

R E M A R K S

on the

NATURE and **TREATMENT** of **ASTHMA**

with an appendix of 25 cases

by

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I N T R O D U C T O R Y .

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So much has been written on Asthma that some regard the subject as threadbare. If any one, tempted to this idea, will but read the recent lectures of Dr Sydney Martin⁽¹⁾ and Dr Samuel Gee⁽²⁾ on asthma, he will find that the nature of the disease is still much more discussed than settled. There are several reasons for this. (1) Asthma is not a fatal disease. Pathologically, it does not leave an anatomical record, except in respect of bronchitis and emphysema which are not peculiar to it. One rarely sees pathological specimens labelled "Asthma." (2) Asthma is not a disease often found in general hospitals, where accurate observations are easier to carry out than in general practice. The asthmatic patient belongs essentially to the general practitioner for whom it is necessarily difficult to examine all the organs involved. On the other hand when the asthmatic patient is sent to a consulting physician he may be again referred to a specialist, and the chances are that neither of them gets the opportunity to see the whole disease.

It was a perusal of the lectures of Martin and Gee and the omission from them of certain facts which had long been known to me and some of which had not, so far as I know, up till then been mentioned, together with the frequency of asthma in the district where my work is carried on, that led me to think it worth while to give a record of my observations in a compact form. Various circumstances, inseparable from

(1) Martin. Brit. Med. Journal 1898. Vol II p 1861 et seq
(2) Gee. " " " 1899 Vol I p 719 et seq

from the life of a general practitioner, have hindered me hitherto, and since that date one or two observers have, on one point at least, stated facts similar to mine.

Instead of raking up the timeworn literature of the subject one will get a ^{better} survey of the various views held as to asthma by summarising these lectures of ^{authorities} authentic like Gee and Martin.

After excluding the ^{dyspnoea} dyspnoea due to emphysema and chronic bronchitis, cardiac and renal asthma, and pressure dyspnoea, Martin says, Spasmodic asthma is essentially due to nervous changes and may be started by varying conditions, such as pollen (Hay Asthma), indigestion (peptic asthma), bronchitis (bronchitic asthma), infectious diseases affecting the lungs or nervous system (pneumonia or influenza), chronic disease of nose and throat (which is not of so great importance as is sometimes stated). After remarking on the complicated relations of the respiratory centre (on the motor side through the thoracic, ~~p~~ phrenic and vagus nerves, on the afferent side through the vagus and the sensory nerves of skin and upper respiratory tract), he says that this centre may be disordered by influences local or distant and starting from the periphery by the sensory nerves of the upper respiratory tract or from the lungs themselves. He mentions the/

pathological theories - acute inflammation of bronchial tubes (Berkart's view) he dismisses at once: for non-inflammatory or vaso-motor swelling of the bronchial mucous membrane he says there is no evidence. He favours the usual view (Reisseissen's, Gairdner's, ~~Salter's~~) that there is spasm of the unstriped muscular fibres of the smaller bronchial tubes, because it explains the sudden coming and going of the auscultatory phenomena, the patchy and rapid appearance and disappearance of the respiratory murmurs and rales at various parts of the chest. He holds that sufficient emphasis has not been laid on the nervous system, else how account for "cat asthma", "Hay asthma", and even "bronchitic asthma" since it is not every case of bronchitis that develops asthma? Asthma, then, on Martin's view is a neurosis due to the "condition of the central nervous system - a condition which cannot be defined categorically but only expressed vaguely as that which is present in the individual predisposed to asthma" - surely a very lame pathology and one all the more remarkable as being given by one who has done so much for pathology from the biochemical standpoint.

It is interesting to contrast with this Dr Gee's views on asthma, ^{given} just three months later.

Remarking on the pathology he says that "signs of bronchitis ✓"

"bronchitis and ^{pulmonary} ~~preliminary~~ emphysema are always found, and although no one would call them the anatomical characteristics of asthma, yet the fact that they are constant, demands that they should not be passed over or dismissed with the remark that they are mere complications." "Either bronchitis and emphysema constitute the morbid anatomy of asthma or it has no morbid anatomy. In either case we must reluctantly conclude that structural lesions cannot help us to a definition."

Of the nervous hypothesis, for which Martin has just voted, Gee says, "one thing seems to be certain - namely, that if there be such a disease as bronchial spasm, pure and simple, it must be a very rare condition, because it is inadequate to explain the catarrh which manifestly attends most, if not all, asthmatic seizures." "The whole subject of bronchial spasm is involved in so much obscurity that we cannot admit more than that it perhaps takes a share in the production of asthma, but is seldom if ever the chief or only cause." He quotes with approval Robert Whytt, "that a true nervous or spasmodic asthma without any other fault in the lungs ^{than} their uncommon delicacy or irritability of their nerves is a disease which we seldom meet with" -- a conclusion almost diametrically opposite to Martin's. Inspiratory spasm is/

is not the cause of the asthmatic paroxysm, Gee says, because the difficulty is often one of expiration; and finally he dismisses the nervous hypothesis. He adopts the hypothesis of "pneumonic asthma, for we have no third explanation before us." He then goes on, with a view to getting at the pathology of asthma, to consider certain closely related diseases viz., (1) a peculiar form of bronchitis coming on suddenly, especially in children, attended by severe dyspnoea but not by much fever and passing off in a few days, with a great tendency to recur. (This he says is really asthma) (2) spasmodic croup which is not laryngismus stridulus. (3) paroxysmal coryza (4) paroxysmal bronchial flux and (5) hay fever. By this breadth of view he gets nearer than Martin, in some respects, to the ultimate pathology of asthma and asks, ⁱⁿ ~~is~~ conclusion, "in what does the asthmatic tendency consist? why are some persons prone to asthmatic catarrh and others not? Why does the disposition run in families? What is the bond which in so many cases connects the several diatheses of asthma, eczema and gout?" An attempt to show that Dr Gee's view, which seems nearly the reverse of Dr Martin's, is merely the obverse of the shield, and in some measure to answer his concluding questions, was the origin of this paper.

The **RELATION** of **NASAL DISEASE**
and of other **LESIONS** in the **RESPIRATORY TRACT**
to **ASTHMA.**

It is evident from a perusal of the above papers that the symptomatology of asthma is as protean as ^{view} as to its nature. It is not intended here to discuss its symptoms. The emphatic feature in all its forms is its spasmodic nature. Asthma might be described as a spasmodic pulmonary - not laryngeal-dyspnoea, which seems to have many causes, is sudden of onset and in its disappearance, if not sudden, is somewhat abrupt and leaves no marked lesion behind. Cardiac and renal dyspnoea are thus excluded. They are not really spasmodic; though renal dyspnoea is very apt, unless the urine be examined, to be mistaken for true asthma.

Spasmodic croup and laryngismus stridulus are also excluded as being laryngeal, and this may seem rather odd, for we cannot exclude asthma of nasal origin. They are allied, perhaps closely, to asthma, but they are laryngeal in seat as well as in origin. There is a further reason why the exclusion of laryngeal dyspnoea and the inclusion of nasal asthma is not so inconsistent ^{as} it seems. The larynx is not an organ of respiration: it is merely the organ of voice necessarily interposed in the airway. The true respiratory orifice, the orifice of the ^{respiratory} cavity, is the nose. This is one point the cases appended emphasize. Like other orifices, it is the most sensitive area of the correlated viscus, or viscera, if one may be forgiven the expression. Disease of the orifice will produce spasm of the viscus, just as irritation at the neck of the bladder will produce vesical spasm, or fissure of the anus will produce spasm of correlated muscles. Consider also how the uterine contraction starts from the os,
or/

or that of the stomach from the pylorus. Before going further it may be as well to remember that the mucous membrane of the nose derives its general sensation through the fifth nerve, which is also the vaso-motor nerve of the nose, and that this mucous membrane is largely a specialised erectile tissue requiring constant vaso-motor control. As Bosworth⁽¹⁾ says, "the nasal passages contain an exceedingly important, perhaps the most important and certainly the most intricate apparatus, connected with the function of respiration, of the whole respiratory tract, and one on whose normal functional activity depends the integrity of the whole of the mucous membrane of the respiratory tract below." He quotes experiments to show that all the moisture and nearly all the heat of the expired air are in normal conditions derived from the nose during inspiration and not from the lungs. Their function is the interchange of gases and not the heating or moistening of these gases. The nose also purifies the inspired air. Remembering this, even though it may be overstated, one can at least be prepared to give credence to the statement that nasal disease is closely associated with asthma, not only reflexly and because the nose is the sensory area of the respiratory tract but because the bronchial tubes and lungs have thrown on them the duty of warming, moistening, and cleansing the inspired air. Martin and Gee both refer to the nose as of no great account in the pathology of asthma.

(1) Bosworth, Diseases of Nose & Throat (1889) Vol II p. 89.

asthma. It was Hack⁽¹⁾ of Freiburg who in 1884 emphasized the importance of nasal disease in asthma and other troubles. This view was carried to excess by his followers and it is already suffering from the usual reaction.

As a general practitioner I wish to emphasize the importance of nasal disease in asthma.

First, from the statistical point of view. The appended record includes 25 cases which are all the cases of asthma I have had the opportunity of ^{thoroughly} treating ~~and~~ ^{or} examining. Only two of these cases had no nasal disease: i.e., in 90% of these cases of asthma there was also nasal disease. In other words, in 90% of unselected cases of a spasmodic disease which affects the respiratory tract and in which it is admitted the nervous system must play some, and probably an important, part, the most sensitive area of that tract, the respiratory orifice, was not normal. Apart altogether from any theory, the mere figures are significant, and they are not the figures of a specialist. It is not suggested that the nasal disease is an equally important factor in all the cases. In cases VI ~~and~~ [&] XXII, XXIV it is of quite minor importance. Still less is it suggested that curing the nasal disease will cure the asthma. Nasal disease tells on the lower respiratory tract in three ways. (1) Reflexly-

orifice!

(1) Hack. "On an operative Radical Treatment of certain forms of Megrim Asthma, Hay Fever" See Morell Mackenzie's "Diseases of Throat & Nose" Vol II (1884) p 360.

orifice, is bound to send impulses to all parts of the respiratory tract. Gowers has somewhere pointed out the analogy between ^cparoxysm of sneezing and tetanus.* (2) Where the nasal disease is obstructive, the onus of warming, moistening and purifying the inspired air is thrown on the bronchi and lungs which are thus constantly being irritated and made liable ^{to} more or less chronic inflammation. (3) A diseased area is always a nidus or focus of inflammation liable to attract disease to itself and to spread it elsewhere. But the importance of nasal disease in many cases of asthma is, even apart from statistics, one of the most certain facts in medicine. In some cases it is demonstrated by the fact that if you cure the nasal disease you cure the asthma.

The first case that made me realise ^{the} full importance of nasal disease in asthma was a miner, aged about 30, who came to me some eight years ago. For three nights he had not slept owing to persistent, severe asthma. As he seemed able to breath easily enough with his mouth shut, I was about to prescribe for him, thinking there was no need to examine his nose. However, I thought it wiser to make sure, and to my great surprise discovered in the left nostril a single, somewhat fibrous polypus ^{mucous} not of the usual mucus type, about the size of a small cherry. This I removed with the snare and the patient so far as I could learn had no more asthma. This however is not a very common result.

Again!

*. Violent sneezing & tetanus both involve contractions of muscles in
 he .*. Violent sneezing & tetanus
 head, neck, trunk & limbs.

Again in an asthmatic patient you may, by ^{interference with} ~~inference of~~ the nose, allay or produce an attack of asthma. The appended records show that while operative treatment ~~On~~ the nose is a material aid to relieving asthma, yet very frequently ~~asthma~~ a few days after a nasal operation, especially one with the cautery, the patient will have a severe attack. This is due to reactive inflammation and swelling of the nasal tissues. Inflamed surfaces come in contact with and irritate each other. This "operation" asthma occurs usually about the third day. See cases II, IV, XII, VIII. The most remarkable and indisputable proof of the influence of nasal disease in asthma is the effect of applying a solution of suprarenal ^{extract} ~~solution~~ to the nose in cases where the attack is accompanied by engorged turbinals. (So far as I am aware I was the first, at least in this country, to treat asthma in this way. Swain⁽¹⁾ of New Haven, U.S.A., had in May 1898 pointed out the value of suprarenal solution in acute congestion of the nose and in hay-fever, and Solis-Cohen⁽²⁾ used it dissolved under the tongue for relieving his own asthma.) If in such a case you apply a Cocain solution and then one of suprarenal extract to the turbinals, within five or ten minutes they will be blanched and shrunken, the dyspnoea will have gone, the wheezing

(1) Swain "Laryngoscope" Jan 1899 Vol vi p 41.

Mullen (International Clinics Vol iv series 7) quoted in Med. Annual 1899 p 417, recommended suprarenal solution as a haemostatic for nasal operations. Bates, of New York, was probably the first to apply the remedy for local affections ("Laryngoscope, loc. cit. p 42)

(2) Solis-Cohen. Philadelph. Med. Journal 13-8-98 quoted in Med. Annual, 1900, p 90.

wheezing rales in the chest will have disappeared and, in some cases at least, the cardiac and hepatic dulnesses, previously reduced, will have again increased. (See Cases II, IV, XVI.) This fairy tale I have verified repeatedly, but it needs explanation and qualification. The effect varies at different times in the same case and is less in some cases than in others, depending partly on the thoroughness of the application, partly on the nature of the nasal disease. The effect is greatest where the turbinals are engorged and lying against the septum; it is least where there is actual inflammation of nasal tissues. The above treatment will leave normal tissue and simply engorged over-full turbinals, almost white and quite shrunken; inflamed tissue is much less affected and stands out red and defined. Again, as to the alteration in the chest resulting from this treatment, it is probable that the more or less complete cessation of the rales is largely due to the quiet breathing as compared with the preceding labour-ed efforts of the patient and that the increase of the hepatic and cardiac dulnesses results from the lung ceasing to be overdistended. Moist rales and alterations resulting from emphysema are not affected. But the alteration in the patients condition, is, at least sometimes, marvellous. Some patients come to me or send for me frequently just to get relief in this way. The action seems to be a reflex one produced by the local effect of the drugs on the nose. It is not due to the vaso-constrictor action of suprarenal extract absorbed into

into the system for (1) the time is too short to render such systemic effect probable and (2) I have frequently applied suprarenal solution to staunch bleeding from skin incisions in small operations, and this it does promptly and for a considerable time.

Before passing from this question of nasal disease in asthma it is well to remember that asthma and adenoids may complicate each other; but that the dyspnoea of adenoids and that of asthma are different, the former being nasal both in seat and origin, the latter being pulmonary in seat though it may be of nasal origin; that while both may occur in young children and are worst at night, and while the adenoids may have much to do with the production of nasal disease, of bronchitis, and through them, of asthma, yet removing the adenoids will not cure the asthma. This was well exemplified in Case XV. This child aged 5 suffering from asthma, bronchitis, nasal obstruction and adenoids, had its adenoids and nasal hypertrophy removed by Dr Walker Downie with great improvement in its general health and mitigation, but not cure, of its asthma. On the same day Dr Downie removed very large adenoid growths from a case that looked very similar; a child who had bronchitis and great dyspnoea during sleep but no asthma and no great intranasal hypertrophy, and the dyspnoea entirely ceased. Again, when in adults the nasal cavities are full of large growths the patient is apt to have dyspnoea because, being forced to use the mouth for breathing as well as

as for chewing and speaking, these two latter acts especially chewing, prevent the patient getting sufficient breath and every now and then he feels like to choke. And in some patients this is worst at night. Remove the growths and you cure the dyspnoea just as with adenoids. Doubtless such cases sometimes get the reputation of being cured cases of asthma and case XIX may be amongst them. In this matter the general practitioner has an advantage over the specialist. *He sees the whole case.*

Considering the appended cases still further, we find sometimes hypertrophy of the lingual and faucial tonsils.

(Cases XIX and XXII.) The lingual tonsil is a structure that is apt to be neglected. When hypertrophied it is apt to cause constant irritating cough. Though it probably has little to ^{do} ~~do~~ with asthma it ought always to be examined and if necessary treated.

Case XX seemed most benefited by treatment of lingual tonsil, though the nose was also attended to. Painting base of

of tongue with Cocain solution, seemed to allay patient's attacks of asthma a good deal.

