Then there is the "stomach" cough: on the authority of Hess there are physiological experiments to prove its possibility, and coughs have been produced artificially by appropriate stimulation to the oesophagus or stomach. I think the most usual explanation is that the attack of gastritis, or whatever it is which is the stomacic element of the cough, leads to a general catarrhal state of the mouth and pharynx, also seen in the coating of the tongue, and this causes the cough. I never diagnose the condition unless I see the coating of the tongue extended right over the root of this organ and a red, glazed condition of the posterior pharyngeal wall. On the whole, it is a diagnosis to make with great caution, as it is a difficult one to live down with parents, and is apt to be sneered at.

Lastly, there is the question of cough as a habit spasm. Following an attack of whooping-cough a child is liable to go on coughing and whooping for months, for no more obvious reason than another child will go on twitching its mouth long after a patch of impetigo has vanished from the lip. To diagnose a cough as a habit spasm, however, necessitates a very careful examination of all other possible causes, and, as with any diagnosis resting on exclusion, it has inherent weaknesses. In this category also is the so-called barking cough of puberty, a cough which is labelled as hysterical in some of the textbooks. Here again diagnosis is largely by exclusion, and to attribute a cough to a purely psychical origin requires a very close examination of the whole environment of the child. As in *Alice*, it may be that

"He only does it to annoy
Because he knows it teases."*

TREATMENT

If the origin of the cough has been accurately localized the question of treatment becomes relatively simple. Obviously, since the prime object of a cough is to remove a foreign body from somewhere in the respiratory tract, the cough should be encouraged when there is the possibility of achieving this, and discouraged otherwise, as in most of the instances I have outlined. In other words, measures should be taken to stop almost all coughs originating above the bifurcation of the trachea: this also holds true for many coughs below this point. It follows that expectorants have a very limited use, and in my opinion too much attention has been directed in the past, as far as children's medicine is concerned, to the question of "depressant" and "stimulating" varieties. For most coughs in children a sedative linctus is best, such as tinct. camph. co. mx, oxymel scillae mx, glycerinum mx, spir. chlorof. mij, aq. ad. 5i, for a child of 2 years, as required.

Obviously, the first step in the treatment of cough is to eliminate or treat the cause as far as possible. As this will mean measures varying from radiation of an enlarged thymus to evacuation of an empyema it is not possible to discuss each type of cough in detail in this respect. I would like to emphasize, as I have already done elsewhere,⁵ the importance of irritant gases as a cause of inflammation of the respiratory passages. In London the acidity of the atmosphere is sufficiently powerful to make serious inroads upon the stonework of the Houses of Parliament, and it is not surprising that many infants cough from their earliest days. Still⁶ has also called attention to the possible irritation produced by a smoking fire in the nursery. Adequate treatment of coughs in childhood requires, in certain instances, a study of home surroundings.

The steam-tent in the treatment of respiratory disorders of childhood has a very limited field of usefulness and

many drawbacks. In some animal experiments I carried out last year* it was found that hot moist air quickly caused fatal oedema of the lungs if the temperature was allowed to approach that of the body. The most beneficial effect in the relief of artificially produced spasmodic conditions of the bronchi was obtained when the air was merely kept fully saturated with water vapour at room temperature. This is best achieved by a kettle on the hearth, well away from the child's bed; no steam should be visible in the room at all. Used in this way moisture is of assistance in the relief of spasm, whether this be in the larynx or in the bronchi. It should be remembered in this respect that bronchitis with spasm is frequently a manifestation of asthma in the young child, and remedies based upon modern work should be employed—namely, correction of constitutional errors in digestion, removal of all possible sources of animal emanations and dust, and drugs to stop the acute attack.

