these cases fails to react quickly to renal lavage then I am accustomed to take a pyelo-radiogram to see if I can detect narrowing of the ureter, fibrosis of the renal pelvis, or even an abscess in a portion of the kidney.

Ureterograms.

In most cases it is possible to show up the anatomy of the ureter by this method and exclude phleboliths, glands, etc. In a certain number of cases of ureteric calculus the calculus does not appear on the x-ray plate, especially if the stone is lying in front of bone. A catheter is passed and is found to stop four to six inches up the ureter. A film is then placed beneath the pelvic portion of the ureter, sodium bromide injected, and an x-ray picture taken. This may then show up a stone impacted in the ureter, the ureter dilated below the stone, the catheter running up to the stone, and very often some of the bromide will run past the stone and show up the dilated ureter above the stone and a stricture at the site of the stone. Fig. 18 illustrates this particularly well.

Fig. 19 provides another interesting example. The patient came to me suffering from pyelitis of pregnancy which persisted after the child was born. I catheterized the right ureter and filled it up with colloid silver. It shows very well the dilated condition of the whole ureter. As I have pointed out before,³ the ureter in pregnant women undergoes dilatation due to loss of tone in the muscle of the ureter rather than to pressure of the uterus. This tone returns very slowly after pregnancy. This is the first time I have been able to demonstrate this fact by the ureterogram in the living subject.

Cystograms.

From time to time it is of value to take x-ray pictures of the bladder when filled with opaque solutions. I find that sodium bromide, even in strengths of 10 or 15 per cent., is apt to be very irritating to the bladder and cause great pain, especially if the bladder is inflamed. I therefore prefer to employ 10 per cent. colloid silver. This does not cause any pain in the bladder, and shows up quite well enough for practical purposes. Pictures should be taken in the antero-posterior and also in the lateral positions. The method is useful chiefly for showing up the extent of bladder pouches. One can see the openings of the mouths of the pouches with the cystoscope, but very often one can have no idea as to how large the pouches are until one takes a cystogram (Fig. 20).

Another use for this method is to study the outline of the bladder and prostatic cavity after prostatectomy, and it is also possible to determine by this method whether the ureters are allowing fluid to run from the bladder into the kidney in cases of back pressure. The bladder is filled with colloid silver, and the patient is placed with the head down. In some cases the colloid silver is found to run back from the bladder into one or both kidneys in cases of back pressure, also in some cases of tuberculosis and other infections of the kidney with dilated ureters.

Finally, Fig. 21 shows a case of recto-vesical fistula in a man following on a fractured pelvis. I was able to cut down, excise the fistula, and cut off the rectum from emptying its contents into the bladder.

Conclusion.

Pyelography has established its position as a valuable method of clinical research in urology. It is indicated particularly in cases of severe abdominal pain of doubtful origin, to determine also the nature of abdominal tumours, and to complete the diagnosis in many cases of haematuria and pyuria.

REFERENCES.

¹ Pyelo-radiography: A Clinical Study. *Proc. Roy. Soc. Med.*, Surgical Section, 1913, vol. vii, pp. 16-40. ² Carelli: Royal Society of Medicine, December 8th, 1921. ³ *Common Infections of the Kidney*, p. 97.

THE Fifth Congress of French-speaking Paediatrists will be held in Paris on July 10th, under the presidency of Professor Weill of Lyons, when the following subjects will be discussed: (1) The place of pituitary and pineal glands in infantile dystrophy, introduced by Dr. P. Lereboullet. (2) Infantile diabetes mellitus, introduced by Dr. Rohmer of Strasbourg. (3) Congenital scoliosis, introduced by Drs. Mouchet and C. Roederer. (4) The rearing of children outside the family, introduced by Drs. H. Méry and Ribadeau-Dumas. (5) Infantile hygiene in Switzerland from the social standpoint, introduced by Dr. A. D'Espine and Dr. Reh of Geneva. Further information can be obtained from the general secretary, Dr. Péhu, 24, Place Bellecour, Lyons.

The Milroy Lectures

ON THE

INFLUENCE OF INDUSTRIAL EMPLOYMENT UPON GENERAL HEALTH.

DELIVERED BEFORE THE ROYAL COLLEGE OF PHYSICIANS
OF LONDON ON MARCH 9TH, 14TH, AND 16TH, 1922,

BY

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LECTURE III.

So far we have investigated the hygienic importance of industry by the topographical and historical methods; the result has been to show that some conditions of industrial life are correlated with factors of excessive mortality; we have failed to establish a causal nexus—to pass from correlation to causation. We have now to consider whether other methods of study lead to more precise conclusions. We might, in the first place, retain the topographical method and adopt another unit—for instance, the physique of the inhabitants of different towns. But unfortunately such data as exist are quite inadequate, so I pass to another line of inquiry—namely, occupational comparisons, the unit of comparison being a rate of mortality.

Occupational Mortality in General.

In this region Dr. Farr was, as in so many other regions, the pioneer, and decennial evaluations of occupational mortality in England and Wales go back fifty years; in any compilation of industrial rates of mortality the English statistics are predominant, and they have done duty in arguments and influenced legislation in all parts of the world. It was therefore to be expected that their value would be frequently criticized. The criticisms to which such statistics are exposed fall into two classes: (1) It is doubted whether the rate of mortality shown in a particular occupation does in fact represent the "real" mortality of that occupation; (2) it is doubted whether, even if the former question is answered affirmatively, an excess of mortality in a particular occupation is really cogent evidence that such occupation is unhealthy.

The weaknesses which authorize the criticisms under (1) are: (a) That the basis of classification has not only been changed from census to census, but is still (I am speaking of the census of 1911) "only truly occupational in parts, being as regards manufacture for the most part frankly industrial. Thus the driver of a motor car is classified as a chauffeur—his occupation; but the workman engaged upon its manufacture is classed either as a motor-car chassis maker or as a motor-car body maker—headings which indicate merely the industry in connexion with which he plies his trade, whether it be that of tinsmith, moulder, fitter, sandblast-man, painter, or what not" (General Report of Census of England and Wales, 1911, Cmd. 8491 of 1917, p. 12). (b) That there is no guarantee that the occupational description in virtue of which an individual contributes to the total years of life lived, to the denominator of the fraction measuring the rate of mortality, will be the same as that upon the death certificate which admits him to the numerator.

