it was found at operation that the patient's colon had not been properly cleared out, and there was a mass of faeces proximal to a growth in the splenic flexure. It would have been much wiser to delay resection in this case until after drainage and irrigation of the colon.

It is noticeable that there was only one death in the cases of palliative short circuit, this being due to broncho-pneumonia. This favourable mortality rate in comparison with that of resection and anastomosis suggests that interference with efficient blood supply of the line of anastomosis by resection of the mesentery is the main factor in anastomotic leak and a fatal issue, and one that must be guarded against most carefully. The risks of imperfect blood supply in end-to-end anastomosis are certainly greater than in lateral. I generally employ the lateral method for this reason, except when it is not possible, as is most often the case with the pelvic colon. It is certain that the mortality for resections in the distal half of the colon, and especially the pelvic colon, have a higher mortality than those of the proximal colon.

I have not attempted in this paper to deal with end-results. The cases in the first series referred to have not yet been followed up. The real test of good statistics is end-results—that is, the percentage of five-year cures of all cases operated on, exclusive of those found to have visceral metastasis at operation. It is not enough to show a good percentage of resection recoveries; these cases should also form a high percentage of the total operation. For example, one surgeon might explore ten cases of carcinoma of the colon, all free from metastasis, and resect three of them (all early cases) with no death, and these three might survive five years, while the remainder with short circuit or colostomy survive only a few months or years. Another surgeon meeting the same series of cases might be bold enough to resect seven of the ten, with one death, and four of the surviving six might live five years. The statistics of the former surgeon would be: operation mortality, *nil*, percentage of five-year cures, 30; and the statistics of the latter: operation mortality, 14 per cent., percentage of five-year cures, 40. When it is recognized what can be accomplished in colon surgery the surgeon who errs on the side of boldness will save more cases in the long run.

1921-1925			1927-1931			Total		
Cases	Died	Percentage Mortality	Cases	Operation Mortality	Percentage Mortality	Cases	Operation Mortality	Percentage Mortality
41	9	21.9	34	7	20.5	75	16	21.0
Obstructed								
11 ^a	4 ^b	36.3	5	3 ^c	60.0	16	7	43.7
Resected								
22	5 ^d	22.7	23	3 ^e	13	46	8	17.7
Anastomosis only								
6	0	<i>nil</i>	3	1	33.3	9	1	11.0
Colostomy only (non-obstructed)								
2	0	<i>nil</i>	3	0 ^e	<i>nil</i>	5	0	<i>nil</i>

^a Three cases resected later and included in resections; remainder colon drainage or abdominal drainage.

^b Two perforated at time of operation.

^c One patient died under anaesthetic before operation.

^d Two shock; 2 peritonitis; 1 cardiac failure.

^e One peritonitis; 1 peritonitis and ileus; 1 pulmonary embolism.

CONCLUSION

I have not attempted in this paper to do more than give expression to my own experience. I am conscious of the fact that few surgeons meet with sufficient cases to get that volume of experience which is really necessary to become expert. Carcinoma of the colon is a far less common disease than carcinoma of the rectum, and I have excised something like five times as many rectums as I

have resected colon growths. As the volume of cases dealt with increases so the full range of possible pitfalls and complications becomes unfolded, and the surgeon learns how to avoid these, and to improve both his technique and his statistics. I know that this is true in my case for excision of the rectum, and I feel confident that with continued experience in the treatment of carcinoma of the colon I shall be able to improve both my technique and my results, more especially if, by stimulating interest in early diagnosis, cases come for treatment at an early stage. In this connexion it is worth noting that my operative mortality of resections from 1921 to 1925 was 23 per cent., and for 1926-31 only 13 per cent., and I may add that the last thirteen cases in this series survived operation.

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NOTES ON 1,500 CASES OF ASTHMA

BY

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LARYNGOLOGIST TO GLASGOW CORPORATION HOSPITALS AND TO
LANARKSHIRE COUNTY HOSPITALS

In this survey only such facts as may throw light on some of the more important points raised in recent controversy will be touched upon. It is particularly essential that in asthma figures should be closely correlated to clinical facts, as neglect of this has led to misleading statistics.

