as prominent as before treatment. Of the fifty cases re-examined, therefore, in only 10 per cent. had a cure resulted, using the term "cure" in the sense that the patient had been made free, and remained free, of symptoms and signs up to the time of the re-examination. In the remaining 90 per cent. marked objective evidence of varicose veins was again present, although in the majority of these symptomatic relief had been given.

NATURE OF THE "RECURRENCE"

The point next arises whether the veins found at the re-examination represented veins not obliterated by the treatment, fresh veins developing subsequent to the treatment, or veins originally obliterated which had again become patent. While there is no doubt that some of the "recurrences" represented incomplete original treatment or the development of further veins, a careful comparison of the condition found on re-examination with the original notes and diagrams made it obvious that, in the majority of cases, recanalization of obliterated veins had taken place. Without being able to give exact figures on this point we are satisfied that recanalization of varicose veins obliterated by injections is a frequent event and a common cause of recurrence. It is unlikely that recurrence is related to the solution used for injection, as the mode of action of all "sclerosing" solutions is probably similar (Patey²). In the present series the solution chiefly used was quinine and urethane, but recurrence was also observed after the use of the other solutions; it probably depends on the tendency of thrombosed veins to recanalize rather than on the particular solution used.

PRESENT POSITION OF TREATMENT FOR VARICOSE VEINS

In the literature, while there are many references to recurrence after injections, detailed analyses of the results of the treatment are not common, and such figures as there are vary. On the one hand, Sicard and Gaugier,³ in the 1931 edition of their book on the injection treatment of varicose veins, stated that, in their experience, only 4 per cent. of all patients treated returned for further treatment, and only 1 per cent. had true recurrences in the obliterated veins. On the other hand, Howard, Jackson, and Mahon¹ stated that recurrence was the rule following injections. Delater,⁴ in his recent book on diseases of veins, takes up an intermediate position, but, commenting on the fact that 20 to 25 per cent. of his hospital patients returned for further treatment within four years, he writes:

"Un variqueux guéri de ces dilatations par la fibrose ne doit donc jamais être abandonné, parce qu'il n'est pas guéri de son insuffisance veineuse."

Of significance also in this connexion is the appearance lately of papers advocating a combination of surgical treatment and injections as being less likely to be followed by recurrence (de Takats and Quillin,⁵ Wright⁶), and of papers analysing the pathological basis of recurrence (Theis⁷).

Considering the whole question of the injection treatment of varicose veins in the light of the late results it would seem that for most cases injection should be regarded as a useful means of palliation rather than as a cure, and that, as with most new forms of treatment, the original estimates of its value were too enthusiastic. It would be unwise, however, to go to the other extreme, because in spite of its limitations injection treatment, either alone or in combination with surgical measures, is probably for most patients with varicose veins still the best that can at present be offered. Moreover, recanalized veins seem to respond as readily to a further course of injections as the original veins.

But the limitations of injection treatment do indicate that finality on the question of the treatment of varicose veins is as yet far from having been reached. At present the tendency in many clinics is towards combining, where possible, surgical treatment—in the form of either ligature or excisions—with injections, both as a means of shortening the time of treatment and in the hope that the end-results will be better. This type of combined treatment is not new, having been advocated by Tavel⁸ as long ago as 1904, although it was never extensively adopted. In more recent years papers on the subject have been published by d'Allaines and Roseau⁹ in France, de Takats and Quillin⁵ in America, and Wright⁶ in this country. De Takats has recently analysed 200 cases treated in this way. At the Middlesex Hospital varicose vein clinic the present practice is to remove under local anaesthesia a segment of the main superficial vein whenever it can be demonstrated that pressure on it in the standing position controls the superior reflux, and to follow this up with injections to the peripheral veins. The immediate results are satisfactory, and the addition of the surgical procedure seems in most cases to shorten the length of treatment; but whether the end-results will be more satisfactory than those of pure injection treatment remains for the future to show.