The occupational complexity of our own profession is not greater than that of most industries, but it sufficiently illustrates the point. The industrial description of all persons admitted to the *Medical Register* since 1886 would be, I suppose, physician, or surgeon, or accoucheur, any one of the terms implying both the others. Consider the troubles that would await a statistician who wished to determine whether the rate of mortality of physicians in the occupational sense differed from that of physicians in the industrial sense. It is quite as much for these reasons as for the desire to base rates upon large numbers that the statistical grouping of occupations is a coarse one.

There is a further point: although the method of inquiry which we are discussing formally renounces geographical or topographical differences, for some of the largest industries of the country the renunciation is purely formal. In 1911 83.2 per cent. of the total number of persons engaged in cotton manufacture were enumerated in Lancashire and nearly all the remainder in the adjoining counties of the West Riding of Yorkshire and Cheshire; 88.7 per cent. of those engaged in wool and worsted manufacture were enumerated in the West Riding of Yorkshire. When we compare the rates of mortality of cotton manufacturers with those of, say, printers,

we are comparing the conditions of persons concentrated in one part of the country with those of workers distributed all over it.

One sees, then, that the returns must be used with great care, even when we wish to answer the simple question whether such or such an occupation experiences a high rate of mortality. When we go on to ask the further question, Is the occupation itself responsible for that high rate of mortality? the difficulties are very much greater, because of occupational selection and the movement from one occupation to another, which may be due to a deterioration in health.

To seek fine shades of meaning in the statistics of the many scheduled occupations, to subject them to the meticulous analysis which modern statistical artists have devised, is often to waste time. But there is something of value in the data, and even the severest critics have not spared themselves in the search for gold amongst large quantities of quartz. I shall restrict myself to a very few broad comparisons.

Special Occupations.

I shall speak of twelve industrial groups, including the clergy, commercial clerks, agricultural labourers, printers, cotton manufacturers, wool and worsted manufacturers, tailors, and shoemakers. The selection has not been made at random. In the first place the numbers of lives exposed to risk—that is, thrice the enumerated census populations—are large, more than 100,000 in each instance. In the second place the groups illustrate different phases of industrial life. The clergy, whose mortality I take as a standard, typify a sober, clean-living, educated, but not wealthy, group under highly diversified environmental conditions. Commercial clerks typify the less successful lower-middle-class population of cities; agricultural labourers are, in the main, very poor persons living in the country, whose sons and brothers swell the ranks of the industrial townsmen; cotton and woollen manufacturers are examples of skilled workers localized in a particular area; printers are skilled artisans not concentrated in any one part of the country; tailors are heterogeneous;

TABLE I.—Occupational Mortality Rates (1910-12) expressed in Terms of Mortality among Clergy. Ages 25-35.

Group.	All Causes.	Phthisis.	Pneumonia.
Clergy	100	100	100
Agricultural labourers	147	151	181
Cotton manufacturers	177	159	303
All textile workers	195	235	320
Tailors	205	341	227
Wool and worsted workers	209	262	322
Textile dyers and bleachers	218	244	324
Printers	218	402	200
Hand compositors	221	480	160
Commercial clerks	233	387	222
Shoemakers	247	449	251
Shoemakers in Northants and Leicestershire	249	539	138

TABLE II.—Occupational Mortality Rates (1910-12). Ages 35-45.

Group.	All Causes.	Phthisis.	Pneumonia.
Clergy	100	100	100
Agricultural labourers	156	165	191
Cotton manufacturers	208	236	247
All textiles	219	259	216
Wool and worsted makers	225	248	232
Textile dyers and bleachers	242	276	332
Tailors	250	428	219
Shoemakers in Northants and Leicestershire	250	566	181
Printers	251	464	239
Hand compositors	259	522	281
Shoemakers	270	486	233
Commercial clerks	281	447	239

TABLE III.—Occupational Mortality Rates (1910-12). Ages 45-55.

Group.	All Causes.	Phthisis.	Pneumonia.
Clergy	100	100	100
Agricultural labourers	107	167	114
Shoemakers in Northants and Leicestershire	163	508	121
Wool and worsted makers	188	295	125
Printers	192	571	175
All textiles	194	347	187
Shoemakers	196	594	153
Hand compositors	197	595	139
Cotton manufacturers	199	349	222
Tailors	202	485	192
Textile dyers and bleachers	212	354	206
Commercial clerks	216	453	171

TABLE IV.—Occupational Mortality Rates (1910-12). Ages 55-65.

Group.	All Causes.	Phthisis.	Pneumonia.	Bronchitis.
Agricultural labourers	79	148	116	194
Clergy	100	100	100	100
Shoemakers in Northants and Leicestershire	120	384	154	338
Printers	130	496	137	413
Hand compositors	132	593	124	387
Shoemakers	136	484	158	500
Tailors	141	493	150	515
Commercial clerks	146	463	201	349
Wool and worsted makers	156	348	193	513
All textiles	159	356	206	872
Textile dyers and bleachers	160	468	208	538
Cotton manufacturers	169	416	250	803

while shoemakers can be divided, very roughly, by a geographical test into factory and non-factory operatives. In the tables are shown the rates of mortality in four decennial age groups from all causes, from phthisis, from pneumonia, and from bronchitis—all expressed as percentages of the rates upon clergymen. I have also inserted in these tables the combined results for the textile industries, for textile dyers and bleachers, also the rates peculiar to one class of printers, hand compositors, and those appertaining to boot and shoe or slipper makers enumerated in the counties of Northants and Leicester, the most important concentration of factory boot manufacture in England and Wales.

Let us begin with a general survey of mortality in age groups. At 25 to 35 agricultural labourers experience a mortality of some 50 per cent. excess over clergy; the skilled industrials of the north-west show a greater excess, but shoemakers—whether the factory operatives of the Midlands or shoemakers as a whole—are the worst off, having two and a half times the death rate of the clergy. The principal assigned cause is phthisis, which amongst the operatives of Northampton and Leicestershire is five times as fatal as amongst clergy and twice as fatal as amongst textile workers. Is this attributable to (a) climatological differences, (b) differences of recruitment, or (c) differences of factory environment? Answers to these questions cannot be much better than guesses. But if—and the speculation is plausible—the nearest area of recruitment is that most usually drawn upon to feed an industrial centre, one might suppose that the supply of unsalted youth, of genuine rustics, to be found within fifty miles of the great Midland boot factories is very much greater than that available for the cotton mills; but, as said before, this is only a speculation. Such little direct evidence as we have respecting immigration into the boot-manufacturing centres does not suggest any large inward movement.