DIAGNOSIS

Chevalier Jackson¹ wisely says that all is not asthma that wheezes, and that "asthmatoïd" wheeze is a frequent symptom of inspired foreign body with missed diagnosis; but when later he suggests that asthmatics should be regularly bronchoscoped, one may reply that there are simpler and more acceptable means of settling the diagnosis and, usually, of giving relief. Adrenaline will not ease "asthmatoïd" wheeze, but usually gives prompt relief in genuine asthma. Eosinophils crowd the sputum in asthma, are scanty in the "asthmatoïd" and mostly replaced by polynuclears; they are usually above 4 per cent. in asthmatic blood, below it, with probably a polynucleosis, in the "asthmatoïd." Wheeze plus eosinophilia is asthma, wheeze without it demands scrutiny of the diagnosis; wheeze plus tough viscid sputum crowded with eosinophils is asthma; wheeze plus purulent sputum with few eosinophils but with many polynuclears is *not* simple asthma. It is too often forgotten that asthmatic sputum is not purulent. Even the clinical history, as these records abundantly show, will prove the diagnosis. Dyspnoea coming on typically between 2 and 4 a.m. or on waking, or after food, is not asthmatoïd but asthmatic. Attention to these points has in this series excluded other dyspnoeic conditions—renal, cardiac, and arteriosclerotic.

EOSINOPHILIA

Increase of eosinophils in the blood beyond 4 per cent. (on an average 8.4 per cent. in 68.5 per cent. of the cases) has been the rule. Of the remaining 31.5 per cent., three-fourths had no asthma at the time of count, so that correlating count with wheeze we find eosinophilia in 80 per cent. This is much nearer the truth than Gram's misleading figure of 44 per cent., quoted recently in this *Journal*.² There is no better guide to diagnosis than the eosinophil count, and no better indication of progress under treatment than its fall—a patient free from wheeze but with a high count is a candidate for a fresh attack. The percentage *may vary quickly*. It mostly rises before and falls after the attack; in menstrual asthma it rises before and falls after the flow. It rises within an hour after ingestion of

urea, and may even be doubled, falls within ten minutes after injection of adrenaline, and may be halved, rises with a hot bath, but may fall, owing to adrenal stimulation, if followed by a cold douche, when the basophils are apt to increase. These are general statements with exceptions, and do not apply where the count is approximately normal.

Bezançon and Bernard, however, in their valuable monograph³ on the eosinophil cell, are not correct in saying that eosinophilia occurs in every case. Eosinopenia may occur in hypopietic asthmatics, as will be shown below. Even in such cases and others with a low count there may be excess eosin staining of the granules in the polynuclears. The highest count in the present series was 68 per cent., and all the polynuclears showed this excess stain. This high count, however, was probably related, not to the patient's asthma, which was slight and improved steadily, but to a slowly spreading eruption of haemangiomatous papules. (Examinations for intestinal parasites always negative.) When asthma has been associated with chronic skin affections, the eosinophil count has been high. Bezançon and Bernard suggest that the eosinophil is more akin to the mononuclear than to the polynuclear cell, but the excess eosin staining of the latter already mentioned is against this view. On the other hand, the high counts in cases with chronic skin complications seem to favour their view that the eosinophil may have an origin from tissue as well as from the bone marrow. They also bring much evidence to show that "eosinophilia is a response to proteins, auto-genous but ill metabolized, or foreign (heterogeneous). In short, it is a sign of toxæmia."