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THE INCIDENCE OF PULMONARY DISEASE FOLLOWING EXPOSURE TO VESICANT AND ASPHYXIATING GASES

BY

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The following is an appreciation, a summary, and an analysis of 118 appeals heard by the War Pensions Appeal Board in Auckland during its mid-year session, 1932. The cases are those of men who appeared before the Board for the first time. Upwards of 100 other appeals by men who had previously been before the Board were heard at this session, but are not included in this review. I am indebted to the chairman, the Hon. Sir Walter Stringer, for his permission to use the official records of the Board in compiling this paper, and the facts and figures are wholly derived therefrom.

During my three years and 306 days of active service in the great war I was engaged as a regimental medical officer and ambulance officer with the New Zealand Division in the battle areas in Egypt, Anzac, and France, and can claim to have first-hand knowledge of the use of gas in war and of its effects on the exposed troops at the time of exposure. The vesicant properties of the so-called "mustard gas" were apparent in innumerable cases, its potency being such that it caused intense hyperaemia and even blistering through the thick khaki clothing. The morale of the men was such that only severe cases were evacuated, and the percentage evacuated would not bear any relation to the total number with demonstrable evidence of cutaneous or respiratory damage. Contributors to the literature on gas damage have differed widely as to the incidence of pulmonary fibrosis following exposure, but all agree that fibrosis is a possible sequela.

This contribution is the outcome of seven years of observation of gas-exposed men with the Appeal Board, and of thirteen years' service as honorary physician at the Auckland Hospital, during which time I have had many cases of acute respiratory disease through my wards. My experience forces me to the conclusion that persistent and acute respiratory disease is much commoner among ex-soldiers than civilians. I have endeavoured to make a comparison between exposed and non-exposed soldiers, and I have attached percentages published by Jacquelin and Mlle Konechowsky for purposes of comparison. Their observations were made at the Marthe Brande Dispensary for Chest Diseases, and seventy-three cases of gas-exposed men were investigated. No effort was made to compare the results with non-gas-exposed soldiers, and no *x*-ray investigations were made. In their clinical appreciations of the cases it seemed to me that they were too prone to attribute gross fibroid changes to direct gas damage. In my view the secondary infective factor must be held to be the chief cause of the gross fibroid changes that are most easily demonstrated by *x* rays. Their description of apical fibrosis, the direct result of gas damage, is inconsistent with my experience. They find that the incidence of pulmonary tuberculosis is high following gas exposure. Though I have previously expressed myself as being opposed to this view, I would now be inclined to suspend judgement until a further and more comprehensive comparison is made. While the Auckland Board recognized the high incidence of respiratory disease among soldiers in 1926, I did not publish until December, 1931 (*N.Z. Medical Journal*.)

Since then the need of intensive investigation of all cases was recognized if a contribution of any value was to be made to the literature on the subject. A comparison such as this between exposed and non-exposed troops is, I believe, unique. It should be borne in mind that the men examined are in no sense a cross-section of the N.Z.E.F., but are in reality selected cases in that they are men who have appealed, many of them solely, on the ground of disability as the result of gas damage. In my opinion it would be incorrect to deduce from these figures that a high percentage of exposed men develop pulmonary fibrosis and its allied sequelae. The factor of idiosyncrasy would seem to operate much as it does in miners' pneumoconiosis. I have been frequently struck by the absence of lung changes in men who must have had prolonged exposure or who were evacuated as "N.Y.D. gassed." Most of the cases evacuated would seem to have been damaged by phosgene principally, and that fact is frequently stated in the official records. My own impression is that prolonged exposure to a lower concentration of vesicant gas is a more potent factor in the production of fibrosis than short exposure to a high concentration. The latter type of case, of course, would predominate among the gas casualties.

PULMONARY FINDINGS

It is interesting to observe that the rate of progress of the lung changes varies greatly. Many men have complained of respiratory abnormalities from the termination of their service and have been pensioned as "bronchitis." Other men seem to have carried on without serious disability for eight to ten years, until the onset of asthma or the loss of immunity to bacterial infection brought them to the chronically "wet" bronchitic stage. It is noticeable that the manual worker has "cracked up" much sooner than the sedentary worker. The symptoms complained of are referable to the lungs and to the heart. They complain of incessant coughing and expectoration, most marked on rising in the morning. Some will give a history suggestive of bronchiectasis. They say that they sweat easily, particularly after coughing bouts. The asthmatics describe attacks of wheezing and of having to sit up at night. All stress their undue susceptibility to "colds." The next most frequent complaint is exhaustion. They say they tire easily, they cannot keep up with other men, and that exertion brings on attacks of coughing that end in vomiting. As soon as they are found out by employers they are discharged as being unfit to do the work. They