In the two cases which shewed no nasal disease, a mother and daughter, bronchitis had been more or less persistent for years before the asthma began, so that the bronchial tubes would be more or less irritable - the very parts that ^{on} one theory, are the active cause of the asthmatic spasm. In the cases recorded, then, we find a lesion, a vulnerable spot, in the respiratory tract in 100%.

One word on those peculiar cases called "cat" asthma, in which the presence of a cat or other animal, causes asthma. That this influence is conveyed through the sense of smell and acts through the nose, is undoubted. There is not one of the senses in which acuteness varies more amongst different individuals than the sense of smell. This peculiar "cat" asthma of itself speaks for the influence of the nose in asthma in general, as also does the irritable condition of nose found in cases of hay-fever which go on to "hay asthma".

The significance of the

PERIODICITY OF ASTHMA.

Though there is a lesion, a source of irritation, in 100% of the cases of asthma, it is not every case of nasal or bronchial disease that develops asthma. This brings us to another striking feature of the cases recorded, ~~of~~ which has not been pointed out before - so far as I can ascertain from the literature I have read and from conversation with medical friends, though Bristowe⁽¹⁾ refers to it in a general way - and one connected with the essential pathology of asthma. Ten of these cases were men, the rest were women and children. In eight of these ten cases the asthmatic attacks occurred more or less regularly on Mondays, sometimes beginning on Sundays, sometimes being worst on Tuesday (Case XII). The two of men not showing this "week-end" periodicity were Case I, a miner who came to me before I was aware of this peculiarity and who had not long had asthma, and Case VI, who was an architect, having regular meals all the week and no great bodily labour. The first time I became aware of this "week-end" periodicity was in 1898 in Case II. The regularity with which he came to me on Mondays for relief was remarkable. Yet this regularity has to be specially enquired for else it may be missed. I found that most working men who had asthma were pretty sure to have their attacks on Mondays. They worked five and a half or six days a week. On Sunday they had very little exercise and usually had more abundant food than during the week and

of!
 (1) Bristowe, *Theory & Practice of Medicine*, 5th Ed. p. 473.

of a different kind and at different hours. A woman's work on the other hand varies much less than a man's as between Sunday and week day. And in most women asthma has not this "week-end" periodicity. In case XXI however, the periodicity tends to show itself. She is a young woman who works hard all week at a tailor's sewing-machine and rests on Sunday. Further, as ~~the~~ boy, Case XXIV, has grown up and especially since he has gone to work, ^{he} has had with great regularity to give up work on Mondays on account of asthma. Case IV was off work a considerable time owing to asthma and operative procedures on his nose, but whenever he resumed work his asthma stuck to Mondays in very striking fashion. Still further, since Case VIII has taken to doing a little cycling on Sundays he has been rewarded for his breach of the Fourth Commandment, as it is in Scotland, by some improvement in his asthma. He has sometimes to work on Sundays and then there is never asthma the following day.

One reason why this week-end periodicity may not already have attracted the attention it deserves is that probably in no country in the ^{world} world is the Sunday more really used as a day of rest ^{than} as it is in Scotland.

Now this peculiarity seemed to me to point very plainly to some error of metabolism, or of elimination, less probably of absorption, consequent on the amount of food not being cut down on Sundays in proportion to the amount of exercise!

exercise, but rather increased.

Another periodicity pointing in the same direction, is that asthma is usually worst at night or just about the time a patient should wake up. This periodicity is well known. Both periodicities, that of night asthma and especially that of what may for convenience be called "week-end asthma," point to a toxic condition of the blood. An expression of this condition should be found in the urine, an expression which should be more emphatic in the urine of night asthma or in that immediately following it than in urine passed at other times, and should be more emphatic during or immediately after "week-end" asthma than in that passed during the middle of the week. If the error be only one of elimination the expression of this error is more likely to be found in urine towards the end of, or immediately after, than during, an attack of periodic asthma. In short we may have to blame a toxin or that old scapegoat, uric acid.

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URIC ACID in ASTHMA.

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With a view to settling the question of uric acid a series of observations was made. The urine was separated into day and night portions of twelve hours each and these were examined separately. There was great difficulty and delay in carrying on these observations in private practice, but the results, though not perhaps absolutely accurate, are relatively accurate enough to allow conclusions to be drawn. To make sure of this a fairly large number of observations was made. Urea was estimated by Doremus ureameter which gives only approximate results, in fact urea tends to be too high. Uric acid was estimated by Rosenheim and Tunicliffe's⁽¹⁾ modification of Gowland Hopkins' process. In it pure uric acid was obtained in the same way as in the latter process, but its estimation depends on the fact that it conjugates with piperidine to form urate of piperidine. A $\frac{1}{20}$ normal solution of piperidine is used for titration; when all the uric acid has been taken up by this, the remaining liquid becomes slightly ^{alkaline} ~~alkaline~~ with slight excess of piperidine. This point is indicated by phenol-phthalein. A few drops of this in solution are added at the beginning of titration so that the liquid becomes pink as soon as all the uric acid is taken up. Calculation is made accordingly. Table I shows urea and uric acid of two men who consider themselves in sound health.

1. is the average of six observations taken ^{on} ~~by~~ one man by myself on three different days.
2. shows the results got by Dr Carstairs Douglas from the same man on another day.
3. shows the results in another man who has nothing

^{wrong!}
 (1) Rosenheim & Tunicliffe. Brit. Med. Journal Vol. 189 p.

| | Urine in cc's | Urea in grammes | Uric Acid in grammes | Remarks |
|--------|------------------|--------------------|-------------------------|--|
| 1. Day | 1051 | 14.2 | .148 | Average of 6 observations on 3 days urine of a healthy man, by myself. |
| Night | 549 | 8.48 | .075 | |
| 2 Day | 1420 | 14.2 | .478 | One day's urine, same man at a different time. Dr Carstairs Douglas |
| Night | 497 | 8.449 | .084 | |
| 3 Day | 597 | 13.129 | .158 | Man with nasal polypi, but no asthma. |
| Night | 398 | 12.332 | .106 | |

Table II

| | | | | |
|---|-----|--------|------|---------------|
| Jan. 25 th 8 a.m. to 8 p.m. | 511 | 14.324 | .233 | Slight asthma |
| 8 p.m. to 8 a.m. Jan 26 th | 313 | 10.004 | .082 | Severe asthma |
| One day) Days in March) Night | 682 | 17.734 | .455 | Severe asthma |
| | 256 | 7.418 | .283 | Asthma less. |
| Average Day | 596 | 16.029 | .344 | |
| o Night | 284 | 8.711 | .182 | |

N.B. The day urines, i.e. twelve hours from 8 a.m. to 8 or 9 p.m. are marked in black; the night urines, i.e. twelve hours from 8 or 9 p.m. to 8 or 9 a.m. are marked in red.

~~Wrong~~ wrong with him except ~~that~~ that he has abundant nasal polypi, but no asthma.

The results here are all much alike except that during the day period in (2) the uric acid is rather high, while that of the night period in both (1) and (2) is rather low. Urea is much the same in all, in no case is ^{urea} ~~urea~~ or ^{acid} uric greater during the night than during the day. It is also to be noted that in (2) the quantity of urine of the day period is ~~thrice~~ that of the night period and thus to some extent accounts for the extremes in quantity of uric acid of the day and night periods. In (1) the quantity of urine of the day period is only twice that of the night. The man who furnished the urine was also working much harder at the time of (2) than at the time of (1).

Table II put below I for comparison shews results got from Case II.

As will be seen, the total uric acid tends to be high as compared with table I but it is much less on the night when asthma is called severe than on the night when it is less: the reverse holds for the uric acid of the daytime. Both urea and uric acid are up on the day period of severe asthma. *

Tables III and IV are from Case (IV). III was during September when asthma occurred every night. As will be seen the uric acid is high, higher sometimes even at night than

*: Note also the diminished quantity of urine in Table II especially at night as compared with Table I. On one night of severe asthma, not referred to in the table, this patient passed no urine at all.

Table III

| | Urine in cc's | Urea in grammes | Uric Acid in grammes | Remarks |
|---|------------------|--------------------|-------------------------|--|
| Sept. 14 th 8pm to } 15 th 8 a.m. } | 469 | 11.253 | .6290 | Asthma more or less at throughout, worst at night & on waking. |
| 15 th 8am to } 8 p.m. } | 426 | 9.804 | .3317 | |
| 15 th 8pm to } 16 th 8 a.m. } | 469 | 11.253 | .3224 | |
| 16 th 8am } 8pm } | 554 | 11.636 | .3766 | |
| 16 th 8pm } 17 th 8am } | 511 | 14.8335 | .4148 | |
| 17 th 8am } 8pm } | 597 | 15.5168 | .2291 | |
| Averages-- | | 12.319 | .3125 | |
| Averages-- | | 13.043 | .455 | |

Table IV (Same patient as in III)

| | Urine in cc's | Urea in grammes | Uric Acid in grammes | Remarks |
|--|------------------|--------------------|-------------------------|--|
| Oct. 27 th 8pm } 28 th 8am } | 284 | 9.0944 | .0731 | Asthma |
| 28 th 8am } 8pm } | 455 | 11.8222 | .2759 | Asthma half day. |
| 28 th 8pm } 29 th 8am } | 341 | 8.525 | .1931 | No asthma |
| 29 th 8am } 8pm } | 398 | 9.1494 | .479 | Asthma |
| 30 th 8pm } 31 st 8am } | 398 | 9.945 | .2996 | Asthma |
| 31 st 8 am } 8pm } | 256 | 8.1824 | .4587 | No asthma |
| Nov 1 st 8pm } 2 nd 8am } | 199 | 9.845 | .2035 | No asthma |
| Average Day | | 9.718 | .4044 | But .2819 = Uric acid in Asthma-free .2851 = " " " Asthma-free periods |
| Average Night | | 9.427 | .1924 | |

Table V (Same patient as in III & IV)

| | Urine in cc's | Urea in grammes | Uric Acid in grammes | Remarks |
|---|------------------|--------------------|-----------------------------------|-----------|
| May. 4 th 7p.m. to 11 p.m. | 191 | | .094 | No asthma |
| 4 th 11 p.m. to 5 th 5 a.m. | 142 | | .0154 | " |
| 5 th 5 a.m. to 10 a.m. | 142 | | .03556 | " |
| 5 th 10 a.m. to 7 p.m. | 317 | | .148 | " |
| | | | <u>.2929</u> = Total for 24 hours | |

Table VI (Same patient)

| | | | | |
|--|-----|-------|-----------------------------------|--------|
| May. 13 th 7pm to 11 pm. | 114 | 4.317 | .3042 | Asthma |
| 13 th 11 pm to 5 am | 142 | 5.346 | .2034 | Asthma |
| | | | <u>.5076</u> = Total for 10 hours | |

than during the day. Urea also tends to be higher during the night than during the day. Note the averages.

IV was during October when asthma was less troublesome. On the whole the excretion of urea is less, that of uric acid decidedly less, but there is no regularity. For instance on the night of October 27th when there was asthma, urea is 9.09 gm, uric acid .073 gm, but on the night of November 1st when there was no asthma urea is again 9.845 gm, but uric acid is .205 gm. If periods of 12 hours during which there was asthma, and similar periods asthma-free, be taken we get for the asthma periods an average of 10 gm urea and .256 uric acid: and for the asthma free periods, 8.8 gm urea and .284 uric acid. So that uric acid does not tie itself regularly to asthma. The next two tables, however, are interesting.

They are from the same patient as the preceding two.

Table V shows the uric acid excretion at different periods of 24 hours free of asthma, and table VI the urea and uric acid of two corresponding periods with asthma the whole time. As will be seen the total uric acid of the latter two periods though covering only ten hours is twice as great as that of the whole 24 hours of the asthma-free period.

A somewhat similar result is seen on Table VII. The case (XXII) is a lady suffering from mal-nutrition, her weight (in walking dress) is only $6\frac{1}{2}$ stone. For 9 hours during which there was asthma the total uric acid is gm.3848:
for/

Table VII (Female)

| | Urine in cc's | Urea in grammes | Uric Acid in grammes | Remarks |
|----------|---------------|-----------------|----------------------|---|
| 9 hours | 5 pm to 10 pm | 170 | 3.9215 | Asthma. Total urea = 5.797 Total uric acid = .3848 |
| | 10 pm to 2 am | 170 | 1.8755 | |
| 14 hours | 2 am to 7 am | 170 | 2.3870 | No asthma Total urea = 5.57 Total uric acid = .1045 |
| | 7 am to 4 pm | 114 | 3.183 | |

Table VIII (Female, bronchitis, emphysema, normal nose, no asthma)

| | Urine in cc's | Urea in grammes | Uric Acid in grammes | Remarks |
|-----------------------------|---------------|-----------------|----------------------|--|
| July 24th 8 pm to 30th 8 am | 438 | 9.9684 | .10378 | Averages Urea Day 7.966 Night 10.366 Uric Acid Day .1692 Night .1213 |
| 30th 8 am to 8 pm | 483 | 7.966 | .1676 | |
| 30th 8 pm to 31st 8 am | 511 | 11.7576 | .0811 | |
| 31st 8 am to 8 pm | 426 | 8.52 | .1814 | |
| 31st 8 pm to Aug 1st 8 am | 426 | 9.372 | .239 | |
| Aug 1st 8 am to 8 pm | 256 | 7.4124 | .1587 | |

Table IX (Female, diseased nose, asthma)

| | Urine in cc's | Urea in grammes | Uric Acid in grammes | Remarks |
|------------|---------------|-----------------|----------------------|---------|
| Feb. Night | 568 | 13.0732 | .2996 | |
| Day | 540 | 12.9595 | .3248 | |
| Night | 313 | 8.4407 | .203 | |
| Day | 426 | 9.8049 | .25753 | |

Table X

| | Urine in cc's | Urea in grammes | Uric Acid in grammes | Remarks |
|-----------------------------|---------------|-----------------|----------------------|--|
| Sept 11th 8 pm to 12th 8 am | 511 | 16.3680 | .3340 | No asthma |
| to " 8 am to " 8 pm | 483 | 12.558 | .17676 | " " |
| to " 8 am to " 8 am | 539 | 10.241 | .1137 | Asthma |
| 15th 12 noon to 8 pm | 284 | 5.9682 | .1097 | Asthma this evening after a hearty dinner, as this is only an 8 hour period. A 12-hour period on this scale would give Urea 8.452; Uric acid .4645 |
| 16th 8 pm to 16th 8 am | 398 | 9.1517 | .2992 | |

for the succeeding 14 asthma-free hours the total uric acid is only gm.1045, say one third. Urea is much the same for the 2 periods. Contrast this with Table VIII which is from a woman weighing about 10 stone, who suffered from chronic bronchitis, marked emphysema, and probably some *bronchiectasis* ~~ectasis~~: and who had neither nasal disease nor asthma. The uric acid is less. Table IX is from Case (X) an asthmatic lady weighing about 8 stone, who frequently has asthma but who happened to have little or no asthma during the time represented. Here again uric acid is high. This lady has to my knowledge for a long time passed excess of uric acid. Finally, on Table X we have somewhat irregular results in an asthmatic patient (Case XX) during asthma and asthma-free periods. There is decided increase in the uric acid of the period ~~as compared on period~~ marked "severe asthma" as compared with other night periods except the first which was asthma-free and has the largest excretion of all. To sum up. The results here stated and others which I have, go to show that asthma patients have a relatively high excretion of uric acid, even when they have no asthma. This excretion tends to be highest during periods of asthma, and may even be higher during the night than during the day, thus reversing the normal. But this rule is not universal for rarely (see Table II) uric acid is least when asthma is worst, as if it were being held up. We can/

can thus see that there is a close connection between asthma and excessive excretion of uric acid, and this answers, to some extent at least, Dr Gee's question, "What is the bond which in so many cases connects the several diatheses of asthma, eczema and Gout?"

The toxicity of uric acid and its power to produce spasm will be considered later.

Note. After I had begun these observations, Dr Armstrong, of Buxton, wrote a letter to Brit. Med. Journal (3rd June 1899, p 1331) expressing surprise that at a recent meeting of the Laryngological Society none but Dr Dundas Grant had remarked on the connection often observed between Asthma, Eczema & Gout. The subject of discussion was Asthma.

