recorded, more frequently for type III than for types I and II, but less consistently and of a lower order than that recorded above. However, the majority of viruses examined by Sabin were isolated early in excretion; in common with our finding, increased neurotropism was found most often at a later stage of excretion. Nevertheless, Sabin considered that such excreted viruses were less highly neurotropic than wild polioviruses disseminated by healthy children during non-epidemic periods.

It is a matter of opinion whether all the viruses excreted by the children in our trial were less virulent than strains encountered in nature. But the degree of alteration from the original vaccine strain appeared to be considerable. In this regard our observations seem to be similar to those recorded by Dane *et al.* (1957) with rodent-adapted type II virus and by Dick *et al.* (1957) with modified type I virus (Koprowski *et al.*, 1956). It will be recalled that Dick and Dane (1957) stated that these virus strains were unacceptable for mass immunization because of their alteration in virulence during passage through the alimentary tract. In spite of the mass use of living attenuated poliovirus vaccine in the Belgian Congo (Courtois *et al.*, 1958) it is our opinion that further research concerning the properties of the viruses excreted by those receiving such vaccines is desirable in order to reconcile the conflicting evidence.

Summary

Fourteen children who were patients at a long-stay orthopaedic hospital were immunized with two doses of British inactivated poliovirus vaccine and were later given living attenuated type III virus vaccine (Sabin) by mouth in gelatin capsules. No adverse clinical reactions were observed. Five of the children developed an alimentary infection, as shown by excretion of virus in the stools for from 9 to 30 days, and in three instances by a further increase in antibody titre. No instance of spread of excreted virus to contacts was detected. The vaccine virus and some of the viruses excreted early in the course of infection did not produce paralytic effects in monkeys inoculated intracerebrally. All five infected children excreted strains of virus possessing enhanced neurotropic properties for monkeys at a late stage of the infective process.

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COMBINED GASTRIC AND DUODENAL ULCERATION

A SURVEY OF 157 CASES

BY

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From the time Sir David Wilkie (1926) first reported on combined ulceration of the stomach and duodenum there has been a considerable divergence of opinion in respect to its incidence. This is revealed in the recent observations by Feldman (1953) and by Johnson (1955). From their papers it would appear that surgeons as a rule observe a much higher proportion than is found in either post-mortem or radiological studies. Johnson also drew attention to gastric retention as leading to gastric lesions in patients suffering from chronic duodenal ulcer. The present study shows the incidence of combined ulcers as encountered in a general hospital, and gives some additional information on duodenopyloric stenosis causing secondary ulcer of the stomach. It also reveals the increased danger of bleeding, making surgical treatment more advisable than with a single ulcer.

Material

The 157 cases reported here were collected from the files of the in-patient and out-patient clinics of the Central Middlesex Hospital, London. Only cases with combined ulcers first diagnosed in the years 1947 to 1954 were included. Definite radiological, surgical, or post-mortem evidence of chronic ulceration was required. Acute ulcers were excluded. In all doubtful cases the x-ray films were reviewed. While the work proceeded, it became increasingly obvious that cases with antral, prepyloric, and pyloric ulcers were not suitable for analysis. There would often arise considerable doubt about the exact location of the ulcer. Extensive scarring due to prepyloric lesions will sometimes lead to secondary duodenal deformity, particularly after perforation. For this reason, only gastric ulcers at or above the angulus were included.

Incidence

During the eight-year period from 1947 to 1954, 157 cases showing both gastric and duodenal ulcers or ulcer scars were observed. In 139 of these, both ulcers were diagnosed radiologically. During the same period 3,744 duodenal and 1,352 gastric ulcers were detected by x-ray study in the hospital, excluding the combined lesions. Based on x-ray findings, the observed incidence is 2.7%. As mentioned above, cases with prepyloric ulcers were not included in the combined ulcer group. Ihre and Müller (1943) observed 26% of all benign gastric ulcers to be prepyloric. Many authors give figures lying between 15 and 20%, but Gott,

TABLE I.—Incidence of Combined Ulcers Based on X-ray Investigation (Central Middlesex Hospital, 1947–54)