are dyspnoeic after slight exertion. Physical signs in the chest correspond largely to those discernible in emphysema—namely, hyper-resonance, loss of lung elasticity, with prolongation of expiration and increase in pitch associated with stray rhonchi and rales scattered over the lower half of the lung fields. Large bronchiectases are rare, and dense fibrosis causing percussion dullness is found mainly in cases where pneumonic and pleural complications have ensued. Blood-tinged sputum is fairly common, but large haemoptyses are rare in my experience. The heart invariably shows right-sided enlargement with gross epigastric pulsation. The superficial and deep cardiac dullness is obscured or absent owing to the overlapping lungs. It is difficult to be certain if there is accentuation of the pulmonary second sound at the base. The "effort syndrome" is of considerable interest. After having observed upwards of 400 of these cases I am convinced that there is a common type for them. The pulse rate accelerates rapidly and unduly after moderate exertion, but will be found to have returned to normal at the expiration of two minutes. A typical reading would be 78–140–80. I will refer to this later as the "right heart" type of effort syndrome, as opposed to the "left heart" type, where the pulse rate remains accelerated at the end of two minutes' rest. Coldness and blueness of the hands and feet is observed in advanced cases, but hypertrophic digital changes are most unusual.

In explanation of the above signs and symptoms I have supposed that vesicant gas has started a fine pulmonary fibrotic process that has resulted in a raised tension in the pulmonary artery and an undue susceptibility to respiratory infections and to pulmonary allergic phenomena. If there has been a minimum of secondary infective damage the lungs may appear to be actually "rarified"—that is, there is less density than normal—and this is seen particularly at the bases in the central areas. Investigation by means of lipiodol has not been extensively used in Auckland in post-gas-exposure chest conditions. I have not found that it revealed pathological conditions that could not have been surmised by clinical and ordinary *x*-ray examination. In one case a bronchopneumonia supervened in three days. The factor of secondary bacterial infection is a big one in all cases of pulmonary fibrosis following vesicant gas damage. The "dry" cases, particularly in sedentary workers, have very little disability. The intervention of bronchitis and asthma combined with manual work produces the highest disabilities most quickly from the point of view of assessment of pension. The British Ministry of Pensions does not accept a negative *x*-ray as excluding gas damage. It subscribes to the doctrine of "continuity of symptoms," and a history of chronic bronchitis is held to be a pensionable disability due to war damage. Unfortunately, *x*-ray evidence in early uncomplicated cases is largely negative. It is not nearly so helpful as in early pulmonary tuberculosis.

While it is recognized that 118 cases are an insufficient number on which to make a generalized statement, they provide an instructive comparison between men who had been in the gas areas and those whose service was confined to Anzac, Egypt, and Palestine, or who had had no front line service on any front. The two chief variants in this comparison would seem to be gas and climate. The latter is a difficult one to estimate. It would seem, on the face of it, that apart from gas one would expect pulmonary disorders to predominate in France. The actual percentages are 35.7 in France and 20.8 elsewhere. When the gas factor is taken into account this difference does not seem to be extreme. The dust, the summer clothing, and the cold of the Anzac blizzard and the Palestinian uplands are probably all factors in the relatively high rate among non-gas-exposed troops.

ANALYSIS

Of the 118 cases analysed seventy were in the gas areas and forty-eight were never exposed to vesicant gas at all; 35.4 per cent. had been wounded at least once and several on three separate occasions. Ten per cent. of those exposed were evacuated as gas casualties, and

50 per cent. claimed to have experienced respiratory troubles when in the gas areas. Their symptoms at the time varied from hoarseness, loss of voice, vomiting, and prolonged coughing to bronchitis, under which latter heading many were evacuated. Forty per cent. of the 118 had nervous disorders varying from minor disturbance such as tremor, exaggerated deep reflexes, insomnia, "night starting" and war dreams, up to neurasthenia of varying degrees, anxiety neurosis, psychasthenia, Parkinsonism, and insanity.