I wish also to add that it is, to say the least, significant that in all the cases of asthma in which the estimation of uric acid was undertaken, it was found to be high & there was more or less disease of the respiratory tract; whereas in each of the two cases where there was marked disease in the respiratory tract (nasal polypii in Table II, chronic bronchitis with great emphysema in Table VII) yet without asthma, the uric acid was not high.

My friend H. R. H. Lawson, who is a chemist & well acquainted with such processes, gave me material assistance in the estimation of urea & uric acid, especially latterly. To him I wish to express my best thanks.

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TOXINS and ASTHMA.

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As I have shown, the relation between uric acid excretion and asthma paroxysm is not constant, and while working at the relationship between uric acid and asthma I became acquainted with ^{Bouchard's} Bouchard's work "Auto-intoxication in Disease"⁽¹⁾ and I think that there, though he does not show the relationship I wish to point out, may be found the true explanation of the asthmatic paroxysm. Speaking of normal conditions he says: ^(p41) "The urines of the day period do not only differ from the urines of sleep by a toxicity twice greater, but the toxicity of these two urines presents differences of a qualitative character. The urines of sleep are always markedly convulsive." (This would explain night asthma which is apt to be worst towards morning.) "Those of the day period are very little or not at all convulsive, but they produce narcosis..... What is certain is that during the day the body forms a substance which when accumulated would induce sleep, and that during sleep it elaborates instead of this narcotic substance, a convulsive substance which when accumulated could produce muscular twitchings and induce waking." Bouchard goes on to point out that the urines of the day period and those of the night period are antagonistic, and that a mixture of the two gives a toxicity less than the toxicity of the less toxic urine. Again on p43 we read, "The toxicity of the urine of sleep being only half of the toxicity of the urine secreted during an equal period

of/

(1) Bouchard. Oliver's Translation.

of the day, we might think that the urine of repose ought to be less toxic than the urine of muscular effort. But it is the contrary which is true. One day of great muscular activity spent in the open air in the country diminishes the toxicity of the 24 hours by one-third, and on that ^{day} the toxicity does not diminish only during the time devoted to muscular exercise. The toxicity which diminishes during work remains less during the repose which follows this work and during the sleep which succeeds this day of muscular activity." And to prove this he adduces experiments. If then we regard the urine as an expression of the blood, Bouchard's remark is exactly what we should have expected him to deduce from observations on "week-end" asthma. And he is a Frenchman, probably unacquainted with the Scotch Sunday. Bouchard's remarks amount to this:- diminish exercise and you increase the toxicity of urine - you increase the narcotic effect of the day urine, the convulsive effect of the night urine. If asthma is due to such a toxic agent, we should expect it to be worse at night and especially on the night following a quiet day; we should expect it to be worse on Sunday night or Monday morning. And I have shown that it is so. Further, with regard to the increase of the narcotic effect of day urine caused by want of exercise, it is to be noted that Case IX, which is a case of week-end asthma, often complained at first of an oppressive somnolence./

somnolence./ Further, Boucard shows that (pp 62,63) ~~that~~ the convulsive substances obtained from urine are insoluble in alcohol, and are two, an alkaloid and potash. This alkaloid is found in less quantity in the urine of the day period than is the narcotic material, but it is of less physiological activity." His previous remarks leave one to infer that it is in greater quantity in the night urine.

Further, many of these cases of asthma show gastro-intestinal disturbance in all the severe cases. (I think it was present in 3 Cases II, VII, IX, X, XII, XVII, XX, XXII and probably others.) Many of them had to avoid certain articles of diet in order to avoid asthma. In case VII a very bad attack occurred after eating a lot of fruit and beans. And the alterations of diet both as to time, kind and quantity on Sundays, also tend to cause gastric derangement, though I do not think to such an extent or with such regularity as to be the sole cause of "week-end asthma." Patients suffer too much not to mend their diet in this way, and do alter it where they find it advisable. The periodicity of the asthma is much too regular for this to be the sole cause of it. But it would be an adjuvant to other toxic conditions, since, as Boucard shows, part of the toxicity of urine is of intestinal origin. (p101) And again p 140 he says "If I suppress intestinal fermentations I cause the toxicity of urine to diminish: I cause it to diminish but not to disappear, since I only suppress one of the natural sources of its toxicity."/

-city." It was thought possible that indicanuria might show some relationship to asthma, especially if asthma were due to an error in absorption. A number of observations therefore, were carried out on Herschell's⁽¹⁾ comparative method, modified to suit 12-hour periods. "Take the 24 hours urine and dilute up to or evaporate down to 50 oz.; 10 c.c. of this prepared urine, 10 c.c. HCL, and 2 c.c. CHCL₃ are mixed together. Compare the colour assumed by the chloroform with a colour scale experimentally constructed to indicate known quantities of urine." It is not necessary to construct a quantitative scale; an arbitrary colour scale is sufficient. The urines passed during or immediately after asthma-~~free~~ periods were compared with the urines of asthma-free periods. So far as the present enquiry is concerned the results are not² worth giving. An indican reaction was not at all constant, was often very slight[&] was worst[†] in a case of "bilious vomiting" which was quite unconnected with asthma and was taken for a comparison. Possibly much more decided indican reactions would have been got by specially selecting the urine passed during or after such asthmatic attacks as were accompanied by violent digestive upset, but the urines were not selected urines and the result showed no constant relationship whatever between indicanuria and asthma. Therefore it is probable that any poison absorbed from the stomach or intestine will only be an

adjuvant,

Herschell. Brit. Med. Journal. 1899 Vol II / 1257

adjuvant to that which is most active in asthma and which probably results from faulty metabolism or defective ^{elimination} ~~elimination~~ or both. The proof of the truth of this hypothesis of the toxic nature of asthma would be the injection of urine passed during or immediately after an attack of asthma, into animals and comparing the results with the injection of urine of asthma-free periods. One would expect convulsions to be produced by much smaller doses of the former than of the latter.

Such a proof it is impossible to carry on in a country town. But I can adduce clinical evidence of the truth of the toxic hypothesis.

Many of the appended cases showed symptoms referable to toxins. Several of them showed Urticaria. Case VI had only one attack of it, though he had also, at a time when his asthma was specially frequent, an attack of Raynaud's disease or "local asphyxia"- a very interesting complication to which I shall again refer, and one which is held by some to be due to a toxin. Case X has frequent Urticaria: in Case XIV it is chronic and occurs nearly every night. In case VII an attack of asthma was usually signalled beforehand by tingling and irritability of the skin which became dry and harsh. This case and Case IX also used to develop petechia~~R~~ of the skin during severe asthma. Many writers have pointed out the toxic origin of urticaria and Bouchard refers (p 164)

dilated stomach with

(p 164) to the connection of cases of paroxysmal dyspnoea recalling asthma, as well as ^{with} sneezing ^{& coryza} ~~and dilated stomach~~. Many cases are on record of erythematous rashes following enemata^g due to the washing of toxic substances from the faeces and their absorption by the bowel. The following remarkable case of the "Acute circumscribed cutaneous oedema" of Quinke⁽¹⁾, or subcutaneous "non-itching" urticaria, was clearly due to a toxin. It is remarkable because, like some cases of asthma, it occurred on four successive Sundays and only on Sundays.

G.F. aged 17, a carter, came to me 12th August, 1894. When he woke on Sunday July 29th he found pronounced, hard, brawny, painless swelling of one eyebrow. It disappeared gradually toward evening. The following Sunday morning August 5th he found similar swelling of lower part of forehead and both eyebrows and between 4 and 5 p.m., swelling of lower lip, not to the same extent but noticeable. The swelling of lip had disappeared when he woke on Monday morning that of the brow disappeared during the day while patient was at work. Today, Sunday, Aug 12th patient comes with similar swelling of upper lip which began after dinner at 2 p.m., and reached its present height (7 p.m.,) in two hours. The swelling is like that caused by a wasp-sting, being brawny, uniform, not putting on pressure, but painless. The lip projects quite an inch below the lower, in fact as far out!

(1) Quinke described the disease in "Monatshefte f. Prakt. Dermatologie, 1882. My own case is transcribed from my note-book.

out as to be on a level with the tip of his nose. Teeth and gums are sound. Patient never touches shell fish: he took fish last Sunday, but not till after the swelling had appeared. He smokes three ounces of black tobacco per week: but he eats much more nitrogenous food on Sunday than on other days. On Saturday night he has always a glass or two of beer. Much less exertion and more food on Sundays, and probably the same applies to Saturday evenings. He came to Hamilton only nine weeks ago: before that he used to work (on Sunday and Saturday) as a shepherd and never had such swelling. He is a strong well-built lad, tongue clean, pulse 72 and soft, heart sound. Urine, straw colour, no albumen, no sugar. Some weeks later I saw him and found that a similar swelling recurred the following Sunday and slowly disappeared. He took a purgative as advised and never had ^{any} ~~any~~ recurrence.

What feasible explanation is there for this case with its "week-end" periodicity than that it was of toxic nature? And if so, then so also of "week-end" asthma. One other peculiar case of "bronchial-flux" of toxic origin should be remembered here, but will be more appropriately considered later.

All of these instances point to a toxin of "convulsive" nature, a spasm-producing toxin, acting on the blood-vessels; and it is at least equally feasible to explain the bilious vomiting and the diarrhoea occurring in some cases of asthma (Cases IV and VII) as due to a spasm-producing toxin acting on stomach and intestine as it is to ascribe them to local irritation, though that may be and often is present.

Let us now sum up the case for the toxic pathogeny of asthma. Many cases of asthma show periodicity. That it is apt to occur at night or on waking is well-known, I have shown that many cases especially among working men, occur chiefly on Sundays or Mondays. Bouchard has shown that the toxins of urine of night periods or of periods of rest are more convulsive (^{spasm-producing} ~~spasmodic~~) than the urine of day periods or of periods of muscular activity. This condition of the urine is but the expression of the toxicity of the blood. This condition of blood and of urine would exactly explain the "night" and "week-end" periodicities of asthma and is, on the other hand, what we should expect to deduce from those periodicities. And there is no other feasible explanation of those periodicities. Further there are phenomena in asthmatic patients (urticaria, deep oedema, "local asphyxia") and in allied disorders, which can be most readily explained on the toxic hypothesis.

Cases of asthma which do not show marked periodicity can be explained on the same hypothesis because they are mostly patients whose habits are less periodic.

Diathesis means ^{biochemical} biochemical tendency. The asthmatic diathesis, on the view urged here, would mean the tendency to form more than the normal quantity of certain toxins or to fail to eliminate these rapidly enough. The accumulation of these in certain patients causes asthma, ^{because} because such patients/

patients have irritable or abnormal respiratory tracts and have, in most cases, lesions in the most sensitive area of the respiratory tract, the nose.

It is easy to understand, therefore, how reflex influences passing from orifice to viscus will find in the convulsive toxins of Bouchard, just the stimulus needed to produce spasm there - in the lungs. Further, the accumulation of such "convulsive" toxins will explain the irritable state of the nervous system on which Sydney Martin lays all the emphasis. It is possible, ~~and~~ even, on this hypothesis to explain those difficult cases of "cat" and "hay" asthma. Smells or dust will give to unusually sensitive noses the stimulus needed by such a poisoned nervous-system to evoke an asthmatic spasm. Goodhart⁽¹⁾, referring to the connection between urticaria and asthma, says "it is sometimes associated with or replaces asthma, as a case of asthma produced by contact with cats will show. I have three records of such cases" On what other hypothesis can we explain the urticaria? This point will become even clearer later on.

The relation of uric acid to "convulsive" toxins remains to be considered. Bouchard⁽²⁾ holds, and quotes experiments to show, that uric acid, even when injected in fairly large quantity, has very feeble toxic power. On the other hand he shows that urea^{is} diuretic⁽³⁾. And there is little doubt that uric acid is not so. An interesting point in this

respect,

(1) Goodhart. *Allbutt's System of Med.* Vol V p 291

(2) Bouchard. *loc. cit* p 57

(3) " " " *Cap. v*

respect can be gathered from the tables I have already given. If the average quantity of urine of asthma-free patients be compared with that of asthmatic patients, we find the latter pass much less urine, even including that passed in asthma-free periods. (Only 12 hour periods have been reckoned.)

| | | DAY. | NIGHT | TOTAL. |
|-------|-------------------------|----------|----------|----------|
| URINE | { Asthma-free patients. | 705 c.e. | 520 c.e. | 1225c.e. |
| | { Asthmatic patients. | 470 c.e. | 356 c.e. | 826 c.e. |

Uric acid got in the same way gives for

| | | | | |
|-----------|------------------------|----------|---------|---------|
| URIC ACID | { Asthma-free patients | .215 gm. | .115 gm | .330 gm |
| | { Asthmatic patients | .311 gm | .225 gm | .536 gm |

and this also includes asthma-free periods. If the figures for asthmatic patients had not been limited to 12-hour periods or had been limited to asthma periods, the excess of uric acid would have been greater. Therefore one influence of uric acid is probably that it interferes with ~~with~~ diuresis and consequently with the elimination of "convulsive" toxins. There is also, probably, a prior relationship. Haig ⁽¹⁾ holds that the uric acid excreted comes solely from the proteids of the food. But this view is hardly tenable in the face of recent researches ⁽²⁾. There is every reason to suppose that it is a product of nitrogenous metabolism. If so, it is a gross one and may be regarded as only a less complete oxidation than urea. Excess of uric acid probably means!

(1) Haig, "Uric Acid"

(2) See a resumé of the whole subject with observations by Dr. Cairns Douglas in *Edin. Med. Journal*, January of this year.

means an error of nitrogenous metabolism in which more subtle, less oxidised, and more toxic, products are evolved.

So that uric acid while holding a more or less constant

relationship to asthma, is probably rather an index of other *products - toxins - formed in the body*
~~products - toxins~~ in the body, as well as a hindrance to that elimination of them which would be better promoted by the more perfect oxidation-product and *diuretic* urea. Amongst these toxins is probably the "convulsive" toxin of Bouchard.

The ACTUAL SPASM

in ASTHMA.

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It is not my intention to discuss in detail the theories as to ^{the} actual condition of the lung during the asthmatic spasm but only to discuss those theories in the light of the view I have ~~discussed~~ advanced. The two leading theories are (1) spasm of the bronchial involuntary muscles ^{causing} contraction of the lumen of the softer bronchial tubes, and (2) some rapid ^{tumefaction} tumefaction of the mucous membrane of the bronchi.

(1) Martin and most men adopt the former view: Gee the latter. (2) Gee says asthma is "pneumonic" in nature, that the obstruction

of humours upon the air passages. is not due to any sort of spasm but in Willis's phrase, *to an afflux*

"We have seen that there are five or six affections of the respiratory tract which are closely allied to asthma [a peculiar form of bronchitis, spasmodic croup, paroxysmal coryza, paroxysmal bronchial flux, hay fever] and which may be said to constitute together with asthma, a special class of diseases, characterised as follows. They set in suddenly, they ^{then} speedily reach their highest point of severity, their duration is short, they are apt to recur, and in the intervals the patient is more or less free from signs of disease until the catarrh becomes chronic.

"All these diseases are catarrhal. Now catarrh implies two conditions - namely increased secretion and swelling of the membrane: but as a matter of fact, neither of these conditions is constant. The defluxion or increased

"secretion/

(1) Martin loc. cit
(2) Gee. loc. cit

"secretion may be absent, or at most may be very small,
 "and this constitutes Laennec's dry catarrh. The swelling
 "is more constant, at least in acute catarrh; in paroxysmal
 "coryza, which has so close an affinity with asthma, the
 "swelling can be seen; moreover, both defluxion and swelling
 occur with great rapidity. The two concomitants of catarrh
 "will explain the most important symptoms of all the dis-
 "orders in question, including asthma."