No. of Cases With Single Ulcers	Combined Ulcers (Prepyloric Ulcers Excluded)		Combined Ulcers (Estimated 25% Prepyloric Ulcers Included)	
	No.	Incidence	No.	Incidence
D.U. . . 3,744	139	$\frac{139 \times 100}{3,744 + 139} = 3.6\%$	185	$\frac{185 \times 100}{3,744 + 185} = 4.7\%$
G.U. . . 1,352*		$\frac{139 \times 100}{1,352 + 139} = 9.3\%$		$\frac{185 \times 100}{1,352 + 185} = 12.0\%$
All ulcers 5,096	139	$\frac{139 \times 100}{5,096 + 139} = 2.7$	185	$\frac{185 \times 100}{5,096 + 185} = 3.5$

* Including prepyloric ulcers.

Shapiro, and Kelty (1954) found 42% of gastric ulcers below the angulus. If an estimated 25% of prepyloric ulcers are added, the number of combined ulcers would increase to 185 and the incidence to 3.5% (Table I). Thus one in every eight hospital patients suffering from an ulcer of the stomach and about 1 in 30 patients having a duodenal ulcer or ulcer scar may be expected to have an associated ulcer of the other organ as well.

It has been suggested by Johnson (1955) that many combined ulcers may be missed on x-ray examination, thus decreasing their apparent proportion. In this series, 18 cases were not diagnosed by x-ray examination. In six of these no barium-meal study was performed at any time before surgery or necropsy disclosed the ulcers. In most cases an emergency situation such as bleeding or perforation was responsible for this omission. In another six cases the last radiological investigation had taken place two to six years before gastric surgery revealed an additional ulcer not shown before. Judging from the symptoms during the interval, the presence of combined ulcers at the previous examination is unlikely. In only five patients was one of the ulcers certainly missed. There were duodenal scars in four of these patients, and an active duodenal ulcer in one, accompanied by a considerable degree of pyloric stenosis. In addition, one gastric ulcer was possibly not detected two months before it was found at an emergency operation for bleeding. In no case examined radiologically were both ulcers missed. Thus the x-ray findings were incomplete in less than 5%. It is believed that careful x-ray examination provides reliable data for estimating the incidence. The high proportion of positive diagnoses reflects the particular care taken in the hospital's x-ray department in respect of barium-meal studies as well as the awareness of the frequent concomitance of ulcers. In the majority of cases the radiological diagnosis was confirmed by surgery or gastroscopy or both.

Time Sequence of Ulcer Appearance

The sequence is of some importance in aetiological considerations on combined ulceration. Previous observations by Feldman (1953) and Johnson (1955) showed that a proved chronic duodenal ulcer preceded the appearance of the gastric ulcer in most patients. This sequence took place in 91 patients (group 1), of whom 20 were women (Table II).

TABLE II.—Sequence of Ulcer Appearance and Sex Incidence

Group	Sequence	Total	Men	Women	Sex Ratio
1	D.U.→G.U.	91	71	20	3.6:1
2	"Simultaneous" G.U.+ D.U.	25	19	6	3.2:1
3	Sequence unknown	20	17	3	5.7:1
4	D.U.→G.E.→G.U.*	13	11	2	5.5:1
5	G.U.→D.U.	8	7	1	7:1
	All groups	157	125	32	3.9:1

* Including one vagotomy without gastro-enterostomy.

In 25 others the ulcer history was remarkably short, rarely exceeding two months. Still the first x-ray examination or surgery performed for complications showed both active duodenal and gastric ulcers (group 2). No sequence could be established in 20 patients with incomplete records or in cases where a long ulcer history preceded the first x-ray study which showed combined ulcers (group 3). Twelve patients underwent gastro-enterostomy for duodenal ulcer 1 to 25 years before a gastric ulcer at some distance from the anastomosis was discovered (group 4). Another patient also included in this group had submitted to a vagotomy without gastro-enterostomy for duodenal ulcer two years before an ulcer of the stomach appeared. Similar cases have been reported by Johnson (1955).