BLOOD PRESSURE

Normal pressure characterizes most cases. I have shown⁴ this holds for chronic cases, and the fact that asthma begins in half the cases before the age of 15 also shows it. But there are two other types of asthmatics—hypo- and hyper-pietic. Both wheeze on exertion, and for the same reason—namely, that they have a narrow threshold on which to meet strain and there tends to be congestion in the pulmonary circuit. But wheeze that comes only on exertion is not asthma, and one may well, especially in the case of the hyperpietic, hesitate about diagnosis. Where wheeze comes also at typical times—2 to 4 a.m., on waking—and where there is, in addition, eosinophilia, one is justified in diagnosing asthma; when it is absent the diagnosis should be rejected. Moreover, as both asthma and hyperpietism may be of toxic nature it is not remarkable that the hyperpietic should develop asthmatic symptoms. This type often responds astonishingly to treatment found useful for asthma—regulation of diet, weekly fast and blue pill, dimol, and iodide and belladonna. Hyperpietics formed only 7.5 per cent. of adult asthmatics in this series. The hypopietics are also a small class—10.7 per cent.—but often more refractory to treatment. They are apt to be languid in mind and body, finicky, and not co-operative. Not only do the adrenals lag, but one wonders whether the pituitary is not at fault, some of the patients being tall and lanky. The usual detoxicating treatment would be harmful to them. Blood pressure of 95/80 is not uncommon—in one man of 5 ft. 11 in. it was 78/65, with eosinophils 2 per cent. The eosinophil percentage in this type is low, on an average 4 per cent.; in one it was 0.5 per cent., but there was excess eosin staining of the polynuclears. The highest tally was 11 per cent. in a man of 6 ft. with systolic pressure 100.

In so-called cardiac asthma the dyspnoea is not of asthmatic type and there is no eosinophilia; such sputum as was present in a recent case was crammed with polynuclears and not eosinophils. But there is stasis in the pulmonary circuit, and there must be an element of this in both the hypopietic and hyperpietic types of asthma

as shown by the wheeze on strain. This must mean congestion, and Chevalier Jackson¹ has observed congestion, not spasm, in the bronchi of asthmatics he has bronchoscoped. Such congestion does not bar bronchiolar spasm, but might rather tend to it. The bronchioli cannot be seen through a bronchoscope, and spasm would hardly manifest in the larger bronchi because of their cartilaginous walls.

TOXICOSIS

That a toxic factor bulks large in asthma I have tried for thirty years to show on the ground of the clinical history and of therapeutic results not equalled, so far as I know, by any other method of treatment. The truth of the theory is now more generally admitted. Bezançon and Bernard hold that eosinophilia means toxæmia. There is further proof in the frequent inversion of the leucocytic formula, the mononuclears being in excess and becoming a minority again after detoxication—for example, a woman, aged 54, with a history of twenty years' asthma, had, in percentages, eosinophils 7, polynuclears 37, small lymphocytes 33, large lymphocytes 23; after a month's treatment, with improvement, the respective figures were 6, 65, 23, 6. Further, in agranulocytosis, which is admittedly of toxic origin, this inversion is carried to an extreme, the white cells being almost wholly mononuclear. In asthma the toxæmia is milder and more chronic, the eosinophils being the response; if they are present at all in agranulocytosis the outlook is more hopeful. Again, if there is toxicosis in asthma, increase of the toxic load by addition of a waste product like urea should show response. It does this, not only raising the eosinophil percentage, but in one case it sent the patient into intractable status asthmaticus, the eosinophils rising from 11 to 22 per cent. Other evidence which I have already given^{4, 5} need not be repeated here.

ACIDOSIS OR ALKALOSIS?

This question is subsidiary to that of toxicosis. Tiefensee,⁶ on clinical and chemical grounds, McDowall,⁷ on the basis of experiments with dilute acids on the bronchi, and others, regard asthma as an alkalosis. If estimation of alkali reserves by the van Slyke process, with or without paraffin, is to be trusted, there has in the series never been the slightest suggestion of this condition—to the contrary, in fact. The normal is stated as ranging between 53 and 73 c.cm. CO₂ bound as bicarbonate in 100 c.cm. plasma, figures below 53 indicating a tendency to acidosis, above 73 to alkalosis. The following two sets of figures can be contrasted.

50 and under, 25 cases wheezing, 2 not wheezing.

50 to 59, 1 case wheezing, 23 not wheezing.