Gee scouts the idea of spasm, but it is plain that he cannot get rid of words implying spasm from his ^{terminology} ~~terminology~~. Further, such swelling and ~~catarrh~~ as he holds out for, are quite consistent with Sir Andrew Clark's ⁽¹⁾ view of a "vaso-motor neurosis" producing changes in the bronchial mucous membrane similar to those of urticaria in the skin. Martin thinks if this were so "a profuse expectoration of a ~~thick~~ mucoid and watery liquid would occur after the paroxysm." The answer to this objection is that one ^{would} ~~would~~ not expect it in some cases and that it does occur in others. There are great varieties in urticaria. Fitzgerald ⁽²⁾ has pointed out that in angio-neuroses ^{neuroses} affecting the skin, the swelling may be cutaneous, subcutaneous or subcuticular, only ^{on} ~~in~~ the last form becoming vesicles or bullae. In ordinary urticaria there is swelling but neither pitting nor vesicles. The centre of a wheal is usually blanched and ^{Schleich's} ~~Schleich's~~ method of procuring surgical anaesthesia by infiltration of weak sedative solutions into the skin, depends on driving the blood

(1) Clark. American Journ. of Med. Sciences 1886, vol 91.
 (2) Edinburgh Hosp. Reports Vol I p 179. } (3) Schleich. See Med. Annual.

blood out of the tissues and causing wheals. All we should expect then, in internal urticaria involving the bronchial tubes is, especially at first, very little, tough secretion. And this is what happens often in asthma. Again many cases of asthma are accompanied by or alternate with urticaria (Cases VI, X, XIV,) Swelling similar to Quincke's deep oedema frequently attacks the pharynx. Fitzgerald mentions a case of Acute Circumscribed Oedema with a history of asthma. Several interesting cases showing the connection between urticaria and asthma will be found in Ramsay-Smith's book on "Angio-neuroses."⁽¹⁾ An interesting case of angio-neurosis is one which occurred in Case VI of my list. There was, while patient was suffering from slight asthma, an attack similar to Raynaud's disease affecting one thenar eminence - a local asphyxia, or "asthma" of the part. The part was pale and painful and later became dusky. The affection did not last more than 48 hours. In case XIV, which is one of sneezing, urticaria and asthma, there was once an erythematous flush across the nose and cheeks like erysipelas, but without pain or marked swelling. It passed off in a few hours.

On the other hand Ramsay-Smith⁽²⁾ mentions cases of angio-neurosis of the skin accompanied by vesicles and bullae, i.e., free transudation of fluid. And in some cases of asthma,

(1) Ramsay Smith. "Angio-neuroses" pp. 31, 61

(2) " " pp. 9, 14.

asthma the secretion is profuse. In cases VII, IX, XXI, it was so and ^{in two of them ~~there~~} ^{at first} the mucus was viscid and intimately mixed with blood. In the first two of these cases, ^{petechial} ~~petechial~~ formed on the skin. Gee, under the heading of "Paroxysmal Bronchial Flux," quotes a remarkable case mentioned by Beddoes, where a woman was, on four different occasions, ^{like} seized by attacks of spasmodic asthma but much more violent, and on their remission attended by very copious discharge from the bronchi. On the fourth occasion the dyspnoea developed very suddenly and she appeared within five minutes almost suffocated and became unconscious for two hours, during which time a very large quantity of frothy serum (mucus) tinged a little with blood was discharged without any visible effort by the mouth and nostrils. Then she began to cough again and altogether put up 3 or 4 pints of fluid. At the end of three hours she became conscious and slowly rallied. The following seems to me a similar case to this, else I should not have thought of quoting it as there is little resemblance to asthma. It is the more interesting as being probably of toxic origin. A stoutly built lad who drove a butcher's cart round outlying districts and who had no food but an apple between breakfast and supper, went to bed on the evening of 25th August, 1896. He looked a little pale. Next morning he was found unconscious in bed, breathing loudly with white foam coming from mouth and nostrils/

nostrils. His chest was full of bubbling rales which could be heard in the next room. There was no dulness about the lungs, the heart could not be heard but was not enlarged. He could not be roused. The pupils, when the eyelids were lifted, were found to be equal and somewhat contracted, there was no squint; the eyeballs were rolled slowly from side to side. There was no paralysis, as patient occasionally moved his limbs. He vomited a little clear mucus while I was present and his pillow looked as if this might have happened during the night. His bowels moved ^{involuntarily} involuntarily at the same time as he vomited. Six ounces clear urine withdrawn by catheter, gave sp.gr. 1017, albumen, and a reaction to Fehling's solution (?sugar). He remained in the same unconscious condition all day. The rales gradually diminished and by midnight were nearly gone; but the respirations remained very fast, 80 per minute, the pulse rate being about the same. The temperature in the morning was 99° (pulse 120); at noon temperature was 101.5, at 4 p.m., 99°, at 6 p.m., 101.5; at 10 p.m., 99°.

The teeth were rigidly clenched till the evening, when he yawned, and the tongue was seen to be foul. A pint of urine withdrawn in the evening gave the same reaction for albumen and sugar as in the morning.

Patient was seen by four doctors who all agreed he would die. So his mother when she returned from the country burned

burned his abdomen with hot applications and next day the patient slowly rallied and towards evening tried to utter a response to questions. ^{Faeces} Faeces were passed twice, and urine once, ~~passed~~ in bed. The urine got by catheter contained no sugar or albumen. Still slight cooing rale in chest. On the third day he was conscious but somnolent, respirations 35, pulse 72. Throughout the illness he was of good colour, neither cyanosed nor pale. The burn of his abdomen took three weeks to heal. The diagnosis was at first between ~~a~~ brain lesion, probably involving the medulla, and a poison. The complete recovery, the condition of urine, and complete absence of paralysis, favoured the idea of a toxin affecting the medulla. Patient shortly afterwards left the district; so I do not know whether he has had other attacks like Beddoe's patient or ever developed asthma.

If such cases are of asthmatic kindred, then asthma is as varied a disease ^{as} angioneuroses of the skin. The amount of the exuded fluid varies in both. Both affections come and go quickly. Lastly, the mucous membrane of the trachea and right bronchus has been seen during asthma to be deeply injected. Observations on this point ought to be more numerous than they are, for in ^{severe} asthma the vocal cords are abducted **To** their fullest extent and held so, as I have shown in cases XVI and XX. Unfortunately I have no note of the condition of the mucous membrane but my impression is that it/

it was congested. There is much then to support the view that asthma may be, in many cases at least, a kind of urticaria due to vaso-motor spasm. To the other view, advanced by Gairdner⁽¹⁾ fifty years ago, that of spasm of the "scavenger" muscles of the finer bronchi, there is no real objection. To say that such spasm is not present because not seen, as *Spee* in effect, says, is asking for a proof which cannot be got. It accounts for the facts; and these muscles are found in the autopsies of such cases to be hypertrophied. On the other hand thickening and injection of the mucous membrane are also found. In short the theory of bronchial spasm is supported on experimental, pathological and anatomical ~~grounds~~ *And as I have said,* grounds. ~~Moreover~~ it satisfies the clinical facts. In these circumstances to ~~reject it~~ *because* because such spasm has never been seen, is absurd. And there is no evidence against it. The other view, that of vaso-motor spasm, cannot be rejected, ~~because~~ *for* such spasm is frequent in asthmatic patients and it also accounts for many facts of many cases.

But there is no need to choose between the two theories. If asthma is due to a "convulsive" toxin the difficulty would be to understand why such toxin should not act ^{*both*} on bronchial muscles and on those of the blood vessels, possibly more at different times and in different cases, on one than on the other. Nothing better can be urged now for this view than what was urged fifty years ago by Gairdner for his own theory.

(1) Gairdner. ^{"The} *On the Pathological Anatomy of Bronchitis &c" Part II pp 39 et seq.*

"The contractility' says Dr Williams "of the bronchi) ^{resembles} resembles that of the intestines or of the arteries more than that of voluntary muscles or of the oesophagus, the contractions and relaxations being gradual and not sudden'. This kind of contractility is precisely that which empties the arteries of their blood after death." And as I have already pointed out, ~~that~~ there is in some cases, spasm of stomach and intestine, causing vomiting and diarrhoea (Case VII) That is, in many cases of asthma there is spasm of smaller blood vessels (petechia^R, local asphyxia, urticaria, deep oedema) also of stomach and intestine; why then may there not be spasm of the bronchi and of the smaller blood vessels connected with them, producing sometimes merely dyspnoea with little secretion, sometimes dyspnoea with much and occasionally blood stained secretion.?

Postscript: Since writing the above, I had an opportunity of examining the larynx during asthma. It was not a severe attack & the vocal cords were not abducted to their fullest & were moving with respiration. The mucous membrane of the larynx itself was injected, especially that of the glottis. The cords were only slightly pink. The upper part of the trachea, which was all I could see, was slightly but not much injected. The interior of the nose was very red & somewhat inflamed & discharging a good deal of mucus. The turbinals were not so turgid as I have seen them during severe asthma in some patient. Application of Cocain & Suprarenal solutions gave great, ^{but} not complete, relief from the asthma. There was no appreciable alteration in the hepatic & cardiac dullness; but there had been little discussion prior to treatment. The mucous membrane of epiglottis became blanched, probably owing to the solution trickling over from the nose. See also Case IX pp 83, 84

The conclusions then to which I would come are:-

1. That in asthma there are two factors
 - (a) a lesion in the respiratory tract,
 - (b) a toxic condition of the blood.
2. That unless both are present, there is no asthma. Sometimes the one factor sometimes the other, is the more important.
3. That there is in most cases an abnormal condition of the nose, which is important because the nose is the respiratory orifice and the most sensitive part of the respiratory tract.
4. That the "convulsive" toxin normally present in the urine and therefore present in the blood, is probably in excess in cases of asthma. There may be ~~more~~ ^{others} in addition to potash.
5. That ~~excess~~ of uric acid has a direct connection with asthma and may have some direct effect in producing spasm, but more probably represents imperfect metabolism and excess of the normal "convulsive" toxin or the presence of others, and more probably acts by hindering elimination of these.
6. That spasm of the smaller blood vessels does exist in many cases ~~at least~~ of asthma in other parts than the lungs, [&] in some cases at least, probably exists there as well as spasm of the smaller bronchi, which probably is present in all cases.

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T R E A T M E N T .

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The treatment of asthma naturally divided itself into two parts, immediate treatment of paroxysm, ultimate treatment of the disease. In both cases we have to consider the double factor, the lesion in the respiratory tract, the poisoned condition of the blood.

I. The paroxysm. If possible examine the nose. If the mucous membrane is injected and especially if the two inferior turbinals are turgid, apply Cocain solution 5% to 15% on swobs, using the weakest solution which will give an anaesthetic effect. In asthma usually the stronger solutions are necessary. Swobs are safer than the spray, though less easy to apply. I usually combine 5% Resorcin with the Cocain: this keeps the solution fresh and increases the anaesthetic effect. Then apply Suprarenal solution. This does not keep well and is best made fresh every day or two. Grind a gr.5 tabloid of suprarenal extract in a drachm of boiled water: the solution is ready in a few minutes, though it is best to have it ready beforehand. To do good it must be thoroughly applied on swobs. You must see the interior of the nose while applying it. To use Cocain without suprarenal, or vice versa, gives a much diminished effect. They cannot be used in combination because they decompose each other. Solis Cohen for his own asthma dissolves a suprarenal tabloid under the tongue every 4 or 6 hours and speaks highly of the result. I have tried it in this way with less effect than by the nose.

Given/

Given by the stomach, suprarenal is apt to sicken in much the same way as zinc sulphate. Sometimes the treatment of larynx or base of tongue in similar fashion will greatly ~~alloy~~ the spasm. Suprarenal solution is said to be specially useful in hay-asthma and this would be an advantage, for Cocain is a snare and often necessitates the use of stronger and stronger solutions.

Where nasal treatment does no good we have ~~to~~ fall back on inhalation. Nitrite of Amyl sometimes eases, often fails, and sometimes aggravates. Many patients, after trying all the Doctor tries, fall back on "asthma powders" composed of stramonium, lobelia, nitre etc. The cigarette is the best form in which to use these. The effect of these powders is said to depend on the formation of the nitrous fumes and of pyridine. An American doctor, evidently acting on this idea, extols the following prescription which he used as spray for his own asthma and which I have tried and found wanting. Antipyrin gr V, Pyridin in 20, Sod Nitrit gr 40, Tr Lobelia, Tr Stramon, Tr Bellad, Vin Ipecac ~~ad~~ in 100, Glycerin

~~Ag ad~~ 3j, Ag ad 3ij

When there is much bronchial secretion a smart effect is often got from mustard-bran poultices to the chest. But poultices are worse than useless in most cases of asthma.

Treatment of the condition of the blood. A saline purge is often needed in many cases. **F**or internal medication the best drug/

drug is Pot Iod or Sod Iod in combination with others.

Except when there is much uric acid being excreted I always combine it with Liq Arsen and often add Tr. Hyoscyam and, if there is not much bronchial secretion, Vin Ipec. Instead of the last two drugs Pot. Brom. may be ~~instituted~~ substituted with advantage. When there is marked uric acid excess Sod Salicyl is sometimes the best combination with Sod Iod. I have known this give more relief than the ordinary Mist. Iod. et Ars; but many people cannot take it.

In view of the fact that Potassium is a "convulsive" poison it may be an advantage to use combinations with Sodium in treating conditions of spasm like asthma. This seems to me doubtful; for sodium belongs to the fluids of the body, potassium to the tissues, and it is these ^{last} ~~time~~ we wish to reach.

II Treatment of the disease. Local. Semon⁽¹⁾ has recently given a very hesitating view as to the value of treating nasal disease in asthma. He says, talking of the results of nasal treatment in asthma; "the worst of it is that the ~~operation~~ proportion of these really successful cases in my experience is very small compared with those which are only temporarily benefited and even much more so in proportion to the absolutely ~~unsuccessful~~ unsuccessful ones." This however, is only another way of saying that there is always more than one factor present in all cases of asthma. This

is!

(1) Semon. Clinical Journal 7th Feb 1900. p 247.

is often the more important factor and the more difficult to treat. Semon goes on to say, in effect, that in advising patients as to nasal ~~disease~~ treatment he does not urge them to have it done, ^{knowing} ~~knowing~~ beforehand that in a very small proportion of such cases only, real and lasting benefit is obtained; but he explains to them "the ^{present} present state of the whole matter without either urging or dissuading from intranasal treatment." How I prefer to put it is: Asthma depends on more than one ^{factor} factor; simply treating one ^{rarely} rarely cures; but there is little hope of curing where the nasal condition is obviously unsatisfactory, unless that condition be treated; the more pronounced the nasal condition the more hope of real but it may not be lasting, benefit. Two things must always be remembered before condemning intranasal treatment,

- (1) the difficulty of being ^{thorough} thorough without doing too much
- (2) the almost inveterate tendency of nasal lesions to recur

What you wish to procure in intranasal treatment is that turbinal structures (including polypi) shall not touch septal structures (including ridges and spurs) This is often difficult to procure without doing too much, for there is the risk, if too much turbinal be removed, of creating a condition similar to what exists in atrophic rhinitis a condition worse than the first. Especially is this the case if the cautery be freely used, against which there has lately been a widespread outcry. And yet it is most useful if

recurrence!

(1) See a paper by Holbrook Curtis in "Laryngoscope" Vol vi Jan/99 p 32, & discussion on it pp 60 to 64.

recurrence is to be prevented. The difficulty of preventing recurrence of intranasal disease is greater than would be readily imagined, as I know who have had very thorough treatment to the interior of my own nose and find recurrence. And because of this recurrence intranasal treatment often is brought into discredit, whereas the right thing is to have it repeated. (See case III where, by the removal of polypi the asthma was practically cured until the polypi returned.)