Evidence for a gastric ulcer preceding a duodenal ulcer was obtained in eight patients (group 5). The early x-ray films of these patients were reviewed by the radiologist, and the duodenal cap was found to be normal in the presence of a gastric ulcer in all instances.

Sex and Age Incidence

Of the 157 patients with combined ulcers, 32 were women, giving a sex ratio of 3.9:1 (Table II). This figure lies between the ratios of 2.4:1 in respect to gastric and 6.8:1 in respect to duodenal ulcers found during a somewhat earlier period at the Central Middlesex Hospital (Avery Jones and Pollak, 1945). There was little variation of this ratio in the different age groups in either this or the present study. Deviations in the small groups may be due to an inadequate number of cases.

The age incidence is represented in the lower half of the Chart, which shows the distribution at the time both ulcers were recognized. For comparison, the age distribution of duodenal and gastric ulcers at the same hospital is included in the upper half (Avery Jones and Pollak, 1945). It is readily seen that the age distribution in combined ulcers is very similar to the gastric ulcer group.

In patients with duodenal ulcer preceding gastric ulcer (group 1) the mean age at which the combined lesion was diagnosed was 54 years, with little variation between the sexes. Symptoms of peptic ulcer appeared at the much earlier age of 39 years on the average.

However, the diagnosis of duodenal ulcer usually was established much later, after an interval averaging almost eight years. Many of the patients showed no free interval before the gastric ulcer appeared, making it impossible to date the onset of the latter. In some, however, there was either a relapse following a prolonged remission or a definite change in the character of the disease, and in these the interval between onset of symptoms and x-ray or surgical confirmation of gastric ulceration was quite short.

In the other groups the mean age at the time both ulcers were discovered similarly lies between 50 and 55 years. In group 5 the gastric ulcer was diagnosed on the average six years before the duodenal ulcer. Intervals ranged from some months to 14 years.

Age distribution. Top: duodenal and gastric ulcers (Avery Jones and Pollak, 1945). Bottom: combined ulcers.

Characteristics

Activity.—As judged by definite x-ray demonstration of an ulcer crater or by surgery, both ulcers were active together at any one time in 98 of the 157 patients. This incidence of almost two-thirds may reflect the severity of the disease in this series.

Free Interval.—A symptom-free interval lasting more than one year was present in 41 patients. This figure applies to the 112 patients with a known sequence only (groups 1, 4, and 5). In the eight cases showing the gastric-duodenal ulcer sequence (group 5) any free interval was always shorter than one year.

Change in Symptoms.—Most symptoms besides pain were too indefinite to allow investigation. Excluding groups 2 and 3 ("simultaneous" appearance or unknown sequence) as well as cases with incomplete records in this respect, enough notes on pain were available in 89 patients. Some definite change was noted in 42 of these, being a change in character of pain only in 29, a shift in site only in another 3, and a change in site as well as character in 10 patients. It appears

that in a minority only can combined ulceration be suspected on the basis of change in symptoms. The significance of vomiting is discussed below.

Site of Gastric Ulcer.—The ulcer was located at the angulus in 45 cases, at the middle third of the lesser curve in 82, and at the upper third in 30. In the presence of multiple ulcers the lesion appearing first was taken into account. No relation of the ulcer site to either age or sex could be established.

Multiple Ulcers.—These were present in 22 cases, with two gastric ulcers in nine cases, three gastric ulcers in three, and four gastric ulcers in one. Kissing duodenal ulcers were observed in nine instances.

Gastric Retention

With regard to pyloric stenosis, the following groups were established. (A) Occasional or no vomiting; no x-ray evidence of delayed emptying. (B) Frequent but transitory vomiting; little vomiting with considerable loss of weight; no x-ray evidence of delay. (C) Delayed emptying, later followed by normal emptying on x-ray investigation. (D) Pyloric stenosis, predominantly organic in nature, as a rule treated surgically.