In the decennium 1881-90, indeed, Northampton gained considerably by migration (864 males and 1,030 females in the age group 15 to 35), but in the following decennium both age groups show a small outward balance (Welton, 357). In a paper contributed to

the *Journal of the Royal Statistical Society* in 1912 Mr. Welton summarized the changes (all ages) shown by the census of 1911 both in Leicester and Northampton. Each showed an outward balance—Leicester of 7,100 males and 3,680 females, Northampton of 2,476 males and 2,040 females. These results do not of course prove that there has not been a considerable inward movement from the country compensated for by emigration of the town born, but clearly they are not evidence in favour of the hypothesis suggested, and must therefore be specially emphasized.

Another way of testing the hypothesis is to examine whether the ratio of mortality rates, using the clergy as a standard, has increased over the last sixty years, since the transformation of the shoe manufacture from a home to a factory industry has been largely effected in that period, especially during the last twenty years. As a matter of fact, the relative mortality at the earlier ages has increased.

Thus, combining Dr. Farr's 1860 and 1870 enumerations, one has for the age group 25 to 35 per mille rates of mortality of 4.7 (clergy) and 9.52 (shoemakers), giving a percentage ratio of 203 for the shoemakers; in the enumeration of 1890-92 the rates are 4.2 and 7.66—ratio 182; in 1900-2, 2.7 and 6.33—ratio 234; while, as we have seen, for 1910-12 the ratio is 249 for the Leicester and Northants shoemakers and 247 for all shoemakers. The ratios in the next age group are 180, 218, 240, and 250; at 45 to 55, 126, 189, 179, 163; at 55 to 65, 135, 139, 128, 120. These figures are consistent with the view that the change from home to factory industry—despite the notorious objections to the former—has not been relatively favourable at earlier ages, and are therefore consistent with the hypothesis. But they do not constitute a strong argument.

The rate of mortality of commercial clerks should be a source of painful reflection; in this age group it is exceeded by shoemakers alone; at 35 to 45 it is the highest of any in our table, and holds the same place in the decennium 45 to 55. No doubt this group bears much the same relation to middle-class industries as the group general labourer does to the hand-working classes—it includes, that is to say, a sensible proportion of weaklings in the lower and of failures in the later age groups. But if we admit any other factors of mortality than those assignable to selection we shall not doubt that the hygiene of what used to be called the black-coated labourer needs as much attention as that of the industrial worker. Next to clerks come printers, whose mortality from phthisis is far beyond that of any group save the shoemakers; their mortality from bronchitis and pneumonia is above the average, but less than that of the textile workers, who experienced a much smaller mortality from phthisis. The two great textile groups show some difference one from another: the cotton manufacturers have the lower general mortality, and the lower mortality from each special cause named.

In the age of industrial maturity, 35 to 45, the position relatively and absolutely has changed; all our employed groups except agricultural labourers and the Midland bootmakers stand worse in terms of the mortality of the clergy than they did. The shoemakers still have the highest mortality from phthisis, but their relative mortality is no worse than before; both the textile groups have deteriorated, and the tailors have deteriorated a great deal. If the bad showing of the Midland shoemakers in the earlier ages as compared with the cotton and woollen operatives was due to the larger proportion of unsalted rustics, then a selective mortality has just enabled them to keep station while the textile workers are beginning to falter. Agricultural labourers, despite the winnowing out of the ambitious fit who have passed into the towns, show no appreciable deterioration.

In the next age group all our industrials have improved relatively to the standard, but this fact must not be made the subject of too comfortable reflections. There are weaknesses in every method of statistical comparison, and the weakness of the particular one here used is that arithmetical necessity imposes upon the percentage ratios some degree of convergence.

It were too ambitious to speak of a "law" of mortality as a function of age, but if we suppose that the force of mortality is compounded of an element independent of age and an element increasing in geometrical proportion with age—the hypothesis of Gompertz, or, as it is usually termed, the Makeham-Gompertz hypothesis—it is plain that with advancing age two formulae of mortality having different constants will converge nearer and nearer one to the other. So that if we find the ratios in my tables growing larger we may certainly infer a deterioration, but if they grow smaller we cannot infer an improvement without special examination (which the data do not justify).

The order of the groups is now quite different: the Midland bootmakers, although still afflicted with a high rate of phthisis, have the next lowest mortality from all causes to the agricultural labourers; tailors are almost as bad as

commercial clerks, but very little worse than cotton manufacturers, who stand higher than printers and appreciably higher than woollen goods makers. In this age group the cotton workers begin to be seriously troubled by pneumonia and bronchitis, responsible for 8.7 and 5.8 per cent. respectively of all deaths, and at rates 77 and 60 per cent. in excess of those upon woollen and worsted workers. You will remember that the mortality from these respiratory diseases is above the average both in Westmorland and Cumberland, so that part of this excess may be climatological, not industrial.

In the age period 55 to 65, the textile workers have reached the worst place, while the Midland bootmakers stand best amongst the industrially employed; bronchitis, responsible for 10.7 per cent. of all deaths in the industry, is much more than twice as fatal to the cotton makers as to the shoemakers, and more fatal—in the proportion of 8 to 5—than to woollen and worsted makers. Printers, although still liable to an excessive mortality from phthisis, stand better than the textile operatives.

Let us concentrate our attention still more closely upon these particular groups of contrasting workers—the cotton manufacturers of Lancashire, the woollen workers of the West Riding, the shoemakers of Northants and Leicester, and the printers of all England. In which of these groups of skilled artisans is the largest number of maximally productive years available? Using the life table method, we find that the average number of years lived from 25 to 55 is 27.58 for cotton manufacturers; 27.39 for woollen manufacturers; 27.23 for printers; 27.17 for the boot and shoe makers in Leicestershire and Northamptonshire. No practical importance attaches to the small differences between these figures; we may take it that in each occupation some 91 per cent. of the theoretically possible term of years is enjoyed. It is, however, instructive to see how quite different courses of mortality have led over this period to substantially the same final result. The textile workers with their low adolescent mortality counterbalance, in this comparison, the increasingly unfavourable effects of later adult mortality. Had we carried the calculation over another decade we should have found an advantage amongst the shoemakers, who were left at 55 with substantially the same number of survivors of entrants at 25 as the cotton manufacturers—namely, 7,719 cotton makers and 7,716 bootmakers out of an initial 10,000 in each case.