Into the details of intranasal treatment I shall not enter except on two points (1) The advantage of using suprarenal solution is great. Not only does it prevent much bleeding and enable one to see what to do and so to do more at one operation, but it helps to define inflamed as distinguished from simply ^{engorged} engorged structures. The latter are blanched, the former are hot. It is to be remembered that bleeding does not occur till about two hours after the use of suprarenal. (2) Asthmatic patients have usually very irritable, tender noses and much resent the use of nasal plugs which they find almost intolerable. A plug is unavoidable after operation on the inferior turbinal: but not, unless in rare cases, after operation on the middle turbinal. In the latter it is usually sufficient to hold a little Aristol powder before the nose and ask the patient to inspire. This covers the wounded surface, dresses it, and prevents ^{serious} serious bleeding. A small dossil of antiseptic wool or gauze is then/

then placed in the orifice of the nostril. This the patient can replace at will and insufflate more aristol powder if desirable. Patients XII and XVI who had previously been treated by specialists, expressed their delight at the comfort they felt in these two respects. In a few rare cases a plug is necessary on account of bleeding after operations on middle turbinal. These can usually be ascertained beforehand and treated for the previous 24 hours with 15 grain doses of **Ca** Calcium Chloride which mostly prevents serious bleeding. The lingual tonsil and larynx ^{should} should not be neglected in the matter of local treatment. As for general treatment the important thing is to try to prevent toxic conditions of the blood and so anticipate paroxysms of asthma. Where asthma is of the "week-end" type a mercurial on a Saturday or Sunday night followed by a saline next morning, will often prevent an attack. Blue Pill seems better than Calomel which sometimes aggravates, Case VII says that whenever she notices her motions getting darker she knows to take blue pill and to this as much as to intranasal treatment she attributes her cure. Apart from this the medicinal treatment is much the same as that given under treatment of the paroxysm. **Arsenic** and Iodide form the best routine combination; **Salicylate** and Iodide, if they can be tolerated, when there is marked excess of uric acid.

The dietetic treatment as insisted on by Hyde Salter should/

should be carefully watched. For great uric acid excess an increase of vegetables and diminution of red meat in the diet is important. Haig would eliminate butcher meat almost entirely; but it is to be remembered that this is the item in which most iron is conveyed. Patients usually know that there are certain articles they must avoid, potatoes and pastry being the chief, but there are individual peculiarities in this respect.

After what has been said on "week-end" asthma, a sufficiency of open air exercise is obviously important, as well as a cutting down of the diet at resting times, both in the evening and at week-end.

In some cases Cod Liver Oil is of great benefit especially in adolescents. In some of the appended cases it seems to me that the cure has resulted from a steady improvement in the whole general condition as the patient has grown to maturity.

Climate has much to do with asthma. This district has a moist relaxing atmosphere and dense clay soil and moreover lies in a hollow. Hence the frequency of nasal disease and so of asthma. If there is one thing which will produce a sodden condition of the nasal mucous membrane it is moisture and hence, as Dr Walker Downie insists, lotions should rarely be used for the nose. It is difficult to select a climate for each individual case of asthma. Much will/

(1) Haig Christid. p 256

will depend on the chief factor in play in the disease.

When there is oedematous hypertrophy of the nasal mucous membrane a dry climate is obviously best. Where ^{pulmonary} preliminary emphysema is great, ~~this~~ ^{the place selected} should also be at a low altitude, though Case VI and another of emphysema without asthma, have greatly benefited by going to Denver, which is rather high for emphysema.

To sum up, most cases of asthma can be benefited (1) by suitable nasal treatment and (2) by suitable treatment directed to prevent the undue formation of toxins and uric acid and to secure their prompt elimination. As a rule, the most difficult cases to aid are those of long-standing bronchitic asthma without marked nasal disease, or those with an inveterate tendency to uric acid excess yet without marked nasal disease. The easiest to aid are those with pronounced nasal disease. Finally, it is not meant that every nasal abnormality needs treatment. (e.g., spurs can often be left alone.) A good, but not invariable, guide is to be got from the effect of Cocain and suprarenal solutions in allaying the asthmatic paroxysm.

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LIST of CASES.

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With regard to the appended cases three points call for remark.

1. **DIAGNOSIS.** The only case in which I think the diagnosis could be disputed is Case XIX. I should hesitate to call it ~~asthma~~; it seemed more like nasal obstruction with similar symptoms to those accruing from adenoids in children. Yet if this were so, it is difficult to understand the great difficulty of breathing constantly recurring at night. I have seen much worse nasal obstruction, without such difficulty. However, in calculating percentages as given above, I have omitted this case.
2. **TREATMENT.** The only point on which I wish to comment is nasal treatment. This was somewhat protracted in several cases, partly through the patient's fault, partly because of the extent of the disease and because I have always acted on the principle of doing too little rather than too much. Some cases I should treat much more thoroughly now. I have also tried so to operate as not to cause patient to lose a day's work in consequence, and have succeeded in most cases.
3. **RESULTS.** With regard to these, "cure" means no return of asthma. "Practical cure" is applied to cases where there is no real paroxysm of asthma though the patient often, and probably correctly, feels he would have it if he did not resort to medicinal treatment, there is "tightness in breathing" but no actual paroxysm. This term is also used of cases formerly subject to severe asthma when the asthma does not recur/

recur unless when patient takes a severe cold (Cases VII, XII)
 In case VII for instance, asthma kept the patient off work
 for seven years; treatment has enabled her to keep at work
 for the last three.

On this understanding, if we include case XIX, there
 are out of 25 cases:-

- (1) 7 cases of cure,
- (2) 4 " " "practical" cure,
- (3) 9 " " improvement, sometimes great,
- (4) 4 " " no "
- (5) 1 still under treatment.

- (1) Of the 7 cases of cure the treatment was purely nasal
 in four. Case I was not long enough seen afterwards to make
 me feel throughly satisfied with it, but the patient was,
 and rather resented my wishing to see him further. He had
 no more asthma while I knew him. Case XIII was one of rath-
 er pronounced asthma. He has had no asthma for a year. In
 case XVIII the asthma was slight. Case XIX may not have
 been asthma. (See above under "Diagnosis.") In these four,
 treatment was purely intranasal. The remaining three cases
 were all adolescents. In two of them (V and VIII) treatment
 was chiefly nasal, but some attention was directed to the
 general health and diet. In Case IX much credit is to be
 given to his having become able to take Maltine and Cod liver
 Oil, the first preparation of the oil has ever been able
 to/

to take; this credit is shared equally by the nasal treatment and, I think, by his having gone to sea as an engineer.

- (*) 4 cases of practical cure. In case III the treatment has been chiefly nasal. After the polypi were removed she had no real asthma but often at bedtime took a dose of her Iodide mixture to prevent an attack which she thought impending. When polypi recurred, so did her asthma. It has again improved since they were treated. In case XVI asthma was severe till the polypi were all removed and the nose healed. In the last 6 or 8 months there has been only one attack of asthma, while patient had a bad cold. Nothing but nasal treatment did this patient any real good. Case VII was one of severe asthma which has been "practically" cured by nasal treatment and by Blue pill and salines at more or less regular intervals. The general health was watched. Case XXII ascribed her cure entirely to Mist. Iod et Ars.
- (3) 9 cases of improvement. In all the improvement has been distinct, sometimes great, enabling patient to work regularly. Those that have improved most are those who have had marked nasal disease. In case VI the improvement has resulted from a change to Denver and from that alone. In case XXIII no treatment did any real good, but there has been no asthma since an attack of pneumonia two years ago. Of these two cases the former had not pronounced nasal disease, the latter none. Two cases (IV and XVI) are still under treatment, but have been long enough under observation to justify/

justify their being classed here.

- (4) 4 cases of no improvement. XXII is a case of mal-nutrition ^{& ~~infection~~} without marked nasal disease. Asthma of two years standing has been little benefited except by treatment directed to digestion and is not likely to improve while patient is in her present circumstances and low-lying house. X is also a case of mal-nutrition (less decided) and of longer continued uric acid excess. Nasal treatment declined. XIV is a case whose health has been steadily drained by a bleeding fibroid. The nasal tissues have long been very irritable and somewhat hypertrophied. This is a case of no improvement. But thorough treatment has not been acceded to. Asthma slight. XXV is a boy with "hereditary" asthma. Bronchitis has been frequent; some intranasal hypertrophy. Treatment declined. (5) One case (XXI) is classed as still under treatment because the nasal treatment was started not long ago, and attacks of asthma have not been frequent.

I. Male, aged 30, Miner.

Severe asthma, fibromucous polypus. Removal of polypus; cure of asthma.

This is the case already mentioned in the text^x, and I regret I can give no further information about the subsequent history of the case.

II. Male, aged 22, Shoemaker, Hamilton.

Bronchitis from childhood, asthma from the age of 14, especially on Mondays. Pronounced nasal disease. This was treated with great improvement to the asthma. Striking effect of suprarenal solution on asthmatic paroxysm, when applied to nasal mucous membrane. Alcoholic habits, two attacks of pneumonia; death during second.

My notes of this case are too long to be given in full. In order to allow for certain facts it is to be remembered that patient was very poor, and as I found out latterly, addicted to drink.

1/5/99. Patient first came to me June 1895 complaining of asthma. He had been subject to bronchitis since he was three or four years old; spasmodic asthma does not seem to have started till he began to work at the age of 14. There was difficulty of breathing, wheezing respiration and mucous spit. These attacks were nearly always on Monday, and were accompanied by "bilious vomiting." He had much more

rest/
^x p10, 8 p5-9

rest and much more food on Sundays than on other days when he had to walk four miles to work and carry his food with him. Patient cannot take eggs because they cause him bilious vomiting. Finding the nasal mucous membrane much hyper-trophied I treated it with the snare and chromic acid, but the treatment was never thoroughly carried out.

The attacks of asthma became for a time less frequent but not less severe. Two years ago he started business for himself, began to have his meals more regularly on week days, to take more exercise and less food on Sundays; and since that time the attacks of asthma have lost their Monday periodicity which was such a striking feature in the case. It used to be quite common for him to be off work owing to asthma on Mondays and only on Mondays. The attacks became now less severe so that he would be off work only once in two or three months and then for two or three days at a time. But slight asthma has become very frequent at night; in fact there are few nights in which he will not have it. He stops work at 8 p.m., and has a light supper of cocoa or tea and bread and butter. Between 10 and 11 he goes to bed and within 15 minutes he begins to have difficulty in breathing and has to get up. If the attack is severe his face gets dusky and on rare occasions, livid. If possible, he burns some asthma powder and inhales the fumes then usually sleeps within/

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within an hour. Three or four hours later he wakes with another attack and has to repeat the inhalation. He sleeps again, but on finally getting up at 8 a.m. he often and suddenly develops another attack. Attacks are rare during the day. If he did not use the inhalation during the night he would have asthma all night. He has often frontal headache probably owing to the condition of the mucous membrane around the frontal sinuses. He has also "creeping" feelings in the scalp. (Compare Case VI.) Patient takes too much tea and too little exercise. Potatoes he avoids because apt to produce severe asthma. Physical examination. Patient is rather an ill-fed, undergrown lad, with somewhat raised shoulders. The chest is slightly barrel-shaped, very resonant to percussion the cardiac dulness being almost obliterated. Liver dulness in nipple line diminished to $2\frac{1}{2}$ " and slightly depressed. There is a fairly full R.M. but no rale except slight moist rale near right base. Heart sounds normal.

Nose. The mucous membrane is as bad again, as if it had never been treated. The whole of it is thickened and beyond hope of restoration to a normal state. Over the turbinals especially, it is thickened, flabby and oedematous. There is constant secretion of watery mucus. There is some/

(65)

some airway in right nostril but none in the left, which is blocked by the turbinals lying against the septum and by a large septal spur. Lingual tonsils slightly enlarged but not sufficiently to interfere with the epiglottis. With the laryngoscope the mucous membrane can be seen right down to the bifurcation of trachea, but shows nothing beyond slight reddening. There is slight thickening of the pharyngeal mucous membrane and a few small varicose veins.

17th. Yesterday I removed the septal spur. Severe asthma came on last night - probably as the result of the irritation caused by the operation - and still persisted on his visit to me. It had not been relieved by inhalation of asthma powder, but was promptly aided by painting the interior of the nostrils with Cocain and suprarenal solutions.

18th. Last night asthma resumed at 7 p.m., but was ^{relieved} relieved by the use of asthma powder. He then stupidly went for a walk and had more asthma during the night. This morning his nose is full of clear, thin, watery mucus. There is great difficulty of breathing especially on inspiration, some cooing moist rales in chest. Suprarenal solution again applied to nose gives some relief but the effect is greatly increased by the subsequent application of 15% Cocain. Within ten minutes almost no rale is to be heard in the chest, /

(26)

chest, and within other five minutes - albeit it is not possible to apply the solutions thoroughly to the nostril operated on - no cooing rales are to be heard and the breathing is quiet and comfortable.

2/6/99. It was on the 15th ult., I removed the septal spur. Whether as the result of this or of patient's carelessness after the operation, or in consequence of influenza which was going about in a sporadic way, I do not know, but on the 20th he was seized after 24 hours of the usual symptoms of influenza with acute pneumonia which involved lower and middle lobes of right lung. Though the illness was a peculiarly dangerous one for him with his starved, ill-developed body and emphysematous lungs, he was out of danger yesterday. It is interesting to notice that while the temperature yesterday and to-day is normal and the pulse 76, the respirations yesterday were 44 to the minute and to-day 34. For treatment, icebags locally and Tr. Mac.Vom and Tr. Digit have been relied on throughout. Paraldehyde was given for sleeplessness.

1.8.99. Patient went to the Dunoon Convalescent Homes after the pneumonia and returned 7th July. From the time he took pneumonia till the second night after his return to Hamilton, (nearly 7 weeks) he has had no asthma. Since that/

(a7)

that he has had slight attacks at bedtime.

17.1.1900. Since last note I have at different times removed from the nose overgrown structures and used the cautery. The result has been that while the operation has usually provoked an attack of asthma on the following day the patient has been, and especially lately, more free of asthma since I removed the septal spur than he has been for years. When he has come suffering from slight asthma the application of Cocain and Suprarenal solutions has always been attended by the same striking cessation of symptoms more or less permanent.

24.4.1900. Owing to some mistake, patient did not return after the 26th January. Probably owing to his habits, he again at the beginning of this month took pneumonia for which he was attended by his club doctor. He died on the 18th. Asthma had again been troublesome during February and March; much remained to be done for his nose. The first attack of pneumonia I was afraid might have been due to the septal operation, but in the light of the second attack a year later which occurred nearly 8 months after I last interfered with his nose and could not have been ^{so} caused, and in the light of his drinking habits, I feel less inclined to blame the operation.

operation.

III. Female, aged 66. Wishaw.

Bronchitis, nasal polypi, asthma. "Practical" cure of asthma by removal of polypi six years ago; recurrence of polypi: great improvement after removal of these. Still under treatment.

Note made, 18.1.1900. Patient first had bronchitis 30 years ago, first became aware of the presence of Nasal polypi 16 years ago, first had asthma 15 years ago. The attacks of bronchitis were frequent and severe even in summer. The attacks of asthma slight at first, gradually became so severe that her friends have thought her dying. Cold in the head was always followed by bronchitis and asthma. Her Medical attendant, though aware of the nasal polypi, did not seem to think that they called for treatment or that they were an important factor in the case.

Patient first came to me six years ago when I removed polypi from both nostrils. For two years afterward she remained practically free from asthma. There was sometimes a little tightness of breathing which made her always keep a supply of Mist Iod et Ars; but she never ^{had} had a real attack of asthma during this period, even though she had a bad attack of bronchitis. Within the last three years the asthma/

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asthma has recurred in a slight form, but neither it nor the bronchitis has been so bad as previously - a result probably to be attributed in part to her taking her medicine. Patient returned to me last November. Polypi had recurred in both nostrils, closing both olfactory clefts. A septal spur in both nostrils; that in the left was a long ridge opposite the inferior turbinal and a considerable source of irritation and obstructions to respiration; that in the right though prominent, not of much importance because far back, growing from only a small base and not interfering with or irritating the turbinals. The polypi in right nostril I snared and subsequently applied ~~of~~ cauterly; the septal spur in left I sawed off to-day.

2.2.1900. There has been practically no asthma since last note till this morning when she had a slight attack. It is to be noted that the morning was foggy and that there is still an inflammatory condition resulting from the operation on septal spur, though the wound is healing well.

18.5.1900. Immediately after last note, patient caught cold while travelling in a railway train and had a severe and prolonged attack of bronchitis. Buds of polypi are again growing from the middle turbinals, in the left nostril they obstruct the olfactory cleft and semilunar hiatus. Re-
moved/

Removed those in right nostril and cauterised their roots. There has been "immense improvement" in the asthma; has never had a bad attack since last note.

IV. Male, aged 35, Miner, Hamilton.

Asthma for five years. Left nostril much crowded owing to septal twist, ridge, and spur. Inflamed turbinals. Removal of spur and turbinectomy. Great improvement of asthma. Still under treatment
Note:- asthma became very regular on Mondays, as soon as patient was able to work regularly.

