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Terror Weapons: The British Experience of Gas and Its Treatment in the First World War

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Abstract

Chemical weapons accounted for only 1 per cent of the 750,000 British troops killed in the First World War and yet caused disproportionate casualties (estimated at 180,100). The considerable investment in the development of new toxins and methods of delivery was designed to maintain the elements of surprise and uncertainty as these accentuated their psychological effect. Soldiers were continually challenged on the battlefield by combinations of different types of agent designed to undermine their confidence in respirators, disorientate them, and erode their morale. At first, army doctors practised defensive medicine, invaliding their patients for protracted periods to the UK or base hospitals. By 1917, progressive study of the physical and psychological effects of different types of toxin allowed physicians to design new management strategies. Borrowing ideas from shell shock, specialist units were set up closer to the front line and medical officers taught to identify crucial points in the course of illness to accelerate recovery times and forestall the accretion of psychosomatic symptoms.

Keywords

First World War; gas; chemical weapons; casualties; shell shock; treatment

'I was terrified of gas, to tell you the truth,' recalled Private John Hall of the Machine Gun Corps, adding, 'I was more frightened with gas than I was with shell fire.'1 Hervey Allen, a US Army infantry officer, recalled the panic often inspired by the threat of chemical weapons and observed, 'gas shock was as frequent as shell shock'.2 The psychological impact of these toxins was confirmed by Lieutenant Colonel C. Gordon Douglas, a physiologist and specialist gas officer, who concluded that 'the particular value of the poison [mustard gas] is to be found in its remarkable casualty producing power as opposed to its killing power'.3 The capacity of gas to inspire fear was apparent from its first large-scale use on the Western Front. At Langemarck, on 22 April 1915, the release of 150 tons of chlorine from 6,000 cylinders caused widespread panic. The chaotic retreat of two French divisions, 87th Territorial and 45th Algerian, opened a 4 mile gap in the front line.4 As these troops had no protective equipment or any training in gas warfare,5 it was scarcely surprising that they fled when confronted with a suffocating, greenish-yellow cloud. In September 1915,

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when the British released chlorine in retaliation,6 similar effects were observed of German troops at Loos:

> A German officer in this sector remarked that as soon as the gas entered his trench, he lost all control of his men, a panic ensued and he was unable to keep them in the front line. He said that, without the gas, we should have had no earthly chance of taking the trenches.7

Uncontrolled anxiety during a gas attack could cause men to tear off their protective masks,8 or act ‘as though they had temporarily lost their reason’.9 Later in the war Charles Wilson, a regimental medical officer with the Royal Fusiliers, argued that mustard gas had ‘partly usurped the role of high explosive in bringing to a head a natural unfitness for war, or less commonly in undermining fitness sapped by exceptional stress in the field’.10 Terms such as ‘gas hysteria’ and ‘gas neurosis’ were coined to describe enduring somatic symptoms once physical lesions had healed.11 Hulbert thought ‘gas neurosis’ akin to shell shock of a non-concussive type because the severity of symptoms bore no relation to ‘the amount of gas inhaled’ but arose ‘in proportion to the individual’s mental and emotional make-up and instability’.12

While the initial psychological impact of gas was explicable in terms of surprise and lack of preparedness, its enduring effectiveness as a terror weapon requires explanation. Almost 60 per cent of deaths in the First World War were a result of artillery and trench mortars;13 by comparison, gas killed few troops. Furthermore, most soldiers exposed to chemical weapons survived and made a good recovery.14 In a post-war study Brigadier Augustin Prentiss of the American Chemical Warfare Service estimated that only 4.3 per cent of gas casualties died, compared with 24 per cent of other types of battlefield injury.15 From mid-1916, respirators offered troops reliable protection against chlorine and phosgene. Yet gas remained among the most feared weapons of the war and continued to exercise a powerful hold over the popular imagination such that anti-war campaigners focused on its use to mobilize support for their cause.