These results admit of certain deductions and prompt certain speculations. The deductions are these. Although phthisis is by far the most deadly of the causes of mortality in early adult life, it does not follow that an industry of which phthisis takes a heavy toll is an unhealthy industry, if we use as our measure of healthiness the proportion of years lived in maturity. We can hardly even suppose that these are pathologically weighted years, that for instance the 27 odd years lived by printers between 25 and 55 are more smitten by ill health than those of the cotton makers; because after 55 the mortality rates of printers are not worse than, but better than, those of cotton manufacturers. Dr. Tebb and I reached the same conclusion by another path. We found that if a very much larger number of occupational groups were treated by the method of correlation deaths from phthisis as the dependent and deaths from all other causes as the independent variable, both printers and shoemakers (tailors, bookbinders, hosiery makers, and cabinet makers were in the same case) experienced a mortality from phthisis out of proportion to their mortality from other causes. Miss C. M. Thompson (see Greenwood, (1), 336) also found that in the London boroughs there was little correlation between the death rate from tuberculosis and from other diseases independent of that between tuberculosis, overcrowding, and industrial employment. Accordingly we may, I think, conclude that the problem of phthisis-ridden industries is not exclusively a problem of the recruitment of weaklings. Of course this would be a mere truism if my survey included any of the occupations dealt with by Professor Collis in his Milroy lectures, subject to a specific risk, such as that of the Sheffield grinders, but I have entirely excluded these from my consideration. It further appears that the special liability of workers in these occupations is not inherent in the art itself. Thus the whole group of shoemakers, which must include a very large number of non-factory shoemakers, does not at ages under 45 experience so heavy a mortality from phthisis as the Northampton and Leicester group, nor did Dr. Tebb and I find the same excessive mortality from phthisis amongst Dutch shoemakers. Again, the English

cotton manufacturers have relatively little phthisis, but the same class of operatives in Massachusetts (Perry) suffer greatly from the disease.

The Validity of the Statistical Method.

A point has now been reached in our journey at which we may pause and ask ourselves whether any further progress be possible without the aid of other implements than those we have so far employed—namely, general epidemiological and elementary statistical methods. Within the last few months two eminent members of our profession have rated the statistical method low. In a new edition of his *Technique of the Test and Capillary Glass Tube* Sir Almroth Wright expresses his conviction that in *therapeutic* research clinical experience “unaided by apparatus and technique” is “of infinitely little account,” and statistical evaluation (except for propagandist purposes) but “old abusing God’s patience.”

The name of Sydenham warns us that either “research” or “apparatus and technique” must be understood in what theologians called a non-natural sense before we can accept Sir Almroth’s condemnation of clinical experience as an instrument of therapeutic advance. By some such casuistry we might hope to bring the Heberdens, the Jenners, and the Murchisons from under the edge of Sir Almroth’s sword. But our charity ought not to reach the “statistical evaluators,” since, I gather, their “accepted statistical doctrine teaches that all false judgements are quashed when we collect enough cases.” These persons, it seems, have but a single method of procedure—namely, “arbitrarily selecting one particular criterion and counting the exact proportion of cases in each group which satisfy and fail to satisfy it.” The doctrine is justly stigmatized by Sir Almroth as false, and the method argues a strange limitation of outlook. Were Sir Almroth able to cite any instance of a statistician who accepted this heretical doctrine and exemplified this erroneous practice, the conclusion he invites his readers to draw would attain the quite respectable rank of a fallacy *a dicto secundum quid ad dictum simpliciter*. No such instance being afforded, Sir Almroth merely prompts a reference to Job xxxviii, 2, and provides one more case of the darkening of counsel.

Sir Arthur Newsholme, who, having a wider experience of statistical methods than Sir Almroth Wright, has no doubt a higher opinion of their value as instruments of research, still seems in agreement with Sir Almroth to the extent of assigning to the statistician a place in the propagandist rather than in the research department of the public health service.

If I have correctly followed Sir Arthur’s argument developed in his address to the American Statistical Association, he would assent to the following propositions: If in a particular city, or group of cities, the rate of infant mortality began to decline after the establishment of infant welfare centres, and continued to decline *pari passu* with the extension and development of such centres, it would be proper to use the statistics as evidence that the establishment of the centres had a causal relation to the decline of infant mortality. If it were also found that in some other group of cities where no such centres had been established the rate of infant mortality declined synchronously with that of the former cities and to the same extent, this second group of statistical facts should in no way modify the conclusion attempted to be drawn, and impressed upon the general public by statistical charts, in the former case. Sir Arthur Newsholme is anxious to repudiate an intellectual heresy which popular wisdom has stigmatized in the adage that there are more ways of killing a dog than drowning him. Because, let us say, infant mortality declines without welfare centres or care committees, it does *not* follow that welfare centres and care committees are useless. But it *does* follow that infant salvation may be attained in more ways than one, and we may fairly ask whether the appraisal of these various other causes is not a proper object of scientific research, to attain which the statistical method is a valuable instrument. I believe that Sir Arthur, in his zeal for the repudiation of Sabellius, has not sufficiently guarded himself against the wiles of Arius. It is, no doubt, very wrong of non-medical statisticians to confound the persons of the etiological Trinity; but it is quite easy for the administrator whose zeal for laboratory results may not always be tempered by expert knowledge to divide the substance. “Il existe,” said a great physiologist, “toute une école médicale dont les sectateurs n’ont, bien entendu, jamais haï les laboratoires, mais pour qui le mot ‘expérience’ tient lieu de tout, comme le ‘tarte à la crème’ de la comédie” (Bert, *Pression barométrique*, p. 516). This sarcasm is not entirely unmerited by some who assure us that the rôle of the statistician is to be the publicity agent of the experimental pathologist. The experimental philosophy of the administrator is not always that of his contemporary in the bacteriological department. I suppose many medical officers of health believe that the reduction of sources of direct infection *in vivo* is the most important means of combating the spread of fatal tuberculosis. The segregation of “open” cases of phthisis has been eagerly recommended, statistics correlating such measures with the declining death rate have been widely circulated, and it has been asserted that such measures are the administrative expression of experimental truths.