The average among 12 was 46 during attack, 59 when free.

Moreover, with detoxication the alkali reserve rises, gastric subacidity rises, eosinophils fall, wheeze disappears.

Case	Date	Eosinophils Per cent.	Alkali Reserve	Wheeze
Mrs. K. ...	31/12/30 5/1/31	8 5	50 67	Severe Gone
McL. ...	8/8/31 31/8/31	14 4	47 51	Present Gone

If asthma recurs the reserve falls and eosinophils rise. This swing of the eosinophils in one direction, and of the alkali reserve in the other, as wheeze comes or goes, is one of the most interesting features of asthma.

ADRENALS

As Cramer⁸ and Magee⁹ have shown, toxic products absorbed from the intestine or elsewhere first stimulate then exhaust the adrenals, a fact that goes a long way to support the toxic theory of asthma. This is corroborated

by the value of adrenaline in allaying spasm. Necropsy also confirms this. In a woman of 41, asthmatic for nine years, and an adrenaline addict, who had injected 7 oz. in a week and 1 oz. in one night, necropsy showed that the adrenal medulla was almost wholly destroyed. Dr. J. A. M. Cameron,¹⁰ who did the necropsy for me, reports two similar cases, one with asthma for ten years, one for twenty. Also there was darkening of the skin in at least two cases, as is common in chronic asthmatics, suggesting affection of the cortex. In one of the three necropsies the cortex was oedematous, in one it contained collections of leucocytes, in the third the whole gland was extremely small. Further, there are cases which suggest that adrenal failure may be an early sign of senile decay. This seemed to be the case in two sisters with intractable asthma, and Hurst has mentioned similar cases.

ALLERGY

Allergic manifestations other than asthma, mainly of the skin, were found in 55 per cent. In most cases direct attention was not paid to this, as the toxic soil on which allergy and asthma flourish was regarded as of more importance than the allergens, a point which is also raised by Bezançon and Bernard.³ For example, a man of 68 with asthma for six years and dermatitis herpetiformis for two years, who had had a year's treatment from a dermatologist without benefit, was sent to me for his asthma. Within three months his dermatitis had vanished and his asthma improved without attention to his skin. Sometimes avoidance of the allergen is the shorter road, and the patient will then always take it. Asthma that comes before sleep but soon after going to bed usually indicates sensitiveness to feathers. A man who got little benefit from me, except for six weeks after resection of his septum, found his asthma disappear during two visits to England, but recur immediately on his return home. His skin flared to tests with egg and with parrot feathers. He gave away his parrot and lost his asthma, yet takes eggs with impunity. Skin tests, however, I rarely need. Three cases improved promptly on removal from mould-laden houses.

HEREDITY

Family history of asthma or its allies was found in only 26 per cent. This is about half the figure found by Comby¹¹ in Paris, Rackemann¹² in Boston, and Bray¹³ in London. Comby wondered why asthma was more common in the city than in the country, among the rich than among the poor. As he emphasizes the importance of heredity as do allergists generally, it is like asking why heredity is more common in the city and with the rich than in the country and with the poor! Others have raised a similar question, but allergists have never answered it. The answer seems simple. My smaller figure is of patients drawn, not from large cities like London and Paris, but from smaller towns, where they are mostly within two minutes' walk of green fields. Asthma is largely bred by our indoor life and the sophistications that go with our civilization. Half of Bray's young patients were single children—proof itself of sophistication—and single children are usually pampered. There are no greater liars than statistics that bolster up the heredity of asthma. It is no more hereditary than gout. Few would now claim that gout, which is disappearing with altered habits, is hereditary—asthma is increasing for like reason. Only in the sense indicated by Professor McBride,¹⁴ in his paper on "Habit, the driving factor in evolution," is asthma hereditary, and this implies the heritability of acquired characters, just as Sir William Roberts¹⁵ indicated for gout. The heredity of asthma is only rarely Mendelian. No matter what the apparent history of heredity may be, most asthmatic children and many adults can be

permanently cured, for a toxicosis is bred, not inherited, but a *tendency* to it may be increased by bad habit prolonged in one direction through several generations.