Lastly, the treatment of bronchitis requires a brief word. Many of the drugs used, such as ammonium carbonate, squill, and ipecacuanha, have a direct irritant effect upon the alimentary tract if used in anything but small doses, and in young children the use of these drugs may lead to a troublesome and often dangerous gastro-enteritis. When the secretion is thick, during the early stages, alkalis are most valuable, and a good combination is vin. ipecac., mij, pot. cit. gr. v, tinct. camph. co. mv, glycerinum mx, aq. ad. 5j, three times a day, for a child of 1 year. Later on a little ammonium carbonate (gr. 1/2) may replace the potassium citrate, and if much wheeziness is present a couple of minims of tincture of stramonium should be added. The use of atropine to "dry up the secretion" in such cases is not recommended; it has, indeed, been condemned by Chevalier Jackson. It is doubtful whether much can be done to aid nature in the mechanism of expectoration, but harm can certainly be done by ill-considered interference.

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AETIOLOGY AND SYMPTOMS OF MITRAL STENOSIS

A REVIEW OF THREE HUNDRED CASES

BY

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It has been stated by Coombs¹ that every case of mitral stenosis is a case of advanced cardiac rheumatism, and this writer has produced clinical and pathological evidence in support of the statement. It does not, however, appear clear in his monograph in what percentage of cases of mitral stenosis a rheumatic history was obtainable. Price² states that mitral stenosis is *usually* due to rheumatism in one or other of its protean manifestations.

The following analysis of 300 cases of mitral stenosis was undertaken on clinical lines in order to assess the importance of the history of rheumatic fever and allied conditions in the causation and aggravation of the disease. During the investigation a number of other factors have been noted. The age and sex incidence of mitral stenosis are discussed briefly, and the symptomatology is also considered.

* Before accepting this modern psychological dogma too hastily the chorus following these immortal lines should be recalled—"Wow, wow, wow!"

* Working with a part-time personal grant from the Medical Research Council.

AGE AND SEX INCIDENCE

Of the 300 cases of mitral stenosis under review 79 were males and 221 females. I have arranged these in decennial age groups, and the result is shown in Table I and Graph 1. In four instances a record of the age was not available.

TABLE I

Age Periods:	5-15	15-25	25-35	35-45	45-55	55+
Males* ...	11	26	21	13	6	1
Females† ...	7	64	73	51	22	1
Total cases ...	18	90	94	64	28	2

* Age not given in one case. † Age not given in three cases.

In Table II and Graph 2 the number of cases at each age period has been expressed as a percentage of the total number for each sex, and the result is of considerable significance. It will be noticed that the maximum incidence among males is a decade before that for females.

GRAPH 1.—Age incidence of all cases.

GRAPH 2.—Percentage of cases at the different age periods.

GRAPH 3.—Initial attacks of rheumatic fever and onset of fibrillation.

It seems feasible that indulgence in athletics and manual labour accounts for the earlier appearance of the disease amongst men, while the strain of pregnancy and parturition in women between 25 and 35 years of age causes their maximum incidence to be definitely later. It has not been possible to decide whether the actual stenosis occurs sooner in males, or whether it is that *symptoms* due to inadequate compensation come on at an earlier date.

TABLE II

Age Periods:	5-15	15-25	25-35	35-45	45-55	55+
Males percentage ...	13.9	33.0	26.6	17.8	7.7	1.3
Females percentage ...	3.2	24.5	33.0	22.5	10.0	0.5

Nearly half of the males have developed mitral stenosis before 25 years of age, while much less than one-third of the females show evidence of the lesion at that age.

ASSOCIATED VALVULAR LESIONS

Fifty patients in this series had aortic regurgitation as well as mitral stenosis. Of these twelve were males and thirty-eight females—that is, 15 per cent. and 17 per cent. respectively of the total in each group. Aortic stenosis occurred eight times in association with aortic incompetence, and on three occasions apparently independently of it. No definite pulmonary or tricuspid lesions were recorded, and it is interesting to note that the Graham-Steel murmur was not found present in a single instance. It is likely that mitral regurgitation occurs to a variable

extent in the majority of cases of mitral stenosis, but it was somewhat infrequently that the diagnosis was put on record, so that the figure of 10 per cent. obtained in the present series is probably too low.

RHEUMATIC HISTORY

(a) *Incidence.*—Rheumatic fever, growing pains, and allied conditions are prominent features in the history of the great majority of the cases. The figures obtained are as follows: rheumatic fever, 150; chorea, twenty-seven; rheumatic pains and swellings (mostly apyrexial), twenty-three; and growing pains, fourteen.