Table III shows the respective distribution. Not included among the 131 cases are the patients with a former gastro-enterostomy. In these no delay in emptying appeared to be present, and some factor other than retention might be

TABLE III.—Incidence of Gastric Retention

Presence and Degree of Gastric Retention	No. of Patients	Incidence
A. No or rare vomiting; normal emptying	94	71.7%
B. Frequent vomiting or weight loss; normal emptying	12	9.2%
C. Transitory delay in emptying	13	9.9%
D. Organic pyloric stenosis	12	9.2%
Total (cases with gastric-duodenal sequence and G.E. or vagotomy excluded)	131	100%

responsible for gastric ulcer formation. Also excluded are the eight cases with the unusual gastric-duodenal-ulcer sequence and five cases with insufficient details. In group A gastric retention was presumably never present, and thus would not appear to play any part in the appearance of the gastric ulcer in more than 70% of the series. Even this figure may represent a conservative estimation, because it is by no means certain that frequent vomiting or loss of weight is indicative of decreased emptying. Severe pain and even fear of pain may give rise to both.

Other Complications

Of the 157 patients, 59 had a total of 89 haemorrhages. This incidence has to be considered in relation to duration of the disease as shown in Table IV. In the group of 91 patients with the known duodenal-gastric-ulcer sequence, nine suffered from haemorrhages while they had a duodenal ulcer only, the disease lasting on the average 12½ years. At

TABLE IV.—Incidence of Bleeding in Single and Combined Ulcers Related to Duration of Disease

	No. of Patients	Patients with Haemorrhage		No. of Haemorrhages	Average Duration of Disease in Years
		No.	%		
Duodenal ulcer (Group 1 only*)	91	9	10	11	15 { with haem., 12.5 without haem., 16.4
Combined ulcers (Group 1 only*)	91	27	30	36	1.4 { with haem., 1.1 without haem., 1.6
Combined ulcers (all groups)	157	45	29	64	
Total	157	59	38	89	13

* See Table II.

the time combined ulcers appeared there were 36 instances of bleeding in 27 patients. The haemorrhages then took place in a period barely exceeding one year. The gastric ulcer was proved to be the source of bleeding in 16 of these patients, while the others may have bled from either stomach or duodenum. Thus the incidence of bleeding appears much higher in the presence of combined ulcers in spite of their short coexistence.

In many instances the combined ulcer disease was terminated by partial gastrectomy, often after the first haemorrhage. This would alter the proportion of haemorrhages in respect of time. To obviate this difficulty a special group of 49 patients was investigated. These 49 cases could be followed for a period of at least one year after the diagnosis of gastric ulcer following duodenal ulcer was established by x-ray examination. Six patients suffered from a total of eight haemorrhages while they supposedly had a duodenal ulcer only, the disease lasting 722 patient-years in the 49 cases. On the other hand, during the period of combined ulceration with 130 patient-years, the number of haemorrhages was 12, with 10 patients bleeding. Thus there was one haemorrhage for 90 patient-years with single as compared with one for 11 patient-years with combined ulcers in this group. These findings correspond reasonably well with the data of all patients with the same sequence.

Age was not responsible for an increased incidence of bleeding, the patients with haemorrhages being on the average four years younger at the time both ulcers were discovered.

In all groups taken together there were 89 haemorrhages in the 157 patients, and at least every third patient bled at one time or another. Although in several instances it was difficult to correlate the bleeding accurately with the presence of either single or combined ulcers, in 45 patients at least the haemorrhage took place when both gastric and duodenal ulcers were present. Here again the much higher incidence is apparent, inasmuch as the bleeding took place during a period averaging little more than one and a half years. Some patients, however, may have bled from a lesion other than a gastric or duodenal ulcer, particularly in the group with a previous gastro-enterostomy.

The 24 perforations in 22 patients were with one exception duodenal. There were nine cases of hourglass deformity of the stomach.

If pyloric stenosis is included, 92 of the 157 patients thus suffered from one or more major complications—again a striking proportion. The complications are listed in Table V.