17.9.99. Has had asthma for five years especially when he gets out of sorts during an east wind, or has disordered stomach. He generally vomits during an attack. It is especially frequent on Tuesday mornings so that he tries to watch his diet on Mondays. He has more food and at other hours on Sundays than on week days. Has had asthma nearly every night this past week, especially on getting up in the mornings.

Note. The septum is somewhat S-shaped so that while there is only a slight convexity high up in the right nostril so as to ^{impinge} ~~infringe~~ on middle turbinal, but leaving the lower part of this nostril quite patent and useful, the left nostril/

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nostril is practically excluded by the convexity being here low down and having a long antero-posterior ridge growing from its summit. An inch and half from vestibule, this ridge is prolonged into a sharp spur which has caused a deep indentation in the inferior turbinal. It is almost impossible to interpose a probe anywhere between the ridge and the inferior turbinal. The middle turbinal also bulges downward toward the inferior and toward septum, so that the posterior part of left nostril is very crowded.

26.9.99. Removed spur five days ago, and to-day the poly-poid anterior end of left middle turbinal.

11.10.99. Asthma two or three times since last note.

21.10.99. No asthma for ten days. Cauterised face left middle turbinal. To take gr.2. calomel every Sunday night.

8.11.99. Sawed off the septal ridge.

6.12.99 Asthma frequent partly as result of operation. Has twice taken the calomel and on both occasions had severe asthma afterwards. 16th. Has been rather better and able to work most of the last fortnight, but to-day asthma drove him home. 6.1.1900. Asthma still frequent. This may be partly due to the soft and spongy condition of the parts operated on. In removing ridge on 8th November a flap of mucous/

was

mucous membrane, reflected as advised by some authorities
 This is evidently a mistake as it leaves ~~an~~ redundant tissue.
 The left nostril still remains too crowded. Uric acid
 deposit ^{in urine} ~~in evening~~. 10th. First attack of asthma since last
 note came on this morning, but did not prevent him working.
 Was sick and vomiting last night. Had to stop Sod. Salicyl
 mixture owing to diarrhoea. The slight asthma still present
 just now has been promptly abolished by the application
 first of Cocain then of Suprarenal solution to the left
 nostril which still remains far too much injected. To take
Sod Salicyl gr x Sod Sod gr v Inf Kent to 3 p t i d.
 17th. Only one slight attack since Mist Salicyl. Iod was begun.
 18th. Took none of the mixture yesterday and bad asthma came
 on this morning lasting till evening.
 25rd. (Tuesday) 24 hours asthma.
 26th. Asthma began at 6 a.m., just now (6 p.m.,) it is less,
 but distinct, and promptly relieved by Cocain and Suprarenal.
 2.8.1900. No asthma till to-day from 6 a.m. till 10 a.m.,
 Has been working all week. 5rd. No asthma. This is the first
full week's work he has done for six months.
 7.8.1900. Has had asthma every Monday for the last five weeks.
 i.e., every Monday since he started to work regularly. The
 attacks come on about 6 or 7 a.m., and last to 10 a.m., some-
 times longer. Cocain and Suprarenal applied to left nostril
 relieve/

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relieve. Complains of Flatulence. To take *Betol grⁱⁱ*,
P. Carb. Lign gr^{xx}. *Plthi grⁱⁱ* at bedtime and ^{to allow} a 5 gr.
Suprarenal tabloid to dissolve in mouth on Sunday night and
at beginning of attack Monday morning.

20th. Removed middle part of left inferior turbinal bone
using cautery and scissors.

21st Symptoms like influenza which is epidemic, but probably
inflammation of nose: pain in head and cheek, temp 102.

Much relieved by Phenacetin and Caffein.

28th. No asthma since operation.

13.4.1900. No asthma for a month till last night a slight
attack came on more like bronchitis with wheezing and
mucous expectoration. Note. (1) Patient has been trying night
duty which does not usually agree with him; (2) there is
small granulation at seat of wound on left inferior turbinal
which is very tender. (3) the middle part of left middle
turbinal is somewhat inflamed and very tender and impinges
on septum, and will have to be removed.

14th Bronchitis yesterday and to-day.

25rd. Chromic acid to left inferior turbinal.

19.5.1900. Has lost only one day's work since last note on
account of asthma. Before he came under treatment he was
often off for weeks at a time. Patient's general condition
is/

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is much improved. To-day performed turbinectomy of ^{enflamed} part of left middle turbinal. This part was ^{impinging} impinged on septum, and suprarenal solution, while blanching the rest of the mucous membrane, left this part red and definite.

21st No asthma. Was working to-day; ^{Monday} / 26th. Still no asthma.

V. Female, aged 21, Bothwell.

Bronchitis and asthma from childhood. Nasal polypi and hypertrophy of mucous membrane. Nasal treatment five years ago; cure of asthma. Lately sneezing ^{slight} fits: recurrence of hypertrophy of turbinals.

Still no asthma.

18.5.1900. Patient first came to me in 1895 when she was 16, complaining of stopping up of nose, sneezing and asthma, the latter two very frequent. She was somewhat anaemic. Her mother said the girl had been subject to bronchitis in infancy and developed asthma at the early age of three. Patient would have to sit up in bed at night for shortness of breath. The asthma seems to have been bronchitic in type there being a good deal of wheezing. I applied Chromic Acid to the hypertrophied nasal mucous membrane gave her a nasal spray of weak spirit and treated the general condition with Cod Liver Oil and Iron. Her general health greatly improved. Nasal respirations became more free and the asthma disappeared. She returned two years later, again complaining of nasal obstruction.

obstruction, and at different periods in 1897 and 1898 I removed polypi and cauterised. There has been no recurrence of asthma.

To-day patient comes complaining of indigestion. On enquiry it is ascertained that sneezing fits have been recurring lately. Again there has been a slight feeling of nasal obstruction, but no more asthma. There is again slight hypertrophy of both middle turbinals and evidence that polypi will again form, though as yet no actual polypi.

VI. Male, aged 38, Architect, Hamilton.

Bronchitic asthma, emphysema and collapse of lung.

Nasal hypertrophy and irritability. No improvement of asthma from nasal treatment. ^{Urticaria} ~~Articaria~~:

"local asphyxia" of thenar eminence. Great improvement on going to Denver U.S.A.

15.5.1900. My notes of this interesting case are somewhat imperfect. The summary of those taken in 1898 is as follows.

Since childhood patient had been troubled with bronchitis, and during at least the last ten years had been subject like his father to bronchitic asthma. Attacks of bronchitis or of asthma had been very frequent during the last five years, often causing patient to leave work for weeks at a time. Latterly he has become distinctly anaemic. About September of 1897 while suffering from asthma he had an attack of "local asphyxia" suggestive of Raynaud's disease. The thenar eminence/

eminence of right hand became blanched and painful and later dusky blue. This did not last more than 48 hours. He has never had another attack. In February 1898 he had severe "cold in the head" and bronchitis, and as the catarrh was passing off ~~asthma~~ ^{at night} came on, accompanied by Urticaria ~~at night~~ chiefly on the limbs. He did not remember having such wheals before. A persistent complaint with him was a feeling of coldness in the scalp and back of neck and difficulty of keeping warm (Compare Case II). During the last year he has been feeling shivery even in warm weather. The following note was ^{made} two years ago:- "There is marked emphysema of the lungs, cardiac dulness is obliterated; at the base of the right lung there is very deficient R.M. Both inferior turbinals and the right middle turbinal are hypertrophied; there is also a thickened, spongy area on each side of septum, touching of which causes sneezing and which puts on pressure."

I advised treatment of the nasal hypertrophy with the electro-cautery and two specialists in Glasgow, who saw the case, agreeing with me, I carried this out, without much benefit. I had long advised change of climate and Dr. Samson Gemmell who saw patient at the request of his brother, ordered this as a matter of urgency; Dr Gemmell found collapse of right lung at base. At the end of the year 1898 he went to/

to Denver, where, especially during the last year, he has very much improved.

VII. Female, aged 34, Teacher, Uddingston.

Bronchitis until 21; asthma began at 27, was very severe, accompanied by petechia^e and striking general symptoms. Hypertrophy of nasal mucous membrane. Local and general treatment. Result is that patient who had been off work nearly 7 years owing to asthma has been at work steadily since treatment was carried out.

4.8.97. Came to me over a month ago complaining of bad asthma. Both of her mother's grandfathers had asthma: otherwise the family history is good. Patient had bronchitis in childhood and until she was 21. Asthma began when she was 27 and has continued ~~persistently~~ ~~well~~ for the last seven years. The attacks are very bad; she is unable to move even in bed, the slightest ^{movement} moving causing great distress: the skin and nails get livid, the veins stand out, there is profuse perspiration, great palpitation, and patient sometimes swoons. There is not much spit; it is tough and sometimes stained with blood. She gets no relief from the expectoration, has much cough and great difficulty in inspiration, but expiration is also difficult. Menstrual discharge may come on/

on quite irregularly during a violent attack. An attack like this will last 36 or 48 hours and leaves her quite exhausted. A profuse branny ^{desquamation} then comes off her skin; her hair gets coarse. The desquamation leaves her skin in its natural condition, fine and velvety. In a few days she will usually have another attack with a repetition of these symptoms. An "illness" with her means 4 or 5 attacks like the above during 4 to 8 weeks. The onset of the attack is specially interesting. Usually about 4 a.m., patient will feel "tightness of chest and tingling of the skin"; then she will begin to pass pale limpid urine in great quantity and she knows by this alone or by her feeling of irritability, that in an hour or two she will have an attack of asthma. She will pass much urine every half hour or so for the first eight hours of the attack; then scanty, muddy urine. The polyuria is often preceded as well as followed by scanty urine loaded with urates, and is often accompanied by sickness and bilious vomiting, flatulence and diarrhoea. Patient never has true urticaria. There is slight but not marked emphysema. I have never found albuminuria.

Nose. Mucous membrane of left inferior and middle turbinals is ^{still} thick and congested. Last month I removed polypi and applied electro-cautery which I have again applied to night. Amyl Nitrite gives temporary relief from asthma; and Pot. Iod./

Pot.Iod,Liq Ars and Tr.Hyoscyam in mixture have prevented any bad attacks. To use a Camphor-Menthol nose-spray.

6.8.97. A slight attack of asthma this morning doubtless due to a great "feed" yesterday of fruit &c. This was followed by the "irritability" mentioned and then by asthma which was relieved by a seidlitz powder.

19.8.97. Has had a pretty bad attack not relieved by Amyl Nitrite but relieved by three doses of the Pot.Iod mixture and cured within 24 hours.

21.9.97. A very bad attack shortly after ~~first~~ ^{last} note lasting 36 hours. She got no relief till she took Blue Pill followed by seidlitz powder. Four hours later she was all right but a week later she had bronchitis. None of the asthmatic attacks referred to since she began treatment have been accompanied by the usual lividity, orthopnoea or desquamation.

17.2.98. Patient says she is very much better. She attributes most benefit to the Blue pill, which she takes only fortnightly instead of weekly as directed. No asthma this year and only a slight attack last December.

2.6.99. Only one bad attack since last note viz., last September, which is usually the worst month in the year for her. She wards off attacks by taking blue pill about once a month, being warned by a feeling of "tightness across the chest" by her motions becoming darker and her urine getting thick./

thick. She feels her nose more comfortable; before treatment the nose felt swollen and congested and she was constantly sneezing.

15.5.1900. A slight attack of asthma last July and again in September following a bad Quinsy. Whenever she feels slight sickness and tightness about the chest, she takes Blue pill followed by a seidlitz powder next morning. She had bronchitis at Christmas but no asthma. The result of the treatment has been that whereas she was off work seven years before treatment she has been steadily at work for nearly two years and could have been at work sooner. There is decided thickening of anterior ends of both middle turbinals and they impinge on the septum: but both inferior turbinals look very well.

VIII. Male, aged 35, Ironworker, Airdrie.

Asthma for years, specially bad on Sundays, mitigated by exercise. Sneezing fits. Hypertrophy of turbinals and of septal mucous membrane; septal spur. Nasal treatment only partially carried out because patient quite satisfied with the great relief procured.

22.10.98. Patient first suffered from asthma 10 years ago and first came to me three years ago. At that time I removed some hypertrophied mucous membrane from both nostrils and/

and sent him to Dr Walker Downie for cauterization. This was done and for a year patient had complete relief. Lately, however, asthma has been bad, especially in wet weather. Sneezing also has been troublesome, especially when he washes his face or is exposed to dust or smell of sulphur. Sunday is nearly always his worst day. The only difference between his week day life and his Sunday life is, that he has no work after one p.m. on Saturday till Monday morning. His meals are the same on Sundays as on other days. He thinks that when he has a run on his bicycle on a Sunday morning he is less troubled with asthma; and on the rare occasions when he has to work on Sunday he has no asthma.

The mucous membrane of right middle turbinal and of posterior end of right of right inferior turbinal is hypertrophied. There is a thick soft patch on left side of septum. I cauterised face of right middle turbinal. To spray nostrils with Carbol. Camphor Parolein.

26th. Had a bad attack of asthma the two days following cauterization. To take ~~tegr.~~ Calomel every Saturday night and if this does not ward off an attack, to take Suprarenal Tabloids $\frac{gr}{v}$ thrice daily.

18.2.99. No asthma since last note except a very slight attack on Sunday a week ago. Sneezing fits quite gone.

Note that patient who had to travel 12 miles to see me, did not/

not have nasal treatment finished. He writes 7th May 1900 to say that he has continued a great deal better since operation, incomplete as it was. He sometimes has a little "stuffy feeling" in the mornings, from which he readily gets relief by smoking an "asthma cigarette." For the last two years patient has not had asthma in January or February, he is most apt to have it in close warm weather; he gets relief when rain falls. Never any sneezing fits which used to be caused by washing his face and which often ended in asthma. An attack used to last a whole or several days: now it only occurs for a few minutes in the morning.

12.5.1900. Patient came by request to show himself. Both middle turbinals are somewhat hypertrophied so as to impinge on septum, especially the right which is inflamed and threatening to become polypoid again. There is a septal ~~Kapur~~ spur on left side opposite inferior turbinal.

IX. Male, aged 30, Engineer, Uddingston.

Asthma since childhood, petechia² and somnolence during attacks which are mostly on Mondays. Marked hypertrophy and irritability of nasal mucous membrane. Nasal and general treatment, especially Cod Oil. Cure.

10.11.98. Patient came to me a month ago on account of asthma which has troubled him more or less since boyhood.

Last/

Last year he never was a fortnight free of asthma till the summer. This year he has already had one attack, precipitated by "biliousness." During a bad attack he gets "black in the face", dark red "blood spots" (petechia^e) come out, he suffers from headache and becomes somnolent. The attacks nearly always occur on Monday mornings. He has been using *Pot. Iod*

Hyoscyam Mixture and a spray of Sod. Nitrit, *gr 40* Pyridine *m 20*, *Antipyrin gr 10*, *Vin Ipec. ʒi*, *Lobel. ʒi*, *Bellad. ʒi*, *Chamom. ana m. ʒ*
Aq ad ʒi ʒss
 2 gr. Calomel on Saturday nights. Today cauterised hypertrophied mucous membrane of right inferior turbinal.

29.3.98. Feeling as if he were going to have asthma. Mucous Membrane of larynx is uniformly injected: the vocal cords also are pink. Tongue also furred white. Painted larynx and nares with Cocain^t, Resorcin in Hazeline and again in a few minutes the larynx with weak glycerine of Tannin.

8.3.98. Has been more free of asthma this year than ever before, though he has a tendency to it when he has "cold in the head". Two weeks ago on returning from coast he did not feel well and had a violent attack of asthma two days later with petechia^e on the face. The attack lasted three days. Yesterday morning (Sunday) another attack, from which he is still suffering, came on: but he was not sick as he usually is. He is somewhat dusky: the inspiratory difficulty is greater than the expiratory. The whole mucous membrane of/

of larynx is injected, the cords pink and with sticky mucus between them. The mucous membrane of trachea is also injected: that of the nose is very much so especially on the right side where the inferior turbinal is so swollen as to touch the septum. The left inferior turbinal is not swollen but there is hypertrophy of middle turbinal. Some enlarged veins on both sides of septum, which is irritable, a touch causing sneezing even after application of weak Cocain solution. 15th. Removed anterior end of right inferior turbinal and applied electro cautery.