The first comparison I wish to make is as regards asthma and tuberculosis.

	Gas-exposed.	Non-gas-exposed.	Jacquelin's Figures.
Asthma ...	10%	4.2%	11%
Pulmonary T.B. ...	5.7%	6.2%	17%

It will be noticed that our figures for asthma are in close agreement with those of Jacquelin. As regards tuberculosis, the percentage is a little higher in the non-gas-exposed than in the gas area men. I have previously expressed the opinion that the immunity factor in pulmonary tuberculosis is a greater one than that of extraneous gas injury. I have also been of the opinion that Jacquelin's figures for pulmonary tuberculosis are too high, and that is certainly borne out by this comparison. At the same time it should be noted that cases in which pulmonary tuberculosis had developed would have had symptoms that would bring them to the Pensions Department for relief at a much earlier stage. There were two cases of syphilis, one under each heading. The one exposed to gas had clinical evidence of pulmonary fibroid changes, but lost his appeal on the ground of uncertainty of causation. There were likewise two cases of penetrating wounds of the thorax. One had developed pulmonary tuberculosis. The high incidence—60 per cent. by some observers—of pulmonary tuberculosis in penetrating chest wounds is well known.

The next comparison is between chest affections on service and in the post-war period. Pulmonary tuberculosis is not included in this comparison. •

	Gas-exposed.	Non-gas-exposed.	Jacquelin's Figures.
Chest trouble on service ...	35.7%	20.8%	—
Chest trouble post war ...	50.0%	18.7%	—
Pneumonia post war...	8.5%	Nil	—
Bronchitis post war...	34.3%	12.5%	19%

Chest trouble on service includes bronchitis and pneumonia only. Post-war chest trouble includes frequent and undue susceptibility to colds, constant coughing and spitting, morning cough and expectoration of the bronchiectasis type, and recurring bronchitis. As will be noted, no cases of pneumonia occurred among the non-gassed men. The discrepancy with Jacquelin's figures as regards bronchitis in the post-war period is a big one. I can only conclude that our classification is a less rigid one than his. We have included cases under the heading of bronchitis that he has classified under minor respiratory disorders.

The next comparison is between clinical and α -ray evidence of pulmonary fibrosis in exposed and non-exposed men.

	Gas-exposed.	Non-gas-exposed.	Jacquelin's Figures.
Clinical evidence of pulmonary fibrosis	48%	12.5%	44%
X-ray evidence of pulmonary fibrosis	25.7%	7.1%	—

The clinical evidence of pulmonary fibrosis is based on: (1) Evidence of constant or frequently recurring respiratory infection. Outside medical and reliable lay evidence of this symptom is given due consideration, as is also reliable evidence of the occurrence of attacks of asthma. Special efforts are made to exclude paranasal infection. In this series there were six cases of "blocked nose" amongst the gas-exposed men and two in the non-gas-exposed. Most of them had been operated on at some time during or since the war, and in two cases sinus trouble was still present. The majority were apparently cases of septal deviation without sinus infection. (2) Clinical evidence of loss of lung elasticity associated with hyper-resonance. (3) Evidence of right heart enlargement. This sign is a valuable one; epigastric pulsation was constantly present in all cases of pulmonary fibrosis occurring in both series. (4) Obliteration of cardiac dullness.

Jacquelin's figures are in fair agreement when it is considered that our cases were examined three years later than his.

The policy of the Auckland Board at present is to refuse responsibility for cases with a negative α -ray, unless there is continuous and reliable evidence of chest trouble since the war. In the Board's experience it is useless to look for α -ray changes unless the clinical evidence is well established. For this reason in 23 per cent. of cases where clinical evidence could be said to exist no α -ray investigation was made. The following figures show the relative percentages of pulmonary fibrosis in the two groups. Pulmonary tuberculosis and syphilis are excluded, but post-pneumonic fibrosis is included.

	Gas-exposed.*	Non-gas-exposed.
Pulmonary fibrosis ...	32.8%	8.3%

The above figures include only those cases where the disability was sufficient to justify the awarding of a pension.

The following comparison shows the relative incidence of so-called "effort syndrome" symptoms.