Edward Spiers and L.F. Haber have documented the scientific resources deployed to devise and identify new chemical weapons, together with innovative ways of manufacturing and delivering these toxins.16 In addition, Donald Richter has explored the heroic efforts devoted to finding protective measures and training soldiers in their use.17 Helen McCartney discovered that witnessing the effects of chlorine was a defining moment for the Territorials of the Liverpool Scottish as it brought home the horrors and inhumanity of war.18 In his study of morale in the German and British Armies, Alexander Watson argued that gas created uncertainty: unlike shrapnel, it killed from the inside, eroding a soldier’s sense of control, while raising the terrifying fear of being suffocated.19 As regards the treatment of gas casualties, Mark Harrison has explored the evolution of their management within the provision of medical care in France.20 Spiers has also looked at the narratives of gas in the post-war period and how these fed into popular culture and the political debate about the future use of chemical weapons.21 However, with the exception of Tim Cook’s study of Canadian gas services,22 less research has focused on the psychological impact of gassing. This paper explores how different groups of soldiers responded, how symptoms developed over time, and the investigations conducted by medical officers to improve diagnosis and

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treatment. It analyses the effectiveness of the managerial strategy of the British Expeditionary Force (BEF) to reduce the amount of time that gassed soldiers spent in hospital and increase the proportion that returned to active duty.

I Physical Effects of Gas

Although chlorine was readily detected by its pungent odour and yellow-green clouds, phosgene was more difficult to identify, being colourless and having the smell of freshly cut hay. In December 1915, for example, the Germans introduced phosgene, which was six times more potent than chlorine and could be inhaled in fatal doses without the coughing and discomfort associated with chlorine.23 Furthermore, the symptoms of phosgene could be delayed for several hours, making immediate diagnosis problematic. Indeed, it was estimated to have caused 85 per cent of all deaths from chemical weapons during the First World War.24 To distinguish between the unpleasant (tear gas and the chlorarsines) and the lethal (chlorine, phosgene, and mustard gas) took nerve and training. Indeed, chlorarsines caused short-term but intense respiratory distress designed to disable temporarily but also to terrify.25 Colonel A. Bertram Soltau, consultant physician in France for gas cases, emphasized the importance of chemical weapons in the genesis of ‘nervous disorders’: there ‘is nothing’, he argued, ‘probably more liable to cause panic than the idea of being choked … the dread of being slowly strangled’.26

The surprise element, so important for gas, was maintained throughout the war by the continual development of new toxins and delivery methods.27 When outlining British policy in June 1916, Field Marshal John French had argued that it was ‘essential that the nature of the gases discharged from cylinders and in projectiles should be varied from time to time in order that the enemy’s protective measures may be rendered as difficult as possible’.28 Planners deliberately exploited ignorance and fear of chemical weapons. For example, an infantry assault on the British First Army at Vermelles on 27 April 1916 was preceded by lachrymatory gas shells and clouds of harmless smoke. Before the second attack, an hour later, lethal chlorine was released, designed to surprise soldiers who had removed their respirators.29 Used against the British from July 1916 onwards, the gas shell enabled a range of toxins to be delivered rapidly without warning. In addition the British produced the Livens projector, which, according to captured German troops, was the most demoralizing weapon the Allies possessed, making life ‘utterly unbearable’.30 First used at the battle of Arras in April 1917, the Livens projector propelled a drum containing 30 lb of chemical (usually phosgene) over a range of 1,700 yards.31 In batteries of 25 they delivered a massive quantity of agent with accuracy and little warning. In response the Germans devised a ‘Gaswerfer’, which gave a high concentration of lethal gas by firing hundreds of large phosgene projectiles over a limited front.32

In 1976 Ludwig Haber estimated that 6,060 soldiers of the BEF had died as a result of gas (about 1 per cent) and a further 180,100 (3.3 per cent) had been injured.33 Official statistics compiled by Mitchell and Smith recorded 185,706 gas casualties admitted to hospitals in France, with 5,899 deaths.34 According to this data, admissions rose steadily throughout the war; those for 1918 (113,764) were almost double those for 1915–17 (71,942).35 However, this temporal rise was primarily a function of increased use, the quantity of chemical

consumed having risen from 3,870 tons in 1915 to 65,160 tons in 1918.36 Greater numbers of patients did not represent a widespread failure of treatment or protective measures. In fact, deaths fell as a proportion of admissions from 4.5(3,226) for 1915–17 to 2.3 (2,673) in the last year of the war.37 Reduced mortality was a consequence of better quality respirators and improved clinical techniques, notably oxygen therapy.38