This makes a total of 214—approximately 75 per cent. of the present series. Of the 271 cases of *known aetiology* it is seen that a definite rheumatic history was elicited in 80 per cent.: mitral stenosis with rheumatic history, 214; mitral stenosis of non-rheumatic history, fifty-seven; and no aetiological factor found, twenty-nine.

(b) *Multiple Attacks.*—A large number of cases gave a clear history of more than one attack of rheumatic fever—107 patients had one attack, thirty had it twice,

nine had three attacks, five had four attacks, and one stated that five attacks occurred. It was noticed that the first attack not infrequently failed to leave any cardiac symptoms, and that the patient dated his trouble to the second or third attack.

(c) *Other Details.*—That rheumatic fever is a disease of childhood and adolescence is strikingly borne out by an analysis of the age of the onset of the first attacks, where the data could be ascertained (see Table III and Graph 3).

TABLE III

Age Period:	1-5	5-10	10-15	15-20	20-25	25-30	30-35	35-40	40-45	45-50
No. of cases	4	22	42	37	13	9	4	2	0	1

In Graph 3 the ages of onset of rheumatic fever and auricular fibrillation have been plotted, and will be considered later on. It is clearly seen that more than half of the cases of rheumatic fever occur before the age of 16, 86 per cent. before the age of 21, and approximately one-third of the patients are infected between the ages of 10 and 15. It was noticed that seventy-five cases of rheumatic origin developed one or more attacks of febrile illness (including chorea, but excluding recurrent attacks of rheumatic fever), which definitely appeared to aggravate the condition of the heart. The time of confinement to bed for the rheumatic fever cases during and following the acute attack varied from one week to twelve months. In eighty-seven cases, where the exact duration of stay in bed was known, the average was from five to eight weeks.

NON-RHEUMATIC HISTORY

The cases of non-rheumatic history were fifty-seven in number, and although a considerable proportion of these could have been included in the rheumatic division, it was considered expedient to assign them to this group. This was especially true of a number of cases of influenza, tonsillitis, and scarlatina.

In this group forty-one persons dated their symptoms back to definite infection as follows:

Influenza	19	Tonsillitis	6
Scarlet fever	4	Pneumonia	4
Typhoid	2	Measles	2
Puerperal fever	1	Quinsy	1
Diphtheria	1	Acute nephritis	1
"Gassed" in the war ...		1	

The remaining patients gave a history of a number of possible aetiological factors, but could not date their symptoms back to any particular illness. In three cases the only finding was a strongly positive Wassermann reaction.

No evidence whatsoever was found of any causative factor in the twenty-nine cases in this group. Many of them exhibited no symptoms, the lesion being found at routine examination. It is possible that a number of these cases were of rheumatic origin—the patient having forgotten or ignored growing pains and other mild manifestations of the infection.

COMPLICATIONS

1. *Embolism*.—Six cases of embolism—all cerebral—occurred in the present series. Two were straightforward cases of mitral stenosis, and the other four had auricular fibrillation. The only male in the series had had the fibrillation treated with quinidine sulphate, and embolism occurred a short time afterwards, with fatal results (Table IV).

TABLE IV

Case	Sex	Age	Lesion	Etiology
1	F	38	Mitral stenosis	Rheumatic fever at 10 and 33
2	F	35	Mitral stenosis. Auricular fibrillation	Rheumatic fever at 10
3	F	30	Mitral stenosis	Rheumatic fever at 11 and 17
4	F	45	Mitral stenosis. Auricular fibrillation	Rheumatic fever at 17
5	M	38	ditto	?
6	F	44	ditto	Rheumatic fever at 18

2. *Haemoptysis*.—History of haemoptysis was given in twenty-one cases (7 per cent. of the total). Of these eight were males (10 per cent.) and thirteen were females (6 per cent.). It thus appears that the incidence of haemoptysis is higher in males, but it is not wise to stress this point in view of the small number of cases under consideration. Auricular fibrillation was present in six cases, four had aortic incompetence, and three were associated with pregnancy. It is possible that haemoptysis may tend to be more prevalent at two distinct age periods: (a) between the ages of 20 and 30; (b) at about 45 years of age (Table V).