15th. (Monday) Asthma came on today at 4 a.m., patient omitted to take the calomel which he was directed to take on Saturdays. The right inferior turbinal is, of course, injected as result of operation, but the nasal mucous membrane generally ^{is not nearly} so injected as a week ago. The vocal cords are injected and there is sticky mucus between them.

Patient thinks smells have much to do with causing his attacks He says he has a friend 4 miles off whose attacks often synchronise with his own. He attributes this to the weather but it is probably because of the "week end" nature of the attacks in both cases.

24th. Removed excess membrane from left inferior turbinal.

24.10.98. Patient had a "cold" and a bad attack of asthma a fortnight ago. Feels very weak. Removed a polypoid part of/

of left middle turbinal and cauterised base.

11.12.98. No asthma since last note. Has not felt better in his life; this he attributes to his being able to take a certain preparation of Cod Oil which he never could previously take.

17th. Removed posterior end of right inferior turbinal which was polypoid. It is noted that even a 15% solution of Cocain does not shrink the inferior turbinal much and only for a short time, reapplying the Cocain only makes the turbinal swell up.

24th. Cauterised posterior end right lower turbinal and also face of right middle turbinal.

25.3.99. Viscid purulent secretion from pharynx for a week. It is seen to coat the pharynx. To gargle with borax and Sod. Bicart and use parolein spray to nose. Says he had similar trouble with pharynx some years ago. No asthma.

25.5.99. Throat very well. Only one attack of asthma since last note. Is very well and does not follow treatment regularly. Advised to take Blue Pill every Saturday night and a tumblerful of hot water every night.

May 1900. No asthma since last note. ~~He has been very well since~~

X. Female, aged 37, Shotts.

Laryngismus stridulus in childhood; frequent nasal catarrh. "Irritable" nose and sneezing fits.

Asthma/

Asthma for seven years. Urticaria. Uric Acid.
 Hypertrophy of right middle and inferior turbinals
 septal spur. Nasal treatment declined. Relief
 during attacks from Mist. Sod Iod, Sod Salicyl.

12.3.1900. In childhood had attacks of Laryngismus
 Stridulus and night terrors. Fifteen years ago on first
 coming to Hamilton she began taking frequent colds in the
 head to which she has been less subject since going to Shotts
 three years ago. She has had "an irritable nose" since she
 was 19, when she noticed that while sewing flannel or work-
 ing with wool her nose became extremely irritable, there
 was intense tickling in the nostrils with occasional sneezing
 fits. The first definite attack of asthma occurred shortly
 after the birth of her first child 7 years ago. Since that
 she has had a few severe attacks, not oftener than once a year,
 but very frequently in the mornings she has slight breathless
 and wheezing attacks which pass off during the day. She
 has a very itchy skin, often Urticaria. Urine often shows
 a thick deposit of uric acid.

Nose. Left Nostril may be regarded as normal. In the right
 there is a large septal spur or ridge pressing on the inferior
 turbinal; the middle turbinal also is in contact with the
 septum. Both turbinals are slightly hypertrophied. Patient
 says there is a slight tendency for this nostril to drip a
 watery/

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watery secretion when she holds down her head. The tissues of the pharynx are thickened and congested: the uvula has been amputated; the under surface of the epiglottis is decidedly red.

14.5.1900. Patient declines nasal treatment in meantime. A vegetable diet is recommended, but patient finds this difficult to procure. Mist Pot. Iod et Ars gives some relief from asthma, as also a mixture of Pot Iod ~~et Ars~~ *et Ammonium Brom.* What gives most prompt and decided relief is a mixture of Sod Salicylat and Sod Iod.

XI. Male, aged 20, Bricklayer, Kirkmuirhill.

Asthma from childhood; nasal catarrh. Polypoid condition of both middle turbinals. Nasal and other treatment. Great improvement.

21.9.95. Asthma since childhood during the summer months till 4 years ago, since which it has occurred only during August and September except that three years ago it did not come at all. It has never occurred during winter. He has great difficulty in breathing especially in inspiration, wheezes much but does not spit much. Nearly every winter he has "cold in the head" but no breathlessness, no wheezing. Digestion good except after an attack of asthma begins.

Chest has very little of the barrel shape, cardiac dulness slightly encroached on. Heart normal, slight cooing rale/

rare all over the chest. (Patient had had asthma this morning)
 White fur on tongue. Polypoid condition of anterior end of
 both middle turbinals, Mist Pot Iod et Ars.
 25th. Asthma gone. There is a dental abscess.

2.11.95 Since last note polypi were removed and Chromic
 acid applied and there has been no asthma for a month. About
 that time there was a slight attack brought on by "cold in
 the head". The addition of Tr ~~th~~lyoscyan and Vin. Ipec. to
 above mixture was a great help.

XII. Male, aged 49, Blacksmith, Ferniegair.

Asthma for two years and a half. Large number of
 nasal polypi and thickening of nasal mucous mem-
 brane. Nasal treatment. Cure, except that a "cold
 in the head" caused a slight attack of asthma.

28.5.99. Patient came to me in March on account of asthma.
 He never had asthma till December 1896; the attacks then
 remained very persistent till June last. He did not know
 he had nasal polypi till told by a doctor in September 1897.
 He immediately went to Glasgow Bar Hospital where a number
 of large polypi were removed from both nostrils from time to
 time till December 1897. The asthma continued however, six
 months longer till good weather set in in June. Asthma then
 ceased till February last, since which it has been very
 persistent. At first the asthmatic attacks were very bad
 and usually came on at midnight, waking him from sleep. For
 months/

months, even after the operations at the Glasgow Ear Hospital ceased he could not sleep except sitting in a chair. He went to work at 6 a.m., often suffering from severe asthma and often having to stop on the way: but on his return for breakfast at 9 a.m., the asthma would be going off. He seldom has an attack during the day.

He takes tea four times a day, butcher meat seldom except at breakfast. Potatoes, ~~and~~ porridge, ~~and~~ buttermilk, or soup are apt to cause asthma accompanied by belching up of bitter liquid, after which he may get relief from the asthma. Water is seldom taken. Constipation used to be the rule till the asthma started, but for last two years bowels have been regular. Until he had asthma he never had a cough. Since February the attacks have been much milder except twice, once being after a meal of peasebrose.

When patient ^{first} ~~once~~ came to me two months ago I found a large number of mucous polypi in both nostrils and have since removed on different occasions about thirty of them. Yesterday I also divided a synechia which had formed high up in the left nostril between middle turbinal and septum probably as the result of the operations at Ear Hospital.

30th. Bad attacks of asthma the last two nights, no sleep. Heart and lungs seem normal, perhaps slight diminution of R.M. but no marked emphysema.

17.6.99/

17.6.99. A fortnight ago had a very bad attack of asthma after a trip to the coast. Four doses of Pot Iod and Arsenic Mixture did no good, so he took, as he often does, half a teaspoonful of sulphur and got relief.

29.7.99. Asthmatic attacks occur at night and especially at 2 a.m., The whole of the mucous membrane of right nostril, now that polypi have been removed, can be seen to be thickened and diseased. To day I removed the anterior end of right middle turbinal which is tending to "polypoid" "degeneration", also a thickened ridge below orifice of antrum. The middle turbinal is tending to become "cleft" Base of tongue and larynx examined today show no special abnormality.

5.8.99. Three polypi very high up in right nostril removed today.

12.8.99. Cauterised face of right middle turbinal.

24.8.99. Patient's digestion has not been good lately and he has been losing weight. Asthma has been bad and for the first time since I started to treat him he has been off work.

Today I removed hypertrophied part of left middle turbinal and applied cautery to hypertrophy in septum.

Note. Even before I started to treat him patient's asthma has usually been worst on Monday nights and Tuesday mornings. This has no doubt been aggravated by the operations which nearly always were done on Saturdays. Suprarenal Tabloids taken/

taken internally greatly upset patient* and aggravated asthma, as did draughts of water at bedtime.

April 1900. No asthma since last note (8 months).

27.5.1900. Patient took a bad cold in the head a fortnight ago and had a slight attack of asthma.

XIII. Male, aged 54, Painter, Hamilton.

Nasal catarrh: sneezing fits; asthma especially in "hay season" for 16 years. Polypi in nose and hypertrophy of nasal mucous membrane. Nasal treatment, cure of asthma: great improvement of sneezing fits. Son affected with paroxysmal sneezing, no asthma.

14.10.97. Complains of constant cold in head and frequent sneezing followed by asthma. Attacks of what was called, and probably was, Hay Fever began 16 years ago. Whenever he went into the country in summer or even when he was approached by a farmer in "hay time", he would have violent sneezing and coryza, followed by asthma. The worst attack he ever had was at a farm. At first the attacks occurred only in summer but now they are apt to occur in winter. A hearty laugh will cause asthma lasting for hours. There are nasal polypi, mucous membrane thickened red and spongy at various points. Lungs are somewhat emphysematous, the cardiac dulness being obliterated, the ribs move very little on respiration/

* See p 50

respiration, which is mostly accomplished by the diaphragm.

Have removed several polypi from right nostril.

11.11.97. Cauterised mucous membrane of right side of septum which was spongy.

27.9.98. Small polypi having recurred high up in right nostril have been removed. They are too high up to allow of safe use of cautery to their bases.

1.10.98. Cautery punctures in right side of septum.

25th. Removed several polypi from left middle turbinal four days ago and today cauterised turbinal.

15.11.98. Cautery again to left middle turbinal. Patient has had no asthma since I operated on his nose a year ago. The intense tickling which used to be present at point of nose is gone: the attacks of sneezing still occur when he is exposed to dust but are much less frequent.

15.12.98. Cautery punctures in left side of septum.

20.1.99. No asthma except when he laughs heartily then he will wheeze for a whole night. A small polypus is again budding high up in left nostril. Removed this. Sneezing attacks most frequent on Sundays.

8.2.99. Cautery puncture in hypertrophied area on right side of septum, tickling of which causes violent sneezing.

Urine collected on Saturday evening gave no deposit of Urates, that of Sunday and Monday evenings did.

22.2.99. Nose now looks very well.

15.5.1900. /

15.5.1900. No more asthma: no sneezing for many months.
 Note. Patient's son aged 30, came to me 28.8.97, complaining
 of Paroxysmal Sneezing and Coryza of six years duration. No
 asthma. Hypertrophy of nasal mucous membrane. Cured by
 light applications of cautery and nasal spray of Menthol-
 Camphor in Parolein.

XIV. Female, aged 40, Hamilton.

Sneezing fits, Urticaria and nasal obstruction for
 five years. Slight asthma during last year.

Nasal treatment not carried out, no improvement.

15.5.1900. Patient had no nasal trouble when she came to Hamilton 9
 years ago. During the last five years she has been much
 troubled with exhausting sneezing fits especially on washing
 her face in the mornings, with profuse watery discharge dur-
 ing these fits, and with nasal obstruction. The turbinals,
 especially the two inferiors, are much hypertrophied and
 irritable: the mucous membrane of the septum also is unusually
 irritable. I advised treatment with the electro cautery and
 within the last year have applied it twice to one side of sep-
 tum and once to ^{left} inferior turbinal left. I had an opportunity
 of showing the case to Dr Walker Downie who advised continu-
 ing this line of treatment, but for various reasons and
 especially because of the great irritability of nasal mucous
 membrane/

membrane, and of the feeling of exhaustion consequent on interfering with it. The treatment has not been carried further, and no real ~~relief~~ benefit seem to have as yet occurred. Within the last year patient has for the first time in her life had three or four very slight attacks of genuine asthma. For the last 5 or 6 years patient has been subject to chronic articularia. She has during the past ten years suffered great loss of blood from a ^{uterine} uterine fibroid.

XV. Male, aged 5½, Hamilton.

Bronchitis becoming asthmatic, adenoids and hypertrophied turbinals treated with some improvement.

Cessation of asthma during whooping-cough.

Note made 25.5.1900.

Attacks of bronchitis began when patient was about a year old and to be asthmatic in type when he was 2½. These attacks came on mostly at night and about a year ago occurred nearly every night. Dr Walker Downie removed adenoids and the hypertrophied anterior ends of the inferior turbinals last September. After this the asthma ceased for a week. These attacks occurred regularly once a week or once a fortnight instead of daily, and were less severe and less prolonged. There has been a decided improvement in the general health as there always is after the removal of adenoids and doubtless this has helped the asthma. Patient can/

can now sleep with his mouth shut and still makes some but very little noise in breathing. This points still to some nasal obstruction, which is not to be wondered at, as the complete removal of hypertrophied tissue in the narrow noses of adenoid children is almost an impossibility. Two months ago, the patient took in a moderate way another spasmodic disease, whooping cough, and has never since had a spasm of asthma. The whooping cough still persists.

XVI. Male, aged 21, Lawyer's clerk, Greenock.

Bronchitic asthma from childhood; attacks worst on Sundays. Pronounced nasal disease, polypi, oedematous and hypertrophied mucous membrane: spur, Great improvement of asthma by nasal treatment which is not quite completed.

15.10.99. Patient had bronchitis when two years old: has been subject to asthma as long as he can remember. The attacks are always worse at night and are worst on Sundays. He knows of nothing that brings them on except that he never has cold in the head without having asthma. Is not troubled with indigestion or constipation; a soft boiled egg sickens him but does not produce asthma.

Five years ago polypi were removed from both nostrils by a Glasgow Specialist. A fortnight ago he was seen by a doctor who examined his nose and told him it contained no polypi. Three days later he came to me and I found polypi in both nostrils/

nostrilsspringing from middle turbinals, the under surface of which, as also the inferior turbinal are thickened and oedematous. I removed the polypi from right nostril and two days later cauterised the middle turbinal; yesterday removed the polypoid and hypertrophied parts of left middle turbinal. Just before he came to me he had been a fortnight off work owing to asthma, but has not since had asthma and has been able to resume work at Greenock. To take occasionally, Mist Iod et Ars with Pot Brom and *In Hyoscy* added.

21.10.99. Cauterised left middle turbinal.

16.12.99. Removed a large ridge from right side of septum a week ago and today applied Chromic acid to the granular parts of wound.

15.1.1900. Removed hypertrophied parts of left inferior turbinal. Plugged the nostril.

16th. Was called to day to see patient who had developed a pretty severe attack of asthma result of the operation and patient's taking a long walk the same night against orders. Great difficulty of inspiration and expiration chiefly the latter. Interior of left nostril much congested and blocked by secretion. Glottis widely open and hardly moving with respiration. Chest hyper-resonant full of snoring rales, cardiac dulness small, liver dulness reduced to an inch and a half in nipple line. Applied Cocain and ~~Suprarenal~~

Suprarenal solutions to interior of nostril, but chiefly of left. Within 5 minutes the laboured breathing ceased, the snoring rales mostly disappeared, leaving only soft moist rales at various parts of the chest but mostly at right axillary base. Cardiac dulness is greater, liver dulness measures three inches: glottis still dilated but less so and seems more mobile with respiration. The airway in both nostrils is now quite free though the left is still somewhat congested.

31.3.1900. No asthma since I began to operate on him except the attack mentioned in last note. He occasionally has slight "tightness" of breathing on waking on Monday mornings, but he would not call it asthma. He says he has had less asthma since he came to me than he has ever had. Urine is said to be often thick and red (Urates). A month ago removed anterior end of left middle turbinal and to-day a large part of soft tissues of right inferior turbinal.

14.4.1900. Have twice cauterised the wounds of last note. Middle turbinals though not now covered by hypertrophied mucous membrane still impinge on septum: and there is now for the first time visible, being no longer screened by hypertrophy in front of it, large polypoid growth of posterior end of right inferior turbinal.

5.5.1900. Two days ago (Thursday) patient was exposed to

a/

a violent dust storm, and had his second attack of asthma since treatment began. It lasted only an hour. He had been feeling right nostril rather sore and irritable for some days previous: so to-day I removed posterior end of inferior turbinal and also the whole of its under surface and part of its upper surface, all much hypertrophied.

14th. Asthma for nearly a week- probably the result of operation. Patient did not ^{think} think of letting me know till this morning and did not take his medicine. Right nostril is of course inflamed. Application of Cocain and Suprarenal promptly relieved asthma, pulse fell from 108 to 84. Temperature is normal.