TABLE V.—Complications, Operations, and Deaths in 157 Patients With Combined Ulcers

	No. of Patients	Incidence
Gastric retention { Possible	12*	9%*
Proved	25*	19%*
Haemorrhage	59	38%
Perforation	22	14%
Hourglass stomach	9	6%
One or more complications	92	59%
Operations { Partial gastrectomy	68	43%
All types	82	52%
Deaths	5	3%

* Of 131 patients (see Table III).

Deaths

During the eight-year period there were five known deaths more or less directly due to the ulcer disease. The causes of death were as follows. One patient died from bronchopneumonia after a successful partial gastrectomy for active duodenal and gastric ulcers at the age of 68. Subacute bowel obstruction four months after gastrectomy required another operation, from which the patient did not recover. A second fatality related to surgical treatment occurred in a man aged 53 operated on for both gastric and stomal ulcers

after a gastro-enterostomy established 15 years earlier for duodenal ulcer. Death took place following reoperation necessitated by bleeding shortly after partial gastrectomy. Another patient, aged 69, had well recovered after suture of a perforated duodenal ulcer, but died suddenly from massive bleeding due to chronic gastric ulcer. A fourth male patient with a 30-year history of peptic ulcer died two years after a successful partial gastrectomy. At necropsy, renal disease suggestive of Burnet's syndrome due to prolonged alkali medication was found. The fifth patient, a man of 60, died 24 hours after emergency admission for a bleeding gastric ulcer accompanied by a duodenal scar.

Operation

Of the 157 patients, 82 underwent one or more operations for peptic ulcer, including simple suture for perforation and gastro-enterostomy. Several patients were operated on twice. In 68 partial gastrectomy was performed. In at least eight cases this was done as an emergency measure for bleeding. Forty patients undergoing gastrectomy were over 50 years of age, and, among these, 24 were more than 60 years old.

Gastric Secretion.—The available data on 79 patients could not be compared, because gastric secretion had been assayed by different methods.

Discussion

A radiological diagnosis of combined gastric and duodenal ulcers was made in 2.7% of more than 5,000 ulcer patients in the eight-year period 1947–54. Inclusion of an estimated 25% of prepyloric ulcers would raise this figure to 3.5%. This is in fair agreement with the findings of Morrison and Feldman (1951), who in a special investigation observed clinical and radiological evidence of combined ulcers in 4.2% of 500 peptic ulcer patients. A considerably lower proportion was reported in most general x-ray and post-mortem series reviewed by Feldman (1953). Papers not mentioned by this author include those by Needham and McConachie (1950), Shapiro and Schiff (1952), and Stewart, Sanderson, and Wiles (1952) indicating that 1.8 to 4% of bleeding gastric and duodenal ulcers show combined lesions. Smith, Boles, and Jordan (1953) found a concomitant duodenal deformity in 19.7% of patients with benign gastric ulcers of all sites. Glenn and Harrison (1951) reported 12% and Rauch (1952) 3.2% of combined ulcers in their surgical patients. Johnson (1955) personally observed concomitant lesions in one of seven duodenal and one of four gastric ulcer patients. He suggested a general incidence of about 5%, including asymptomatic cases. There is reason to believe that the higher incidence generally reported by surgeons may be caused by the selection of patients.

No doubt the most reliable figures on combined ulcers are to be found in Watkinson's (1958) analysis of the necropsy findings in the late Professor Matthew Stewart's department at the University of Leeds. The incidence reported here is by far the highest with the exception of Sir David Wilkie's findings in 1926. This is explained by the painstaking care taken in the department in the detection of all ulcers and ulcer scars. Of a total of 1,848 chronic ulcers found between the years 1930 and 1949, 227 were combined gastric and duodenal ulcers, 174 in men and 53 in women. The calculation of the incidence is best based, however, on the findings in patients who died from causes other than ulcer disease. There were 1,232 of these patients, and 142 showed combined lesions. This gives an incidence as high as 11.5%. In the same group there were 799 duodenal and 284 gastric ulcers. The proportion of combined lesions among gastric ulcers (284+142) thus reaches 33.4%, and among duodenal ulcers (799+142) 15.1%. The sex ratio of combined ulcers in patients not dying from their ulcers was found to be 1.58 compared with 1.97 in the duodenal and 1.09 in the gastric ulcer groups, the male patients always predominating. The greatest incidence was found between the ages of 45 and 74 in males and above the age of 75 in females.