But the experimental pathologists of our time are not unanimous. Professor Calmette, indeed, seems to regard exposure to an infection held within not very clearly defined bounds to be a blessing in disguise. He dissents from much which was recently orthodox in respect of paths of infection, reducing the importance of milk contamination to a very modest place, although he is quite willing to “considérer comme dangereuse, surtout pour les jeunes enfants, l’ingestion répétée des laits crus riches en bacilles” (313). Upon the basis of experimental and observational studies of the last twenty years the etiological problem of tuberculosis has been formulated by men who are certainly not “arm-chair statisticians” in terms very different from those which inspired the faith of our sanitarians even ten years ago. Colonel George Bushnell of the United States Army Medical Service has recently published a summary of this evidence. Having epitomized the now substantial literature testifying to the havoc wrought by tuberculosis upon a native race exposed to the influences of civilization suddenly, having made it appear that these ravages are largely independent of the goodness or badness of the conditions of life, and having contrasted the extent and clinical evolution of the tuberculosis of the civilized races with that of the savages, Colonel Bushnell writes:

“Civilized man can never escape the dangers of infection with the tubercle bacillus. But if we did escape the danger of infection, we should also lose the benefits of tubercularization. . . . At the time when control of expectoration first bulked so largely in the eyes of the sanitarian the community was divided in his thought into two classes as respects tuberculosis, the tuberculous and the healthy. Very slowly and reluctantly since that time the knowledge of the true facts has been acquired, and still more slowly and reluctantly has it been disseminated—in fact, it still seems incredible to many that tubercularization should be so wellnigh universal as it undoubtedly is. If it seems likewise a terrible fact that conclusion is erroneous. The error lies in the assumption that tuberculosis is necessarily an evil. We fix our eyes upon the advanced case of tuberculous disease, a pitiable sight indeed, and that for us spells tuberculosis, but we forget that such a case is the comparative exception, the illustration of a failure in immunization; we forget that the majority of the population never know that they are tubercularized!” (182). Dr. K. F. Andvord of Christiania, in a very suggestive paper issued a few months ago, has reached conclusions which differ from those epitomized by Colonel Bushnell in one respect, but in others are similar to his. Dr. Andvord holds that in a tubercularized community the source of nearly all fatal tuberculosis of adult life is infection during the first year, the fatal issue being a consequence of auto-reinfection. To support this thesis he relies upon two lines of argument. In the first place, the direct evidence of Bergman of Upsala, who has investigated the after-history of 1,004 children of tuberculous families. Bergman found (I am epitomizing at secondhand—the original is not accessible to me) that all the persons in his sample who died of tuberculosis in childhood or adult life and 90 per cent. of those still alive but suffering from clinically demonstrable tuberculosis had been exposed to infection within the first years of life, but that not a single death from tuberculosis could be found amongst those whose exposure had been deferred until after the fourth year. In the second place, Andvord shows that the age incidence of fatal tuberculosis, and the statistical observations of Dreijer, Bang, Harbitz, and others on the frequency of a tuberculous or “pre-tuberculous” condition, may be reconciled on his hypothesis. Hence he thinks “the most effective prophylaxis would be the protection, by every possible means, of children, notably those in the first three or four years of life, from the more severe infections, either by removing the source of infection from the children’s neighbourhood, or, if possible, by the isolation of the children” (115). He also thinks it necessary to obtain more accurate knowledge of the stages which precede pulmonary tuberculosis, particularly in late childhood and adolescence, and “at least as important to attempt to minimize or remove predisposing causes, and thus possibly to prevent re-auto-infection as to protect the individual from the danger of infection by his surroundings” (116). A practical conclusion in harmony with that of my distinguished predecessor in this lectureship, the late Dr. Bulstrode.

I have not cited these results because I hold them to be ascertained truths (the reasoning seems to me loose, and the total neglect of selection strange), still less for the purpose of twitting those who have rebuked the medical shortcomings of the lay biometricians with being themselves somewhat in the rear of the pathological march of mind. But I may fairly claim them as justifications of the standpoints of Professor Karl Pearson, Dr. John Brownlee, Professor Raymond Pearl, and, if I may venture to add my name to this distinguished company, myself. Whether it be true in the more general sense of Colonel Bushnell’s argument that the fatal evolution of tuberculosis in members of a tubercularized community is a function of environment in its most general expression; or whether, as Dr. Andvord believes, this pathological accident can only occur to those infected early in life; or, again, whether a change of survival values be not the really significant thing: to discover the intrinsic or extrinsic factors *over and above the bacillary infection* is an object of the greatest importance. We cannot afford to neglect the intrinsic predisposing factors to which Professor Pearson has devoted attention, the contrasting epidemiological types which Dr. Brownlee has displayed, or the general physiological

and economic procatactic factors which fascinate me. But if this be true we must accept the corollary that now and for many generations the statistical method will be needed; the time is not yet when the harvest can be separated into wheat and tares by some more exact methods of the laboratory; we do not know which is wheat and which tares. For these reasons the statistical method must be ranked *not* as the *only* but as one of the principal instruments of research in preventive medicine and epidemiology, and should be a more serious item in the training of the student. It is discreditable to our intellectual culture that the young sanitarian learns little of statistics and nothing of history.

Etiology.

Let us now return to the etiology of industrial disease and examine particularly the case of phthisis. If my general line of argument be correct, we ought first to examine the broad distinctions between industrial and non-industrial employment. Obviously the first and most general distinction is that those industrially employed are for the most part inhabitants of towns. I have already spoken at some length of internal migration, but there still remains much to be said upon the point. The conditions of life to which the youth in the country is exposed are widely different from those awaiting him as a factory employee. In the professional classes the transition first from the home to the public school, then to the university, and finally to the office or consulting-room, is usually without abruptness. But it needs little imagination to perceive that for the lads and girls who leave our country districts in search of employment the transition may be a very sudden one. More than sixty years ago this subject received incidental notice in a classical report by the late Dr. Edward Smith, F.R.S., on the health of the London printers. Dr. Smith examined a number of young apprentices, and observed that "usually they are pale and thin, with sunken features and large pupils, but in other cases they are fleshy and have colour, and do not show a low type of health. Very generally they affirm that they are well, take their food heartily and digest it well, and have not degenerated in health." He adds: "It has been stated that country boys sooner lose flesh and colour than town boys, and are among the earliest to fall into serious disease" (Sixth Report of the Medical Officer of the Privy Council, 1863, Appendix, p. 386). When one considers that an indoor trade imposes upon the young peasant a way of life very different from that of the village, and that his earning capacity is small, it would not be surprising if the physiological conditions of his life were extremely unfavourable and the opinion quoted by Dr. Smith well founded.