	Gas-exposed.	Non-gas-exposed.
Effort syndrome ...	42.8%	27.1%

By effort syndrome due to gas damage is understood impaired capacity for sustained exertion, as well as the exercise tolerance test showing the right heart type of syndrome. It has been held by some observers that these "effort syndrome" returned soldiers should be regarded as displaying the sequelae of adrenal exhaustion or the effect of adrenal over-stimulation during their front-line service. In my opinion this explanation may hold for some of them, but the association of lung damage and chest infections is so constant that precedence must be given to the gas explanation. It is possible, and probable, in many of the cases of so-called "D.A.H.," that the "adrenal" factor has been predominant.

As regards the impaired effort syndrome in the non-gas-exposed, it is of the left heart type with the pulse rate remaining accelerated at the expiry of two minutes' rest. In the gas-exposed men the abnormal effort syndrome is only so attributed when all other organic troubles have been excluded. The non-gas-exposed men included impaired effort syndrome from all causes.

Competent authority in New Zealand states that the death rate from respiratory disease amongst returned soldiers is 42.4 per cent. as against 32 per cent. for the civilian population, an increase of just over 30 per cent. These figures include pulmonary tuberculosis, and make no differentiation between gas-exposed and non-gas-exposed men. They also do not include men dying from pneumonia in the post-war period who had been exposed to vesicant gas but had not been sufficiently incapacitated as to be pensionable. If our view is correct there must be an appreciable percentage of men whose progressive lung changes had not then, and have not now, reached a pensionable stage, but who would be unduly susceptible to death by pneumonia at any period since the war. It is thus evident that, if such statistics could be obtained and analysed on the lines of the figures given in this paper, it is possible that the contrast would be even more striking.

One soldier, for instance, who had been over two years "on the fire step" in France, was twice evacuated for "bronchitis" while the Division was in the Messines area. He had had continual "colds," as he expressed it, since the war, and was convalescing from his third attack of bronchopneumonia when he was seen by the Board. This man had never applied for a pension, and had never been accepted as a pensionable disability for his chest, although α -ray and clinical investigation showed generalized fibroid changes in the lungs. If this man had died in one of his major attacks he would not appear as a possible post-gas casualty in the present figures.

While the published opinions of observers of the calibre of Messrs. Fenton and Burrell cannot be disregarded, it is important to know what special facilities they had for observation and investigation of a large number of cases

* Fibrosis occurring here was associated with gas exposure, but not necessarily directly due to it.

in recent years. As against their opinion must be placed the conclusions of trained observers such as x-ray specialists, clinicians such as Jacquelin, and, least of all, the Auckland records of the War Pensions Appeal Board. I am tempted to believe that the opinion that fibrosis does not occur to any extent following vesicant gas damage has been formed at a stage when the sequelae had not had sufficient time to manifest themselves fully. In recent years the Board has had to adjudicate on a number of cases of death from pneumonia in non-pensioned middle-aged soldiers. Their deliberations would have been greatly assisted by a competent post-mortem investigation.

As regards treatment, the main indication is the avoidance of infection. Pulmonary fibrosis can be subdivided under two headings, wet and dry, according to the sufferers' immunity to catarrhal organisms. As soon as immunity is lost their disability increases enormously, and bronchitis and asthma are much more frequent. The moist coastal climate in the northern areas of New Zealand is not nearly so favourable as the drier inland pumice country. We are fortunate in being able to obtain a reliable stock catarrhal vaccine in Auckland, and in my experience benefit is obtained by its administration, preferably towards the end of the summer.

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THE "CLOVER LEAF" SLING IN PARALYSIS OF THE SERRATUS MAGNUS

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The principle of rest followed by graduated exercise, starting from the zero point and increasing slowly until the muscle moves through its maximum range, has been ably put forth by Mackenzie¹ as the rational form of therapy for paralysed muscles. In the treatment of any paralysed muscle, then, our primary object is to ascertain the position of physiological rest and to maintain it.

