (page 847) would come as a surprise to most classical scholars. The fixation time of six weeks for a simple fracture of the mandible (pages 81, 118) seems somewhat long, and the use of antibiotics is recommended rather too frequently. The illustrations are superb, not so much the clinical photographs as the diagrams of the underlying skeletal structures and the relationship of the bone to fracture lines or displacement. The book is well produced in clear type and has an extensive bibliography.

Altogether an outstanding book. P. J. S.

DISEASES OF THE NOSE AND THROAT. By Sir St. Clair Thomson, M.D., F.R.C.P., F.R.C.S.; revised by V. E. Negus, D.Sc., M.S.(Lond.), F.R.C.S., with the assistance of G. H. Bateman, M.A., B.M., F.R.C.S. Sixth Edition. (Pp. xvi + 1,040; figs. 353. 90s.) London: Cassell, 1955.

THE sixth edition has been produced instead of reprinting the fifth edition, which was exhausted.

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Woodman's operation for bilateral laryngeal paralysis is described in some detail. It is now thought by the author that improved results are now being obtained by operation, although he still favours a valve tracheostomy tube in most cases. The operation of pharyngo-œsophago-laryngectomy is described in detail. The improvement in the prognosis of laryngeal tuberculosis with the widespread use of streptomycin is noted. Streptomycin is also advised as the best treatment for scleroma.

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In criticism it would appear that the author advocates the formation of units requiring very large capital expenditure, but a more careful survey shows that he recommends the gradual building up of the unit till the point is reached where between 85 per cent. and 90 per cent. of the patients in hospital are daily occupied in some form of occupational therapy.

I have no doubt that this work will be most beneficial to all whose work takes them in contact with mental hospitals. If the principles set out are followed, not only will a great change be apparent in the mental health of those in hospital, but all employees of the hospital, from doctors and nurses to tradesmen and domestic staff, will find new interest in their work, and will approach their patients with a new understanding. J. A. S. M.

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Dr. O'Sullivan gives a clear history of the development of occupational therapy in the United Kingdom, America, and on the Continent.

The application of this form of treatment in a mental hospital is the main theme of the text, but the practical, economic, and commercial sides of the activities are given full consideration. All the activities as described are directed towards bringing the patient in contact with reality and establishing in the hospital a social and community spirit so essential in helping towards recovery.

Occupational therapy is so frequently regarded as the simple employment of a number of patients in various handicrafts that it is refreshing to find equal emphasis being placed on recreational, social, and re-educational activities.

A chapter is devoted to the re-education of regressed patients and describes in detail the planning of a scheme for their rehabilitation.

For those engaged in the administration of hospitals the chapters dealing with administrative personnel, the formation of special occupational units and the use of occupational therapy in the wards is extremely helpful.

For non-medical personnel the sections describing mental illnesses and the selection of special classified occupational activities for the various types of illness should be of great value. Not all activities in occupational therapy can be described in detail in one text-book, but the author gives a comprehensive analysis of willow craft, cane craft, wood craft, and weaving.

In criticism it would appear that the author advocates the formation of units requiring very large capital expenditure, but a more careful survey shows that he recommends the gradual building up of the unit till the point is reached where between 85 per cent. and 90 per cent. of the patients in hospital are daily occupied in some form of occupational therapy.

I have no doubt that this work will be most beneficial to all whose work takes them in contact with mental hospitals. If the principles set out are followed, not only will a great change be apparent in the mental health of those in hospital, but all employees of the hospital, from doctors and nurses to tradesmen and domestic staff, will find new interest in their work, and will approach their patients with a new understanding. J. A. S. M.

REFRESHER COURSE FOR PRACTITIONERS. Specially contributed articles from the Journal of the Indian Medical Association. Volume 1. (Pp. xiv+364. Rs.8.) Calcutta: Journal of the Indian Medical Association.

THIS volume contains a collection of articles on certain diseases commonly encountered in general practice. They have been reprinted from the Journal of the Indian Medical Association and follow the lines of similar articles in the British Medical Journal.