If one, purely for the sake of argument, assumes that a change from rural conditions to urban life is a factor of sickness and mortality, then it follows that the mechanism of the deterioration, its etiology in a narrower sense, should be discussed, if only because in testing the general hypothesis it may be practicable to assemble particulars which would also throw light on the mechanism of any change. Three contributory causes are suggested: (1) Changes of diet; (2) reduction of bodily exercise; (3) the immediate effects of atmospheric change.

The Factor of Nutrition.

In 1862 Dr. Edward Smith carried out the widest survey of the dietetic habits of the poorest working people which has yet been undertaken here, a study which is in urgent need of repetition. Using Dr. Smith's data and much other scattered information, Miss W. A. Mackenzie has recently published an instructive comparison between the budgets of the working classes in 1860, in 1880, and in 1914. From our point of view the estimated consumption per "man" of the poorest 10 per cent. of wage-earners is of particular interest. Miss Mackenzie's statistics lead to the conclusion that in 1860 the daily calorie yield of the purchased diet was about 2,750; in 1880 it was 3,400, and in 1914 3,150. There is a considerable increase of gross energy yield, but a far more striking increase of the consumption of fat. In 1860 the total fat of the diet was about 24 grams per "man" per day, providing rather less than 8 per cent. of the total energy; in 1880 the amount had nearly doubled, being 47 grams per day and furnishing 12.8 per cent. of the total calories; in 1914 the amount was slightly less, 45 grams, furnishing 13.4 per cent. of the calories. The best of these diets provides too little fat.

Professor Starling, in the Oliver Sharpey Lectures of 1919 (p. 72 et seq.), observed that a diet should contain such a proportion of fat that approximately 25 per cent. of the total

calories were derived from the metabolism of fat. In commenting upon Smith's work, Simon noted that Irish agricultural labourers were better fed than English farm workers. But, although all the English labourers had some separated fat in the diet, this was only true of 40 per cent. of Smith's Irish sample, and in the latter the average intake was not much more than one-fifth of the English average.

These facts may be no more than a physiological translation of economic changes, since fat is the most costly ingredient of the diet. But perhaps the direct importance of the matter is greater than one usually supposes.

In 1914 Dr. A. E. Carver of Birmingham reported on an investigation of the diets of labouring families in the city. He performed a sampling experiment. A series of families, in each of which one member was tuberculous, was compared with a control series. Pains were taken to secure an equal distribution of incomes in the groups compared, so that there was no economic bias. Family budgets were analysed with great care, and the diets were finally expressed in "man" values on the lines of Atwater's investigation. The result was to show that, both in respect of total energy yield and in consumption of fat, the families containing tuberculous members fell much behind the controls.

The samples were small, but a study of the figures (for which I have to thank my colleague, Miss Newbold) makes it improbable that they are samples of a common "universe"—in other words, the difference is not very likely to be a chance error.* Of course, there are material difficulties of interpretation. Further study of dietetics has made it probable that Atwater's original coefficients, which Dr. Carver used, have not the right weights, and that they appreciably overestimate the "man" value. But, as the proportion of children was rather higher in the tuberculous than in the healthy families, this method of calculation would tend not to exaggerate but to minimize the difference. A much graver difficulty is that of deciding whether the tuberculous families were tuberculous because they were inadequately fed or whether the sequence should be reversed. In Dr. Carver's sample of tuberculous families in receipt of 3*s.* to 3*s.* 6*d.*, of 30 children in eight families 10 were tuberculous; 6 of the 8 women and 1 man were tuberculous. In his most prosperous group 13 of the 32 children were affected and 3 adult women. When one is sick it is hard to consume rich food, and there is some danger of reasoning on samples of this kind being *ex post facto*. Yet the observations are suggestive. The following is no doubt a still weaker argument. The standardized death rate from tuberculosis of Irish cotton operatives in the Fall River experience was 5.9 per mille for males, 4.97 for females; for non-Irish operatives 2.15 and 2.98. For non-tuberculous diseases the corresponding rates are 4.33 and 6.46, 2.97 and 3.78 (Perry, 198). The mean populations at risk were, however, only 768 Irish males and 1,642 females, the non-Irish amounting to 10,520 males and 11,568 females. The total deaths were 169 Irish and 640 non-Irish.

It is by no means certain that the diet of the Irish peasant is as good now as it was sixty years ago; probably it is less rich in fats. (See Report of Irish Milk Commission, Cd. 6936 of 1913, pp. 11-12.) If the *prima facie* interpretation of Dr. Carver's statistics be the true interpretation, some light is thrown upon the American industrial mortalities. Whether, as some hold, fat is *not* replaceable in human metabolism by isodynamic quantities of carbohydrate (for a critical discussion see Krogh and Lindhart), or whether the accessory food factors should be invoked, are questions which the data do not warrant me in raising. But I agree with Professor Collis in thinking that diet, quite apart from its energetic sufficiency (that an energetically insufficient diet is one of the great procatactic factors of tuberculosis the experience of Central Europe since 1916 has made seem probable), must be taken into consideration. That this is a matter which will especially affect the emigrant from the country into the town is likely. A good many townsmen and townswomen, born and bred, get very poor value for their money in food marketing. The inexperienced stranger is in a still worse case.

"Without claiming that nutrition is the only factor at work in determining the result of infection," writes Dr. Carver, "we consider that indirectly its influence is a most responsible one" (p. 7). The claim is, I believe, well founded.

Bodily Exercise.

Passing now to the second category, the lack of bodily exercise, I have little need to do more than refer to the case stated by my predecessor Wing-Commander Flack. It is not a very rash speculation to suppose that, bad as are the consequences of failure to use all parts of the human machine in any of us, a transition from hard manual labour in the open air to the specialized and cramping movements of a machine tender will be particularly resented by the human body. At present we have hardly any data, none relating to the factory class. But the observations of my colleague Dr. Lucy Cripps upon young women of the middle class reveal such an enormous difference between the standards attained by apparently healthy young women and those of the youths who offer themselves for the Royal Air Force, that it is impossible to doubt that Wing-Commander Flack and Professor Dreyer have opened up an important field of research.

* The differences are 16.9 ± 3.3 grams of fat; 392.9 ± 82.0 calories.