ANATOMY

With the need for increased function of the arm as we ascend the animal scale, the scapula loses its connexion with the skull, thus becoming mobile, necessitating a further differentiation and rearrangement of the cervical muscles to control its movement. Complete usefulness of the arm depends not only on the proper function of the muscles inserted into the humerus and acting on the shoulder-joint proper, but also on the muscles arising from the trunk and inserted into the scapula, either stabilizing it or moving it through the acromio-clavicular and the sterno-clavicular articulations. This applies particularly to the ability to raise the arm above the level of the shoulder. To facilitate this the superficial shoulder-girdle muscles—the sternomastoid and the trapezius—have been evolved from the visceral musculature of the gills and from the metameric musculature of the second, third, and fourth cervical segments, while the deeper shoulder-girdle group—the levator scapulae, the two rhomboidei, and the serratus magnus—has been derived from the ventrolateral division of the lower fifth and sixth cervical myotomes. The serratus magnus has moved its point of origin extensively, finally dissociating itself completely from the levator scapulae, but carrying with it its original nerve supply from the cervical region. The other shoulder muscles arise in the blastema of the limb bud, the pectoral group spreading out over the trunk anteriorly and the latissimus dorsi posteriorly, the teres major becoming separated from the latter, to be

attached to the lateral border of the scapula. The deltoid and its offshoot the teres minor, with the supra- and infra-spinati, develop together, while the subscapularis from the first prefers its own company.

The two important muscles controlling the scapula, therefore, are the trapezius and the serratus magnus, and isolated paralyses of these muscles may occur. It is doubtful whether isolated lesions of the two rhomboidei or the levator scapulae are often recognized clinically. The serratus magnus springs from the lateral chest wall and is inserted into the undersurface of the vertebral border of the scapula, and its function is to pull the scapula forwards over the lateral chest wall and also to keep this bone close to the trunk, although Frazer² states that the upper fibres of the latissimus dorsi are responsible for this. The nerve supply is from the fifth, sixth, and seventh cervical roots. This nerve does not enter into the formation of the brachial plexus; thus, in injury to the superior trunk, the serratus magnus may escape, except in avulsion of the cervical roots. Its upper two roots pierce the scalenus medius, while the lower one passes in front of it. They unite as the long thoracic nerve, and pass out of the posterior triangle of the neck on to the outer surface of the serratus magnus behind the axillary vessels and nerves, occupying a position on the inner wall of the axilla and the lateral chest wall.

AETIOLOGY

The signs and symptoms of isolated paralysis of the serratus magnus have been well summarized by Tournay and Kraus.³ The two outstanding signs are the difficulty in raising the arm above a right angle and the undue prominence of the scapula. They tabulate a dozen other minor signs. From the standpoint of aetiology, the patients fall into two groups—toxic and traumatic. In the first group there has been some antecedent or concomitant infection, such as measles, puerperal fever, influenza, or broncho-pneumonia. In the other group the nerve is presumed to have been subjected to injury or pressure in some part of its course. Violent contraction of the scalenus medius, as in swimming or playing tennis, carrying weights causing traction on the shoulder, anomalies of the first rib, enlarged cervical or axillary lymph nodes, and pressure in the axilla have all been incriminated. If the first is a factor one wonders why the rhomboidei are not more often involved, for the posterior scapular nerve also arises from the fifth cervical root and pierces the scalenus medius. The right arm is usually affected. In the two patients under observation this was the case. In the first one, playing tennis may have been the factor, although he also gave a history of having had enlarged cervical glands which had receded completely. In the other case the aetiology was not demonstrable. The patient was a girl 19 years of age, in good general health, giving no history of previous infections. The evidence for trauma was inconclusive. She swam and played tennis moderately, and had suffered no accident. She was a stenographer, using a typewriter and pulling out drawers in a filing cabinet, but there was no evidence of undue exertion. She merely noticed that her arm and shoulder ached, that she was unable to raise her arm above her shoulder, and that her shoulder-blade was prominent. There was no tenderness along the course of the nerve. X-ray examination of the cervical region revealed no abnormalities. Macgregor⁴ suggests the analogy between this condition and facial paralysis of the peripheral type.

The position of physiological rest for the serratus magnus is assumed when the hand of the affected side is carried across the chest and placed on the opposite shoulder. To maintain this position Mackenzie states that the "collar and cuff" sling is satisfactory, and he shows illustrations of good results following its use. It