Admittedly a study based on hospital in- and out-patients will not allow any conclusions as regards the incidence of the disease in the general population. In hospital patients at least, the incidence is certainly high. As already pointed out by Johnson (1955), this is surprising, because many features of duodenal and gastric ulceration are to some extent mutually exclusive. The differences in gastric secretion between duodenal and gastric ulcer patients are well known. Differences in the motor activity of the stomach and duodenum are not as well documented as the secretory aspects. Most patients with uncomplicated duodenal ulcer are found on gastric biopsy to have a normal stomach mucosa. On the other hand, the incidence of gastritic changes in the ulcer-bearing stomach is high. Such features may appear compatible in cases where the gastric ulcer follows a healed duodenal ulcer after a long interval. However, almost two-thirds of the present cases showed active lesions of both stomach and duodenum. Moreover, in at least eight cases the reverse sequence occurred. On occasion the evolution was quite striking, the gastric and duodenal ulcer appearing to be independent of each other.

Thus in a man aged 40 with a short history of dyspepsia a large ulcer was found at the middle third of the lesser curve. He was successfully treated as an in-patient and four x-ray series did not reveal any duodenal crater or deformity. A few days after he left the hospital he was readmitted for a perforated duodenal ulcer, which was sutured. No active gastric lesion was found at the operation. The duodenal ulcer healed, but the gastric ulcer relapsed within three months. One year later the duodenal ulcer had also recurred, accompanied by two gastric ulcers. When partial gastrectomy was performed after another two months, the gastric ulcers had healed. At no time was there any evidence of delayed emptying. The pattern of gastric secretion did not change markedly when concomitant ulceration followed the gastric ulcer.

That even the pattern of gastric secretion may change is shown in observations reported by Watkinson (1951). In one 42-year-old male patient the healing of a gastric ulcer was followed within five months by the appearance of a duodenal ulcer, both confirmed at operation. In another man aged 33 a gastric ulcer appeared six months after a duodenal ulcer had healed. In both patients there was a definite change in gastric secretion from the gastric ulcer type to the pattern found in duodenal ulcer, and vice versa.

Gastric retention is one factor thought to be responsible for combined ulceration (Johnson, 1955). In less than 30% of the 131 patients analysed in this respect there were any possible signs of pyloric stenosis. Even in cases with delayed emptying it may be questioned whether gastric retention is the cause rather than the effect of chronic gastric ulceration. Retention is present in a similar proportion of gastric and duodenal ulcers. The importance of pyloric stenosis arising from duodenal ulcer may also be questioned on the basis of the sex ratio, which was nearer to the figure found in gastric ulcer. Again the age distribution is more or less the same as in gastric ulcer. It is thought that a factor common to both gastric and duodenal ulcer disease may be at work to cause combined ulcers. Unfortunately, there was insufficient material for a detailed analysis in respect of ulcer inheritance. In several instances, though, the family history was most striking, with proved cases of both gastric and duodenal ulcers in near relatives of the patients. The work of Doll and Kellock (1951), which includes cases with combined ulcers, is most suggestive of a common hereditary background. They observed that 6 out of 11 patients with combined ulcers had near relatives with the same disease.

It is reasonable to expect that combined ulcer disease runs a more severe course than either duodenal or gastric ulcer. Although no conclusive proof can be offered, the present study bears out this impression. The proportion of patients with perforations is not unexpected in view of the findings of Martin and Lewis (1949). On the other hand, the risk of bleeding is certainly high, particularly if