The subjects considered include certain cardiovascular and pulmonary diseases, anæmia, tropical diseases, such as malaria, cholera, and amœbiasis, burns and senile enlargement of the prostate.

The information conforms in general with that in standard text-books and directions for treatment are both comprehensive and precise. W. G. F.

THE BOKE OF CHYLDREN By Thomas Phaire. Edited by A. V. Neale, M.D., and H. R. E. Wallis, M.D. (Pp. 76; plate 1. 7s. 6d.) Edinburgh and London: E. & S. Livingstone, 1955.

THIS book, the first written on children's diseases by an Englishman and published in 1545, is of particular interest to the pædiatrician and medical historian. The honest philosophy and keen observation of Thomas Phaire are readily apparent from his writings, while his "physicke" has a contemporary and fully botanical flavour. O. D. F.

BODY FLUIDS IN SURGERY. By A. W. Wilkinson, M.Ch., F.R.C.S. (Pp. ix+212; figs. 6. tables 29. 16s.) London and Edinburgh: E. & S. Livingstone, 1955.

SUFFICIENT knowledge of the physiological mechanisms which regulate the volumes and composition of the body fluids now exists to permit a logical approach to therapy of fluid and electrolyte imbalance. This book aims to set out, in reasonable compass, the aspects of this subject of particular interest to surgeons in the management of their clinical problems. In the main, this purpose is achieved. The author divides his subject into chapters on the body fluid, the chief body cations, acid-base balance, and the effects of shock and various types of fluid and electrolyte imbalance. Each chapter commences with a short physiological introduction, follows with a discussion of the causes of, and the clinical and metabolic results of derangement of body chemistry, and concludes with a section on treatment. This presentation is satisfactory, and avoids undue repetition in the various chapters.

In the discussion on the exchanges of fluid between the extracellular and the intracellular phases, insufficient emphasis is laid on the importance of the osmotic regulation of the movement of water between the body compartments. This concept is so fundamental that it might be considered more fully and illustrated diagramatically. In the section devoted to the treatment of hyponatræmia and cellular overhydration, the author correctly advocates the administration of hypertonic saline; but he does not indicate that the amount of salt, in excess of water, that is required to achieve correction may be calculated as the product of the fall in sodium concentration below normal multiplied by the total body water (estimated as 60 per cent. of the body weight).

The chapter on potassium contains two errors of statement concerning the metabolic consequences of potassium depletion. On p. 51 it is stated that Black and Milne observed extracellular acidosis in two subjects depleted of potassium by dietary restriction, whereas these workers showed that extracellular alkalosis resulted. Fourman and Ainley-Walker produced potassium depletion and acidosis, but in their study the depletion was induced by the ingestion of an exchange resin in the ammonium cycle. Uncomplicated potassium depletion is always accompanied by an alkalosis. On p. 48 it is stated that the shift of sodium into cells that occurs in potassium depletion results in a contraction of the extracellular fluid. This only occurs if sodium intake is simultaneously restricted. Potassium depletion is usually accompanied by retention of sodium in, and expansion of, the extracellular fluid. Clinically, œdema may occur. In the treatment of potassium depletion more stress might be laid on the fact that in many instances of potassium loss there may also be severe losses of salt, with resultant contraction of the extracellular fluid volume. In these circumstances, the initial concentration of potassium in the serum may be well above normal, despite an overall potassium deficit. Appreciation of this fact would lead to modification of the advice that in pyloric stenosis, "The replacement of potassium is therefore the primary consideration, and potassium chloride should first be administered, followed by a mixture of potassium and sodium chloride" (p. 111). Such a line of treatment would lead to a dangerous hyperkalæmia in a significant proportion of cases.

The concluding chapters on "The influence of associated disease on fluid and electrolyte balance," on "Diagnosis," and on "Treatment" are satisfactory, except that it is doubtful whether the scheme for calculating losses of fluid and electrolyte given on pp. 152-153 would be very useful in practice.