The left inferior turbinal looked very full to-day and probably turbinectomy of right middle turbinal will have to be done.

26th. No more asthma.

XVII. Male, aged 17, Bothwell.

Bronchitic asthma possibly with "peptic" element for 9 years. Nasal catarrh, occlusion of right nostril by deviated septum. Treatment of septum: cure of asthma.

17.5.1900. Patient was brought to me last October on account of asthma. His disease began when he was 8 years old. An attack always started with a "cold in the head" and incessant/

incessant coughing. Bronchitis was frequent during, but generally after an attack and seemed to develop quickly. It is probable that patient over-ate himself. He was taken to ^a Glasgow Doctor who ordered him to be put on skimmed milk. His attacks on this treatment were reduced in severity, sometimes lasting a week but usually only two or three days and were much less frequently accompanied by bronchitis. When ten years old he went one summer for two months to Musselburgh. He had asthma almost the whole time he was there but no bronchitis. This was also the case during the whole of the five months he was at ^{Menton} Montrose. Sneezing, although it occurred, was not nearly so common at the beginning of an attack as incessant coughing. Sulphur fumigation once brought on a severe attack of asthma.

Patient on being brought to me was evidently a mouth-breather. The nasal obstruction I found to be due to a much deviated septum which quite occluded the right nostril. This I asked Dr Walker Downie to remove at the end of October. Patient has not had asthma since.

XVIII. Female, aged 50, Glasgow.

Nasal obstruction and catarrh, slight asthma. Hypertrophy of middle turbinals, polypus, spur. Nasal treatment. Cure of asthma.

14.11.98. Has had "stuffy feeling" in nose for many years, takes "cold" in nose readily, and has a tendency to asthma (wheezing and difficulty in breathing) especially on the evenings/

ings of days on which she **has had** much exertion. Both Middle turbinals are hypertrophied with polypoid growth on left, opposite which, also, there is a spur.

5.1.99. At different times the hypertrophied tissues have been removed and electro-cautery applied. The parts are ^{nearly} near healed.

17.5.1900. Reports herself very well, "cannot say she has noticed any recurrence of asthmatic symptoms or at least they have been very slight."

XIX. Female, aged 39, Ferniegair.

Dyspnoea (? Asthma) for 2 years. Polypus in each nostril, large in one. Permanent cure of dyspnoea after removal of polypi: peripheral neuritis.

20.1.99. Patient came to me 14th June last complaining of difficulty of breathing through the nose. This had been steadily getting worse during the last two years and for seven months prior to her consulting me, she had not got one complete night's sleep. The attacks of dyspnoea were always worse at night and for six weeks before she came to me, she would regularly start up during the night, gasping for breath.

She had a blocked up feeling in her nose and while swallowing felt as if she would choke. She never called her trouble "asthma" but the attacks of dyspnoea coming on periodically/

periodically during sleep as they did, would probably be called nothing else by most medical men, though they have a resemblance to the distressed breathing of adenoids. The case is interesting at least from the point of view of asthma; for it was seen by several medical men who did not seem to realise the degree of her distress or the fact of her nasal disease. She went to the Glasgow Western Infirmary where she says she was told she was suffering from depressed spirits. She then came to me and on 14th June last, I removed from the right nostril a single large polypus and a fortnight later a smaller one from the left. The attacks of dispnoea which used to occur daily never recurred after the removal of the larger growth, and she slept that night as she had not slept for years. This of course was aided by a drachm of Paraldehyde which she took for a night or two. There is no emphysema. Heart normal. Another interesting feature in the case has just occurred. Three weeks ago, while taking down a washing from the rope on a cold frosty evening she felt her right hand get cold, sore and feeble. At first she took little notice as the numbness feebleness and pain were slight, but on the 15th (five days ago) these got much worse. Since that the attacks of pain and numbness have come in spasms interrupting her sleep. First the hand gets numb and after a few minutes it becomes very painful and cold and this condition spreads up the limb/

limb as far as the middle of the upper arm. She cannot tell what colour the limb becomes. The hand afterwards feels as if swollen and becomed unable to grasp tightly. On examination to-day, the limb presents nothing especial in extremal appearance; the skin feels colder than that on the left arm; the grip is feeble. There is pain on pressure over the ulnar and radial nerves in the forearm, also over ulnar and musculo-spiral in the upper arm. There is some analgesia on the tips of the fingers, but not of the hand; some anaesthesia on the front and back of the fingers especially toward their tips. She dare not touch anything cold with the limb and tonight, while it was exposed for examination, an attack of numbness, cold and pain came on, which was much relieved by *effleurage* effleuage. To take Sod Salic, Sod Iod.

24th. The surface of right arm is paler than that of left: the muscles softer and feebler and responding more readily to galvanism than those of the left.

15.5.1900. No recurrence of Dyspnoea. The neuritis of right arm improved more quickly after last note. But patient says that she has occasional attacks of intense tingling pain and numbness in left hand, especially thumb. These attacks occur during the night after a lot of washing and wake her up from sleep. She feels as if the hand would burst. She has/

has once or twice had a slight attack in right hand, but not recently.

XX. Male aged 39, Pipelayer, Hamilton.

Influenza, bronchitis, severe asthma. Paroxysmal sneezing especially in hay season. Asthma apt to be worse Sunday nights and Monday mornings. Mucous membrane on septum hypertrophied and irritable; spur on both sides of septum; left nostril very narrow. Hypertrophy of lingual tonsil. Electro cautery to septum and lingual tonsil: medicinal treatment. Great improvement.

2.9.99. Eight years ago took influenza and his first attack of bronchitis; has had bronchitis every winter but one since he came to Hamilton five years ago. Has frequent fits of sneezing, especially in hay season, when he will sometimes sneeze as long as ten hours. At these times asthma is generally very bad, especially if he also happens to have slight bronchitis. Until a fortnight ago patient has not had a very bad attack of asthma for 3 years, though usually in the morning he would be "so hard up for breath" that he could not put on his own boots, but had to get his wife to do it; he would then go out and in twenty minutes would be all right. A fortnight ago, however, he had an attack which lasted four days and which was so severe that he thought he was going to die of suffocation. Dr Donald Munro

(my locum tenens) who

was called on the second day, diagnosed asthmatic bronchitis and gave a good deal of relief by administering Amyl Nitrite followed by Pot Brom and Pot Iod. For four nights patient sat in a couch, as lying down made him worse. He notices that abdomen seems to swell during attacks. Has eaten almost no potatoes for 3 weeks. Takes tea rarely, coffee frequently; but if he takes coffee on Sunday nights his asthma is usually worse next morning. If he takes no food after dinner on Sundays he has little or no asthma on Monday mornings. The attacks have been most apt to occur on Sunday nights or Monday mornings.

Chest. Some cooing rales in both lungs, especially near *the* Bronchi. Cardiac dulness is not much reduced.

Nose. Mucous membrane in both nostrils rather red and irritable but not much hypertrophied except on septum opposite middle turbinals where it touches them, is peculiarly irritable, and causes sneezing when tickled. Larynx slightly injected.

Lingual tonsil slightly hypertrophied especially on right side where it is apt to touch the tip of epiglottis which is also slightly more prominent on right than *left* side..

15.5.99. (Friday) No asthma this week except on Tuesday night (12th to 13th) again tonight after a hearty dinner. No urine has been passed from noon till now (7.30 p.m.)

Asthma/

asthma tonight much relieved by painting interior of nose with Cocain followed by Suprarenal Solution..

16th. Bad asthma last night. Interior of nose rather red some sticky secretion plugging upper part of left nostril: septum irritable. Abundant cooing rales in chest chiefly on expiration; dyspnoea pretty bad. Laryngoscope shows glottis widely open: vocal cords not moving at all during respiration. Much, but not complete, relief by painting interior of nose with 15% Cocain solution followed by Suprarenal solution. Has been taking Ammon. Sod and Ammon Chlor all week, but stopped it yesterday. 1.30 p.m., asthma tending to return. 6 p.m., took 2 grains calomel this forenoon. Asthma very bad all afternoon. Inhalation of Amyl Nitrite has given prompt relief. Cocain followed by Suprarenal again applied to nose. This helps a little but it is to be noted that the suprarenal has not blanched the mucous membrane. 8 p.m., Asthma still present but not nearly so bad as at 6 p.m., Inhaling weak Amyl Nitrite vapour has given relief: Cocain applied to base of tongue seemed to give more relief than when applied to nares.

18th Some asthma last two nights relieved by de Joy's cigarettes which he says give him more relief than anything else. Greatly relieved this morning from slight asthma by application of 15% Cocain solution to base of tongue and epiglottis!

epiglottis.

28th. Electro-cautery puncture has been done since first note to the irritable spots on nasal septum; the hypertrophied part of lingual tonsil (which was touching epiglottis) seared with cautery a week ago. Tonight patient is hoarse, vocal cords and parts of larynx above it de ply injected. Asthma much less frequent and less severe. Has been taking Pot. Brom and Pot Iod which he finds relieve him more than Arsenic and Pot Iod.

17.5.1900. Patient regards himself as practically cured. For two years before treatment he would have slight asthma every morning and at least once a week, sometimes twice or thrice in the week, would beunable to go out to work before breakfast. He was often off work for weeks at a time. Since treatment which,owing to patient's neglect was not completed so far as nose is concerned, he has been twice off work for a day, the last time being nearly three months ago. He has had altogether three attacks since his nose was treated; on one occasion, a Sunday, he ascribed it to eating pastry which always hurts him; another attack was on a Monday, the third on a Wednesday. He never has breathless attacks in the mornings. There is a spur on both sides of septum and left nostril is very narrow. The right half of lingual tonsil is still to large: the mucous membrane of larynx all rather red. He is a heavy smoker.

XXI. Female, aged 22, Tailoress, Hamilton.

Asthma/

Asthma for two years generally on Sundays.

Extremities dusky and cold. Acute attack of
Bronchitic asthma with muco-sanguineous spit.

27.3.1900. I was summoned urgently to this case this evening. The illness began yesterday (Monday) with slight difficulty of breathing, gradually increasing, so that she had to leave work at 5 p.m., She slept none last night. When I saw her at 6 p.m. to night, she was sitting up in bed breathing with extreme difficulty and feeling so weak that she could hardly move. Face was of a dusky pallor, hands and feet dusky pulse not of high tension but soft and feeble, heart rapid tumultuous and irregular in action. Occasional expectoration of pink froth (mucus intimately mixed with blood) small, moist, indistinct rales in lungs, R.M. very weak. No cooing rales. Mustard bran poultices greatly relieved: turpentine inhalations were also used given every two hours. Note. Menstruation ceased on Saturday and she was not well that day and Sunday, having a bad headache.

28th. Patient slept well last night and is much easier this morning. The spit is now white mucus, free of blood. Breathing is still rather rapid face and hands are somewhat dusky; pulse 110. The heart dulness is slightly diminished: heart sounds normal. Abundant moist and cooing rales all over the lungs especially at the bases where they are more moist/

moist. The impression I got yesterday was that the condition was one rather ^{of} acute capillary bronchitis than of asthma, but it was indistinguishable from the paroxysmal bronchitis of children which Gee says is asthma. Patient has been subject to asthma for the last two years and would have about six attacks last summer, generally on Sundays. She says hands are always rather dusky: has been much troubled with cold feet and chilblains all winter. No urticaria. To-day I prescribed a saline purge to be followed by Ammon. Iod Ammon Chlor and Liq Arsen.

29th. There was again difficulty of breathing yesterday afternoon but it is gone this morning. Pulse still 108. Temperature has not been above 100 degrees throughout.

9.4.1900. Both middle turbinals are hypertrophied, from the right are growing several small polypi. Removed these to-day. 14th Applied cautery to right middle turbinal.

3.5.1900 Removed anterior end of left middle turbinal.

14th No asthma since attack mentioned.

XXIII. Female, aged 50, Hamilton.

Bronchitic asthma for two years duration. Malnutrition, dyspepsia, disease of upper respiratory tract. No improvement.

Note made 16.5.1900. Patient is a thin sallow woman who eats sparingly and is far too energetic. There is marked chloasma/

chloasma and a general darkness of skin which at first sight makes one think of Addison's disease. Dressed in out-door garb, she weighs only $6\frac{1}{2}$ stone. She is much too energetic however, and the symptoms have been too long in play to admit of Addison's disease. She first had asthma two years ago. It seemed to start after a pretty bad attack of influenza and bronchitis. Sometime after this, she took her first attack of asthma after running. She could not speak. Attacks than began to come whenever she attempted to walk and the breathlessness was soon accompanied by wheezing. Then the attacks became nocturnal and latterly they have also been prone to occur after food especially after dinner. There have been a good deal of flatulence and indigestion and also deterioration of the general health. Patient lives in a lowlying, low-roofed house set on damp ill drained soil. There is chronic thickening of the mucous membrane of the pharynx, hypertrophy of the lingual and faucial tonsils, slight thickening and redness of the right middle turbinal. The left nostril is much too narrow owing to deviation of the septum and a septal spur or ridge. The left middle turbinal impinges on the septum. The mucous membrane of the interior of the larynx is rather injected. The heart is sound. I have twice cauterised the lingual tonsil, but have not attempted much in the way of treatment to the upper part of the respiratory tract because mal-nutrition/

mal-nutrition is evidently the chief factor in patient's condition. Owing to her poor circumstances this is difficult to remedy. Treatment directed to the stomach does most good. She gets ^{temporary} relief from inhaling asthma powder.

XXIII. Female, aged 39, Hamilton.

Attacks of bronchitis for 24 years, of asthma for seven years. No nasal disease. Improved, without treatment, after pneumonia.

17.5.1900. First took bronchitis when 15, since that had it every winter but one. The last few winters bronchitis has been much less severe: severe pneumonia two years ago. Her first severe attack of asthma occurred 7 years ago, though she may have had severe attacks ~~since~~ before that. Her last bad attack was two years ago. During that period of five years she would have two or three attacks yearly, lasting two or three days at a time. They were most apt to occur when she was worried: indigestion sometimes seemed to cause an attack. She thinks they were apt to occur at a menstrual period. She has had nine children, was free of asthma while pregnant, except at the beginning of the last two pregnancies. Asthma is usually accompanied by bronchitis, the bronchitis preceding the asthma: she has often had bronchitis without asthma; rarely she has had asthma without bronchitis, usually as the result of excitement. No real attack of asthma these last two years since she had pneumonia. Urine is always loaded with urates during attacks

Nose/

Nose and throat show nothing specially abnormal. Painting nares with Cocain solution and inhalation of Amyl Nitrite used to give some, but not marked relief. I have not been called to treat patient for asthma for several years and have had no opportunity of trying Suprarenal. She has improved without treatment *(since Pneumonia, two years ago)*

XXIV. A boy, son of case XXIII aged 14.

Spasmodic croup, bronchitis, asthma, usually on Mondays. Nasal hypertrophy.

One of preceding patient's children, a son aged 14, had spasmodic croup when 6, bronchitis when 7, and asthma when 8. Asthmatic attacks are pretty frequent, come on in the mornings and during the last two years occur nearly always on Monday mornings, lasting two days and preventing him going to his work. During these last two years he has rested more and eaten more on the Sundays than during the week. On Saturdays he plays football.

There is hypertrophy of both middle turbinals tending to become polypoid: the left tonsil is hypertrophied and the pharynx is studded with the little granules suggestive of adenoids higher up. Patient does not snore at night, however, and can breathe fairly well through his nose. I have not been called to treat his asthma.

XXV. Female, aged 56, Hamilton.

Mother of case XXIII. Bronchitis, asthma, no nasal disease. "Practical cure" of asthma by medicinal/

medicinal treatment.

The grandmother of the boy mentioned in preceding note and Mother of patient XXIII, suffered from severe asthma for 12 years. She had for many years been subject to chronic bronchitis, but the asthma did not ^{develop} develop till she came to Hamilton. Attacks were accompanied by much wheezing and finished with a good deal of expectoration. She came to me for treatment 9 years ago and from that time till her death 4 years ago from cardiac disease she considered herself cured. It is plain, however, that while she never had another real "attack" of asthma, she was afraid of one coming on and very frequently took a dose of her medicine at bedtime. The treatment was Mist Iod et Ars which she always kept in the house. I examined both nose and throat but found no special abnormality.