TABLE V

Age Periods:	15-20	20-25	25-30	30-35	35-40	40-45
No. of cases	3	5	4	3	3	3
Percentage of cases ...	6.5	9.1	8.3	6.0	7.7	10.0

Signs of gross cardiac failure were absent in all these cases. Tuberculosis was absent throughout the series; clinical evidence of phthisis was very suggestive in one case, but the radiological report was inconclusive.

3. *Pregnancy*.—Of the forty-eight cases where note was made concerning the relation of symptoms to pregnancy:

(a) Six did not complain of any symptoms at all, the lesion being found by routine examination at ante-natal clinics and the patients subsequently referred to the heart department.

(b) Eleven stated that the manifestations of cardiac disability occurred for the first time during pregnancy—usually the first or second pregnancy. The symptoms often cleared up completely after parturition, only to return during the following pregnancy.

(c) Twenty-eight complained of symptoms of varying intensity prior to pregnancy. Half of them were seriously incapacitated, and the other half showed marked aggravation of symptoms, but not to the same extent as the first group.

A number of patients suffering from mitral stenosis weathered anything up to fourteen pregnancies.

4. *Auricular Fibrillation*.—The number of cases that exhibited auricular fibrillation was 87. Of these twenty-six were males (33 per cent.) and sixty-one females (27.6 per cent.). In Table VI and Graph 4 the percentage of cases at each age period of mitral stenosis which

TABLE VI

Age Periods:	5-15	15-25	25-35	35-45	45-55
No. of cases of auricular fibrillation	1	13	28	26	15
Percentage of mitral stenosis who get auricular fibrillation	5.5	14.4	30.0	40.0	54.0

Age not given in four cases.

develop auricular fibrillation is shown, as well as the number of cases of auricular fibrillation at the same age period. It is clear from the graph that with increasing age there is rapid increase in the liability of cases of mitral stenosis to fibrillation.

SYMPTOMATOLOGY

In the vast majority of cases the symptoms were very similar in type, varying only in degree of intensity. Breathlessness on exertion was the commonest complaint, while vague precordial pain—not anginal in type—was frequently mentioned.

The type of onset of symptoms may be classified into seven groups:

1. No symptoms at all may be complained of, the lesion being found at routine examination for life insurance or at ante-natal clinics.

2. There is usually a latent period—freedom from symptoms after the rheumatic infection—which may last any time from a few months to many years. Onset of symptoms is gradual. After a period of adequate compensation, gradual decompensation sets in, in the absence of fresh infection.

3. Some cases never seem to recover properly from the attack of rheumatic fever (or pneumonia, etc., as the case may be). There appears to be more myocardial disease than is usual, and compensation is inadequate from the very first.

4. Symptoms are often absent until the onset of auricular fibrillation. The actual onset of the fibrillation may be sometimes dramatic, and the patient is able to associate it with some particular event—for example, physical strain.

5. Cardiac symptoms may be attributed to some definite occurrence, quite apart from the onset of fibrillation—for example, pregnancy, strain, infection, and domestic worry. In this group might be included a few patients who build up their symptomatology after being told that they have "heart disease."

GRAPH 4.—Incidence of auricular fibrillation (A.F.).

6. Occasionally a patient presents himself with frank D.A.H. type of symptoms, and on examination mitral stenosis is found.

7. Symptoms may vary because of some external factor—physical or physiological. A patient was markedly affected at the fairly high altitude at Nairobi, but was very much improved on returning to sea-level.

SUMMARY

An analysis of 300 cases of mitral stenosis, without gross cardiac failure, has been attempted. It is realized that the comparatively small number of cases may give rise to misleading conclusions, but it is felt that the following are justified.

1. The age and sex incidence has been emphasized. There is evidence that the disease is commoner in women than men in the proportion of 3:1, and that it occurs at a definitely earlier age in males.

2. The importance of rheumatism as an aetiological factor has been shown. At the most *conservative estimate* it was the aetiological factor in 80 per cent. of the cases. It is probable that 90 per cent. is nearer the correct figure in the present series. No other aetiological factor was found present with any degree of constancy.