the period before the appearance of combined ulcers is taken as a basis for comparison. According to Balfour (1932) about 25% of peptic ulcer patients followed for an average of 10 years will show one or more episodes of bleeding. Emery and Monroe (1935) found an incidence of 26.7% in patients observed for an average of 3.5 years. Gastric ulcers are thought to bleed more often than duodenal ulcers (Ivy, Grossman, and Bachrach, 1950). However, Martin and Lewis (1949), in their follow-up of peptic ulcer patients for more than 10 years, observed bleeding in 26.8% of gastric and 35.2% of duodenal ulcers. In the present series the incidence of bleeding in 59 of 157 patients—that is, 37.6%—followed for a similar length of time, is somewhat higher. Moreover, three-fourths of the haemorrhages took place during the period which represented on the average one-eighth or less of the total ulcer history. It is therefore not surprising that conservative treatment was abandoned in favour of partial gastrectomy in 68 of these patients. In fact, the present findings would suggest that surgical treatment is indicated more often in combined ulcers than in single ulcer, whether gastric or duodenal.

Summary

The combination of chronic gastric and duodenal ulcers or ulcer scars was found in 157 hospital patients during a period of eight years. Cases suffering from prepyloric gastric ulcer were excluded owing to the uncertainty of location.

The observed incidence of combined ulceration was 2.7% based on x-ray investigation. With the inclusion of an estimated 25% of prepyloric ulcers the incidence would reach 3.5%. Thus about one in every seven hospital patients suffering from ulcers of the stomach and 1 in 30 patients having a duodenal ulcer or ulcer scar will be found to show combined ulcers.

With due awareness of the frequent occurrence of combined ulcers, the radiological examination represents a reliable means of their diagnosis.

Combined ulcers run a higher risk of bleeding, making surgical treatment more often advisable than in either gastric or duodenal ulcer occurring alone.

Pyloric stenosis does not appear to be a major factor in the pathogenesis of combined ulcers.

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RECTAL TEMPERATURE IN THE NEWBORN AFTER BIRTH ASPHYXIA

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In a previous investigation (Cross, Tizard, and Trythall, 1955) it was shown that if normal babies were given 15% oxygen to breathe there was a fall in oxygen consumption which was so large that the energy loss would be greater than the energy gain from any anaerobic metabolism which might be taking place. From this indirect calorimetry it was suggested that energy production fell under these experimental conditions, and this seemed to be confirmed when Brodie, Cross, and Lomer (1957) showed that infants breathing 15% oxygen heated the plethysmograph in which they were placed less than when they breathed air, though a consistent fall in rectal temperature was not found when the 15% oxygen was given for periods up to 50 minutes. Young kittens and puppies show a fall in temperature directly after oxygen consumption is reduced in anoxia (Moore, 1956a, 1956b).

In babies the absence of this immediate fall in body temperature along with their fall in oxygen consumption in an atmosphere of 15% oxygen might be connected with a relative thermal inertia compared with the smaller animals. Vascular reactions might also play a part, since skin pallor was noticed in the babies. The probability that factors such as these influenced the findings in short-term observations is supported by Sjöstedt and Rooth's (1957) report that in full-term babies the temperature is "a little lower than normal if kept in 15% oxygen for a week."

Although suggestive, these results might not apply to the infant showing apnoea or cyanosis at or after birth. While cyanosis is evidence of anoxia, it is unlikely that the natural condition is ever a pure "anoxic anoxia." CO₂ retention will probably, although not necessarily, be present. An investigation which would in some measure test whether naturally occurring anoxia causes a fall in heat production of the newborn infant was undertaken. We wished to make the investigation as simple as possible so that it could be fitted into the activities of a busy maternity unit, and decided to divide the babies into those that did and those that did not show asphyxia at birth, and to follow their rectal temperatures. As it became apparent that maternal sedation was a complication the scope of our investigation had to be widened slightly, and finally four groups of infants were studied, but the general scheme was not made more complex in any other way.

Material and Methods

Babies were delivered at a labour-room temperature of 78–80° F. (25.6–26.7° C.), wrapped in a towel, and, after they had been held by their mothers, were transferred to the nursery after a period that varied from 10 to 30 minutes. Resuscitative measures used after birth were oxygen, which was given for a few minutes at the discretion of the obstetrician or midwife in charge, and 1 mg. of N-allyl