Ventilation.

These two categories—that of diet and that of bodily exercise—concern the individual; there remains the third—that of the whole complex of environmental influences which the individual cannot modify, which are functions of collective undertakings. In the first place there are the consequences of the localization of industries, and this in the textile manufacturing is probably or certainly one determinant of the death rates from non-tuberculous respiratory diseases. So far as this is climatological, it does not admit of modification. But if the evidence summarized in an earlier lecture has satisfied you that in such towns as Bolton or Warrington the respiratory mortality is in excess of that experienced in contiguous but non-industrial regions, and that it exceeds the mortality of other industrial towns in the southern and central parts of the country, it follows that the measures advocated for the reduction of the smoke nuisance have more than an aesthetic justification.

Next we have the factory atmosphere itself and the new light thrown upon its importance by the researches of Dr. Leonard Hill and his colleagues. Like all absolutely first-rate work, its teaching seems quite obvious when it has been expounded. We have all been drilled in the lore of metabolism, the effects upon the respiratory exchange of the environment. Yet it had occurred to very few physiologists to inquire whether within the range of indoor employments variations could be measured and correlated with human efficiency. Dr. Hill has, I think, completely demonstrated the truth of his main propositions. The measurements made under his supervision of the cooling powers of different factories and different departments of the same factory—in addition to his own publications, mention should be made of such reports as that of Messrs. Bedford and Hambly on boot and shoe factories—establish a strong *prima facie* case in favour of the conclusion that by attending to the indications of the kata-thermometer we shall do much to render the equilibrium of the worker more stable and help him to resist the procatartic factors of disease which it has been my chief object to emphasize.

Difficulties.

But before concluding I must not omit to exhibit as clearly as I can the points which the teaching I have attempted to expound does not satisfactorily explain. The weakest link in my chain is this: One does find certain industries—most strikingly that of printers—wherein a specific liability to a non-occupational disease is observed. The argument that the relative immunity from phthisis and the extreme liability to fatal diseases of the respiratory organs other than phthisis weighing down the later years of textile workers, is a consequence of immunity by selection—whether in the precise sense of the Calmette doctrine of tuberculosis or not—and of subsequently deleterious environmental influences destroying resistance to other pathogenic organisms, this argument is plausible. The explanation of the peculiar position of the Midland shoe operatives in relation to phthisis as a consequence of unfavourable industrial selection and a more favourable subsequent exposure is also a plausible argument and deserving of full verification or rebuttal.

But neither argument satisfactorily explains why a skilled trade, carried out under a diversity of climatological and economic conditions and not subject to a high death rate from all other causes, should occupy the place it does with regard to phthisis mortality. It is easy to show that the atmospheric conditions in printing establishments are often unsatisfactory, but the results which I have seen do not suggest that printing shops are worse than other factories. We must, therefore, admit the possibility that some special and hitherto undiscovered influences are at work. To reveal these both laboratory and field investigations are necessary. Of the former, the special technical studies of conditions which may favour the growth and virulence of the infecting organism, I have not to speak. Of the latter I would remark that here, as in so many branches of industrial hygiene, we are hampered by the fact that few trained scientific observers have sufficient familiarity with industrial conditions to be competent to make investigations. Fortunately, the recruitment of such men as Dr. H. M. Vernon into the army of industrial research workers, and the important results already obtained—for instance, in Dr. Vernon's studies of the tinplate industry—hold out great promise for the future. What is needed, and is at length being obtained, is a dispassionate study by scientific men of all the circumstances of industrial

employment. A great many Balaams have set out to deal with the problem of disease; few of them have been broad-minded enough to take a hint from the ass—a beast quite untrained in scientific method but with a good deal of practical experience. It is a fault not to see the wood for the trees, but one will not be wise to ignore all but a few of the trees.

General Conclusions.

Nineteen years ago the late Dr. Bulstrode ended his course of Milroy lectures with these words:

"If I were told to select three, and three only, of the agencies with which I have in some degree dealt in these lectures as those upon which, assuming always a sufficiency of food, I would place most reliance as regards the control of pulmonary tuberculosis, I should arrange them somewhat in this order. (1) The education of older children in the principles of health, and the periodical physical examination of all school children with a view to improving the health of those prone to tuberculosis, or who may already be suffering from the disease in an unrecognized though not unrecognizable form. The direct and indirect results which would accrue from this would soon make themselves felt in the direction of an unwillingness on the part of the people to occupy slums or to dwell in places where the sun rarely penetrates. It would also result later in a return to local and perhaps central administrative bodies of representatives who were more alive to the importance of the public health aspect of their duties than is always the case at the present time. (2) Some such system of compulsory insurance against sickness and invalidity as obtains in Germany, and which, it is important to note, makes for the provision of sanatoriums, the support of those threatened with illness, and the general well-being of the poorer classes. (3) Better housing and improved conditions of employment of the working classes—that is, more light, more air-space, better ventilation, and greater cleanliness in the home, the workshop, and the factory" (442).

Much has been done in the last twenty years to satisfy these requirements, but there has, perhaps, been overmuch insistence upon particulars, too little regard for general principles. My aim has been to restate some of the broad epidemiological principles which inspired Dr. Bulstrode, to show that their applicability is even wider than to the case of tuberculosis, and to plead for a physiological philosophy of disease.

The ideal physician of Linacre was a man of wide and various learning, and the College has always placed this ideal before its alumni. Translated into practice it means, I suppose, that in our secular warfare against disease we must use all the intellectual weapons we have, and not vainly assert that this or that instrument is alone of value. If in these lectures I have dwelt upon the general reactions of the environment in disposing man to fall a victim to this or that antagonist, and upon the aspects which are most easily displayed by statistical inquiry, it is not because I undervalue other elements of the problem or other methods of research.

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ANEURYSMS OF THE THORACIC AORTA INVOLVING THE LUNG.

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THE two cases of aneurysm of the thoracic aorta described below present several features of considerable clinical and pathological interest. Both aneurysms ruptured within a fortnight of each other while under observation in hospital, and in each case the aneurysmal sac produced extensive changes in the lung. The second case was particularly interesting in its close simulation of chronic pulmonary tuberculosis.

CASE I.