3. Some of the complications of mitral stenosis and their relative frequency at an out-patient clinic are mentioned.

4. The type of symptoms and their mode of onset, as found in the survey of the present series, are considered. The majority of cases over 45 years of age develop auricular fibrillation.

I wish to acknowledge my gratitude to Professor John Hay and Dr. H. Wallace Jones for their advice and guidance, and for access to the records of their cases at the heart department of the Liverpool Royal Infirmary.

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ACUTE APPENDICITIS: SOME MISTAKEN DIAGNOSES

BY

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It is probable that in no other disease is a wrong diagnosis made more often than in acute appendicitis. Anyone used to operating upon the acutely inflamed appendix to any great extent will probably at some time (or times) have found, on opening the abdomen, one of the following: (1) a perforated peptic ulcer; (2) an inflamed Meckel's diverticulum; (3) a ruptured ectopic pregnancy; (4) a torsion or inflammation of the uterine adnexa; (5) a torsion of a pelvic tumour, cyst, or fibroid; (6) an inflamed gall-bladder; (7) an ileo-caecal adenitis; (8) a hyperplastic tuberculosis of the caecum or even a new growth; (9) a renal or ureteric calculus, or the so-called Dietl's crisis. The case is not unknown where the anaesthetist gets blamed for a severe post-anaesthetic pneumonia, but often before operation the temperature was higher than is usual with an abdominal complaint, and yet a causative lesion of the appendix was woefully lacking. Surely many of these, if one will but admit it, are mistaken diagnoses? If, however, the chest has been carefully examined and found clear there is no disgrace in operating, for the alternative of a ruptured appendix is serious; the only disgrace is when the chest is not examined.

The following notes of four personal cases seem worthy of mentioning on account of their rarity. Each was carefully examined, a firm diagnosis of acute appendicitis was made, and in each the diagnosis was badly wrong.

CASE I

Mary B., aged 5. There was a two days' history of upper abdominal pain, which after eight hours had settled in the right iliac fossa. On admission to the Children's Hospital she had an area of maximum tenderness over McBurney's point. The right lower quadrant of the abdomen was rigid. There was a tender mass felt per rectum, the temperature was 99.5° F., and the pulse 118. She had a coated tongue and had vomited four times. A confident diagnosis of acute obstructed appendicitis was made. On opening the abdomen the appendix was seen to be normal, and there were no glands. The caecum was filled by a large cyst the size of an orange, which practically filled the former, and certainly was tending to obstruct the ileo-caecal valve. On being opened the cyst was seen to be lined with pink columnar epithelium—it was, in fact, bowel mucosa without the folds—and to contain glairy mucus. It was clearly a case of a submucous cyst, and probably was one of the enterogenous cysts derived from buds of epithelium which, separated from the primitive foetal gut, became vacuolated and cyst-like. These cysts may lie in the mesentery near its root or near the gut. If in the gut wall they may be subserous, intramuscular, or submucous (see Fig. 1). The case in question proved to be

FIG. 1.—To illustrate submucous enterogenous cyst.

one of the latter—that is, a congenital submucous enterogenous cyst. Though not unique, it is one of the rare conditions found in this region.

CASE II

The patient, a little girl 12 years of age, was admitted in the late afternoon with a history of having been taken ill two and a half days previously with generalized abdominal pain, followed some hours later by vomiting, and, for the last twenty-four hours, pain in the right iliac fossa. On examination her pulse rate was 120 and temperature 97°. The tongue was coated and the breath foul. The abdomen was tender all over—especially over the appendix—and resistant over McBurney's point. The chest and heart were normal. A rectal examination revealed a tender mass in the caecal region. She thought she felt more comfortable since the morning, and that this change had come on fairly rapidly. Her appearance, however, did not support this. The only item of interest was that she thought her left foot and right wrist had swollen during the last day or two, but there was nothing gross to be seen in them. Her urine was normal. The diagnosis of acute gangrenous appendicitis was made, and I was rather inclined to think that it had perforated, considering the fact that she had become very much easier, that her pulse had risen, and that the temperature had fallen. The