R. W.

NURSING CARE OF THE NEWLY BORN INFANT. By W. S. Craig, B.Sc., M.D., F.R.C.P.(Edin.), M.R.C.P., in collaboration with M. F. G. Buchanan, M.B., B.Ch., M.R.C.P.(Edin.), and R. J. Pugh, M.B., Ch.B., M.R.C.P., and M. Pattullo, R.G.N., S.C.M. (Pp. viii+472; figs. 224. 35s.) Edinburgh and London: E. & S. Livingstone, 1955.

THIS book covers fully that most important period, the first month of life. It contains much practical advice and is especially written for nurses. This volume should be of particular value for the pupil or practising midwife and children's nurse; it should be read also by the candidate for the Diploma of Child Health.

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A HANDBOOK OF MEDICAL HYPNOSIS. By Gordon Ambrose, L.M.S.S.A., and George Newbold, M.B., B.S., M.R.C.S. (Pp. xiv + 256. 21s.) London: Baillière, Tindall & Cox, 1956.

A BRIEF history of medical hypnotism is given, following the development of the subject from the religious cults of Ancient Egypt through such workers as Mesmer in the latter half of the eighteenth century, John Elliotsen and the Edinburgh surgeon, James Braid (1795-1860).

The hypnotic state is considered as consisting of three stages, namely, light, medium, and deep or somnambulistic. In the latter stage amnesia for events occurring during hypnosis, sensory hallucinations and pronounced effects from post-hypnotic suggestion are among the many manifestations which appear.

Technique in the methods of induction of hypnosis are briefly described with a short consideration of auto-hypnosis.

The remainder and greater proportion of the volume is devoted to the application of hypnosis in diverse fields of medicine and surgery. A range of subjects from tics to tuberculosis, amenorrhœa to accouchement is covered with a large section devoted to gynæcology and obstetrics. Each section is illustrated with one or more case-histories. These are in many instances scanty, and one might wonder about the diagnosis in the case of "the attractive young woman of 30" with disseminated sclerosis whose "lower limb reflexes were absent." Clinical investigations, if they have been done, are not quoted in any instance.

Not all observers would agree with the statement (p. 79) that in anorexia nervosa "death may occur from cardiac failure, tuberculosis or coma, but *fortunately most cases recover.*" Nor would most gynæcologists be prepared to accept a psychogenic basis for menorrhagia in the presence of pathology of the uterus and adnexa such as fibroids (p. 185). The authors also state that "hypnotherapy is indicated if the patient suffers from heart disease such as mitral stenosis, and is allowed to proceed to term. This is to ensure that labour becomes less of a strain on the cardiac muscle." It is fairly generally known that such cases, if they do not decompensate during pregnancy, have an uneventful labour. There are few obstetricians who would consider hypnotherapy as an adjuvant in the management of pre-eclampsia.

There are numerous controversial statements of this nature. Hypnotherapy is embarked upon in many instances with what would appear to be insufficient appreciation of the organic aspects of the condition, e.g., failure to carry out radiography or exclude congenital anomalies in cases of enuresis.

Bibliography is scant and references in some places incomplete (pp. 49 and 75). The authors stress that this is a hand-book and not a text-book. As such, it is scarcely in the tradition of volumes like that of Hutchinson and Hunter. It is easily readable, but one is left with a confused impression of the value of hypnosis in medicine. It would appear to be a panacea for an extremely wide spectrum of disease if one were to accept the claims made in this book. The cases are poorly documented and no comparison with treatment by other and better established methods is attempted.

E. M. A.

OBSTETRICS AND GYNÆCOLOGY FOR NURSES. By G. W. Garland, M.D., M.R.C.O.G., and J. M. E. Quixley, S.R.N. (Pp. xii + 188; figs. 58. 10s. 6d.) London: English Universities Press, 1956.

THE authors have had a happy thought in including some chapters on obstetrics in a text-book of gynæcology, thereby giving the nurse a proper perspective of the function of the female generative organs.

Possibly the chapters on obstetrics are unnecessarily full to "help the nurse in an occasional obstetric emergency" as the authors hope, but the section on gynæcology is very good. The writing is clear and to the point, with good, simple diagrams, and a useful glossary of the special terms used gives the reader a good start.

This is a good text-book for the student nurse and a useful book of ready reference for those in charge of gynæcological wards.

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