THE NOSE

In adults the nasal condition suggested attention in 58 per cent., but frequently they did not get it, because the toxic state prevalent in asthmatics affects the mucosa of nose and bronchi as well as skin. Detoxication helps all; it has an effect on the nose by reducing mucosal "blobbiness" and irritability. It is too little recognized that brisk exercise constricts turbinates as does adrenaline. General measures are therefore usually tried before direct nasal treatment unless there are polypi or sinusitis, which should be treated at once. In affections of the nose in children the importance of vitamins must be emphasized, for it is certain that they help the mucosae and probable that they help the endocrines.

Sinusitis occurred in 10.5 per cent. In treating this, as in the case of all nasal operations, "ectomizing" surgery should be avoided; the minimum interference should be the aim. The mucosal lining of a sinus should not be removed; in the case of the antrum a free infra-turbinate opening, occasional douching, the use of argyrol or ionization, and time are often sufficient. No part of the lower turbinate need be sacrificed, as by levering it inward sufficient operative access can be obtained. Polypi were found in 14 per cent. They are rich in eosinophils, whereas those from non-asthmatics are rich in basophils. Zinc ionization has been found valuable in helping to prevent recurrence after removal of polypi.

It is true that asthmatics are often worse after nasal surgery. Unless they have been detoxicated they usually have an attack within forty-eight hours of operation; this is to be expected, because the nose, being the respiratory orifice, is the most sensitive area in the respiratory tract; but if the deterioration is permanent it means bad surgery. The importance of a clean nose, yet having enough structure left to function, is shown by the difficulty of curing an asthmatic mouth-breather. Only four such cases in 1,500 can I recall. During sleep the turbinates fill up and mouth-breathing is apt to result, which may be a factor in the frequent occurrence of asthmatic attacks in the small hours.

PROGNOSIS

In the recent book¹⁶ by Coca, Walzer, and Thommen, (p. 269), Walzer declines to use the word "cure" of asthma because, as Kahn says, "a hereditary factor is present, not in a certain fraction of the cases, but in 100 per cent." This is true, but a platitude that holds for all diseases from tuberculosis to corns. "The allergist can only hope that his patient may be kept symptom-free and that the period of well-being will be permanent." This also is probably true—for allergists—because they neglect the conditions which breed allergy and are causing it to increase. To neglect any of the factors making for asthma—hereditary, nasal, toxic, allergic—is blind and futile. Little is said in the book of the toxic factor (attention to which alone will give better results than attention to the allergic factor alone), and in the elaborate detail of the thirty-three necropsies recorded¹⁷ mention is made of the adrenals in none—a glaring omission, considering the virtue of adrenaline in asthma and of the effect of toxicosis on the adrenals. However, Hurst also, speaking not from the allergic angle, says the cure of asthma is not to have it. The answer is simple. If they are not mouth-breathers, have no such defect of chest or limbs as to preclude active life in the open, and if they follow treatment mainly hygienic and dietetic, at least 90 per cent. of asthmatic children under 15 can be cured and stay cured.

Of adults less can be said, but the majority can be cured or so improved, provided there is no hypertrophy of

bronchiolar muscle, as to make all the difference between health and disease. The following table speaks for itself. The eight cases are not selected; they are old cases of whom I have heard *accidentally* while compiling this paper; most of them I had not seen for years. The reader will note that the nose demanded treatment in all but one, was beneficial in all, harmful in none, that the family history was positive in all but two, that the asthma was severe in most, and its duration over ten years in five.

Case No.		Date of Attendance		At Time of First Visit		Nose	Family History	Years Free from Asthma
		First	Last	Age	Duration of Asthma in Years			
I	1,080	1926	1927	14	11	+	+	5
II	149	1911	1912	14	10	+	+	20
III	996	1925	1927	45	1	+	+	3
IV	556	1918	1921	31	27	+	-	9
V	749	1925	1926	28	4	-	-	5
VI	1,480	1930	1930	43	36	+	+	2
VII	677	1918	1922	24	14	+	+	8
VIII	754	1922	1928	60	1	+	+	3

CASE NOTES

I. Single child, puny, irregular at school, in bed three months before first visit. Septum resected. Is now 20, and has never lost a day since treatment; has not wheezed for five years.