W. H., widower, aged 53, a commercial traveller, was admitted to Hampstead General Hospital on January 31st, 1922, with a history of four years' gradually increasing dyspnoea on exertion. There were also attacks of more acute dyspnoea, coming on chiefly at night, and lasting from half an hour to an hour. Pain was felt at the back of the shoulders and down the right arm, with a general feeling of tightness in the chest. This was also worse after exertion. He had a chronic slight cough, and at times suffered from palpitation. There had been no dysphagia, no oedema, and no haemoptysis. He did not think he had been losing weight.

Two days before admission he suddenly collapsed after getting up in the morning, and lost consciousness for three-quarters of an hour. On recovering he felt very weak, and complained of pain all over the chest; later he developed a troublesome cough with haemoptysis. He had always been healthy previously.

Condition on Admission.—The patient was a rather thin and anaemic man in a very weak condition. There was very little dyspnoea while at rest. There was a pulsating swelling immediately to the right of the sternum, having an area of about 2½ square inches, and considerable diminution of movement of the right chest. The percussion note was dull all over, back and front, while the breath sounds were very weak and bronchial. Vocal fremitus and resonance were increased. There were no adventitious sounds. The pulse rate was 70, and the rhythm equal and regular. The volume was moderate and tension poorly sustained. There was no inequality of the radial pulses. The cardiac apex was in the fifth space, 1 inch outside the nipple line, and was heaving in character. The heart sounds were normal, and were heard clearly over the swelling. The systolic pressure was 145 mm. and the diastolic 110. The left pupil was slightly larger than the right.

Progress.—The patient remained fairly comfortable in bed for over a month. The cough and haemoptysis continued. There were occasional slight attacks of dyspnoea. The only fresh sign that developed was a slight oedema of both forearms. The temperature varied from 98° to 101° F. On March 7th, at 5 a.m., free haemorrhage took place suddenly from the mouth, and the patient died in five minutes.

Post-mortem Examination.—A small mass of blood clot about the size of a large walnut was found immediately beneath the chest

wall in the region of the swelling noted before death. The left lung was large and emphysematous, and covered the left border of the heart and pericardium. About 12 oz. of dark yellow fluid were present in the right pleural cavity. The right lung appeared shrunken, was dark in colour, and felt solid. It was not possible to distinguish the lobes. The heart was small and appeared normal. The aorta about one and a half inches above its origin opened into an aneurysm about the size of a foetal head. It occupied what appeared to be the right lung, the remnant of which was stretched over the sac as a thin shell. The cavity of the aneurysm was full of laminated clot, which dropped out through the anterior wall of the sac, and was directly continuous with the walnut-like mass above mentioned. The aorta was atheromatous, and showed marked syphilitic arteritis.

CASE II.

Mrs. E. S., a widow, aged 50, employed as a cook, was admitted to hospital on March 12th, 1922, with a history of six months' illness of gradual onset. Her chief complaint was pain round the lower part of the chest, "crushing" in character, coming on directly after food. There was no vomiting, but considerable flatulence. The appetite was poor, and she did not sleep well. She had been getting much thinner recently. There had been no night sweats. There was slight dyspnoea on exertion. For the past month she had been bringing up blood by the mouth; she said she coughed up a little every day mixed with frothy sputum, the blood being sometimes bright in colour and sometimes dark, while at intervals of about a week she brought up larger quantities (about half a pint) in clots. These large quantities she thought she vomited, although solid food was never noted in the vomit.

Condition on Admission.—The patient was a wasted, anaemic woman. There was moderate enlargement of the terminal phalanges of the fingers. The temperature was 100° F., pulse rate 100, and respirations 24. On inspection of the chest there was definite flattening at the left apex with diminished expansion. On palpation there was slight pulsation below the left clavicle. The percussion note over the left apex and subclavicular region in front and over the left apex behind was dull. Towards the outer part of the dull area the breath sounds were diminished, and vocal fremitus and vocal resonance were less than normal, but internal to this loud cavernous breathing was heard. A few fine crepitations were present. The pulse was equal and regular, of small volume and moderate tension. The apex beat was in the fifth space within the nipple line, and there was no enlargement or displacement of the heart. The sounds were normal.

Progress.—For the first two days after admission the patient was continually bringing up clotted blood in small quantities. The blood appeared in the pharynx without effort, and was unmixed with sputum or food. There was a slight cough, but the blood did not seem to appear with the cough. No sputum was obtained. On the second day after admission she had a sudden and severe haemorrhage; she became blanched and almost pulseless at the wrist. However, she made a fair recovery from this attack, but the bleeding continued, and at times the blood was undoubtedly mixed with fluid stomach contents. Melaena was a marked feature in the case during this time. Pain in the upper left chest was also complained of. The temperature varied from 99° to 100° F. The patient gradually became weaker and more anaemic, and died quite suddenly, but without external haemorrhage, on March 19th, seven days after admission.

Post-mortem Examination.—An ulcer about 1½ inches by 1 inch in diameter was found in the oesophagus at the level of the bifurcation of the trachea. Through the base of this ulcer a mass of blood clot presented. The trachea was normal. The upper half of the upper lobe of the left lung was heavy, deep red in colour, felt solid, and was adherent to the thoracic wall. An incision showed a mass of dense laminated blood clot of a pale buff colour, around the periphery of which was stretched a thin layer of lung tissue. The mass was about the size of a large orange, and contained a cavity no larger than a walnut, which communicated with the arch of the aorta. The lumen of the aorta itself was scarcely altered. There was slight atheroma and syphilitic arteritis. The heart was small, and the valves were normal. The right lung was quite healthy. The intestines contained a considerable quantity of altered blood.

The way in which, in the first instance, a whole lung, and in the second instance a localized part of the lung, were converted into aneurysmal sacs is one of the most interesting features of these cases. The strength of the sac in each instance seemed to be in its dense lining of laminated clot. As regards the origin of the bleeding, in the first case it probably came from the remains of the bronchioles in communication with the sac, while in the second case it is possible that there was a double source of haemorrhage—from the lung and from the oesophagus. Another observation which is well exemplified is the remarkable pathological changes that can take place in the thoracic aorta without producing any change in the cardiac musculature. In the first case the heart was certainly displaced, and, clinically, appeared to be hypertrophied; but the *post-mortem* examination showed that in neither case was there any hypertrophy or dilatation.

Clinically, Case II presented an interesting problem in diagnosis. The history of progressive wasting, cough, and haemoptysis, together with the localization of the physical signs to the left apex, pointed to an apparently advanced case of chronic pulmonary tuberculosis with cavitation. The