II. Single child, puny, mouth-breather, worse week-ends. Nasal and orthodontic treatment. Now a tall, athletic man, no mouth-breathing.

III. Six feet, 11 st., systolic pressure 100. Wheezed on least exertion. Can now do two rounds of golf. Has three children, none asthmatic.

IV. Inveterate asthma. Rejected for army on account of heart disease, which cannot now be detected. Teeth, nose, diet all badly needed attention.

V. Engineer in India; skin tested at Calcutta and found positive to fish, fowl, egg, milk, mutton, beef, tomato, cucumber. Called on furlough to say he has had no asthma for five years.

VI. Planter in Borneo; writes thence to say he has had no asthma for two years. Septum resected.

VII. In his fourteen years' asthma had a free interval of three years while leading open-air life in Australia; on return home was seldom free for next three years.

TREATMENT

This has been indicated above and given in detail elsewhere. I would only add that it may save trouble in the case of an asthmatic lady if before the rhinologist looks at her nose and the allergist tests her skin, they look at her shoe heels and at her feet. Free life in the open is not possible without good feet, nor is a cure of asthma without them, a right life, and a righteous nose.

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THE OCCURRENCE OF POLYCYTHAEMIA AND THE VALUE OF LIVER THERAPY IN ASTHMA

BY

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POLYCYTHAEMIA

In the course of an investigation on the effect of liver therapy in asthma it has been noted that polycythaemia is frequently found in cases of true spasmodic asthma. In thirty-eight cases of asthma, counts of over 6 million red cells were recorded in thirty-one cases (81.5 per cent.); not infrequently the red cell count was over 7 millions; in two cases it was over 8 millions, and in one of these the highest figure was 8,750,000 red cells per cubic millimetre. It has been noted also that the polycythaemia occurring in asthma was of an oscillating character, and that the total red cell count, when taken at different times, showed variations of 1 to 2½ millions. The haemoglobin percentage increase did not follow the rise in the total number of red cells at all closely, and usually the colour index was below 1.

Symptoms of polycythaemia were entirely absent, and although some cases had a "high colour," or cyanosis, this was not a constant feature, and cases with definite emphysema and marked cyanosis often presented a normal or low normal red cell count. Splenomegaly was observed once only in a case, which was, however, exceptional in many other respects. There was no reticulocytosis, and no immature red cells were observed. The condition appeared to be one of relative polycythaemia, which varied according to the state of the patient.

Relation to Emphysema

The presence of emphysema did not materially affect the red cell count, and polycythaemia was found as frequently without emphysema as with it. (See Table I.)

TABLE I

Emphysema	R.B.C. over 6 Millions	R.B.C. under 6 Millions
Present	11	4
Absent	10	2
Not mentioned ...	10	1

Polycythaemia was found both in recent cases and in cases of several years' standing; in approximately one-half of the cases the duration of the disease was under ten years, and in the other half over ten years. In one case a count of over 8 millions was recorded three weeks after the first attack the patient had ever had. The fact that a high red cell count may be found both in recent and in chronic cases suggests that emphysema is not an important factor in the determination of polycythaemia in asthma. Moreover, the condition of emphysema in asthma is often not true organic emphysema, but a state of inflation of the lung, which subsides with suitable expiratory exercises and with the abatement of the asthmatic attacks. Even in cases of true emphysema with bronchitis, polycythaemia is not so constantly found as is generally supposed.

Lemon¹ investigated twelve cases of varying grades of emphysema and chronic bronchitis with cyanosis in order to ascertain the possible variations in the blood volume resulting from deficient oxygenation of the pulmonary blood. The appearance of many of these patients suggested the existence of polycythaemia. He did not find

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