At first British medical authorities had struggled to contain the problem. Not only did numbers killed by cloud gas rise, death rates at casualty clearing stations (CCSs) rose progressively from 3.6 per cent in December 1915 to 19.6 per cent by August 1916 (Table 1). This data, collected by Major T.R. Elliott and Captain C.G Douglas in an attempt to understand the impact of chemical weapons, established that gas could be a deadly and potent weapon. The rising mortality that they documented was a consequence of increased use of phosgene, difficulties in developing an effective respirator, and inadequate training of medical staff in diagnosis and treatment. Indeed, the statistics related to a time when Royal Army Medical Corps (RAMC) physicians believed that most gassed patients would benefit from bleeding (venesection) to reduce strain on the heart and drain fluid from the lungs.39 Not until 1917 was it established that the procedure was actually harmful in cases characterized by a rapid and weak pulse.40

If worn correctly, the small box respirator, introduced by the British late in 1916 (though not fully issued until January 1917),41 provided reliable protection against chlorine and phosgene.42 Largely for this reason, in July 1917 the Germans introduced mustard gas, a vesicant that burned the skin. Known as ‘yellow cross’ from the shell markings, the toxin immediately produced a flood of admissions: 160,970 soldiers presented at CCSs between 21 July and 23 November 1917, of whom 1,859 died.43 It was estimated that 75 per cent had been exposed to mustard gas.44 Deaths initially occurred because the toxin had delayed effects.45 The smell of the gas was ‘not very noticeable and… the immediate effect on troops exposed to it’, Brigadier K. Wigram reported, was ‘only a slight irritation of the nose and throat’, so that most troops had been slow to realize that they had been poisoned.46 Mustard gas achieved its greatest effect in the months immediately after its introduction because ‘the novelty of the condition, the multiplicity of the symptoms, and the entire absence of knowledge as to possible after-effects naturally led to the condition being overestimated’.47 Fear inspired by mustard gas was heightened by its capacity to cause loss of sight. Although most blinded servicemen recovered, the acute photophobia, conjunctivitis, and oedema of the eyelids forced soldiers to close their eyes, ‘so much so in fact that when some of the milder cases were evacuated each man had to be led like a blind man by an orderly to the ambulance car’.48 Such images struck a cultural chord and remain among the iconic symbols of the First World War.49

Data collected by Douglas towards the end of the war, and subsequently published in the official history,50 demonstrated that mortality rates were determined by the nature of the toxin, delivery method, and effectiveness of protective measures (Table 2). Although cylinder gas was subject to the vagaries of the prevailing wind, its use corresponded with a time of inadequate respirators and little anti-gas training. As a result, it generated high mortality rates. By contrast, mustard gas, deployed after the issue of the box respirator when seasoned troops had habituated to the threat of chemical weapons, led to much lower death

rates, even though its delivery was far more precise. Consequently, death rates were as low as 2.5 per 100 casualties for mustard gas and as high as 24.0 for troops exposed to chlorine and phosgene in the period before the introduction of the small box respirator. Interestingly, phosgene delivered by gas shell caused more casualties, but a significantly lower death rate, than cloud gas. The reduced morality was a consequence of greatly improved protective measures, confidence in their use, and the growing provision of specialist treatment. The high death rate for projector attacks related to their capacity to deliver high concentrations of deadly agent with minimal warning.

The physical effects of poison gas, though sometimes terrifying to observe, were in the majority of cases not fatal and most soldiers made a good recovery.51 If, however, a man received a lethal dose of chlorine or phosgene, death commonly came within two days.52 By comparison, mustard gas, first used against British troops on the night of 12/13 July 1917 at Ypres, was designed to disable rather than kill.53 It was estimated that of the 180,100 British chemical casualties, 120,000 had been subjected to mustard gas.54 Provided a soldier wore a gas mask correctly, his respiratory system was protected, but woollen uniforms offered no defence against blistering of the skin.55 Research showed that a thick suit coated with oxidized oils and resins neutralized the vesicant effects of mustard gas, but such a garment was ‘hot and uncomfortable to wear, and for a fully equipped soldier marching and fighting in such clothing would be impossible’.56 Although gas capes were issued to British troops during the Second World War, no corresponding protection was forthcoming in 1917–18. American medics and gunners were, however, issued with an ‘anti-gas suit’ made of cotton sheeting impregnated with linseed or vegetable drying oil.57

II Psychological Effects

Even before the mass use of chemical weapons on the battlefield, the ‘subjective effect’ of toxins on an individual’s mind had been recorded. Early in 1915 British scientists tested the possible use of ethyl iodoacetate (a lachrymator given the code name ‘South Kensington’ after the experimental work conducted at Imperial College of Science and Technology). A number of army officers from Chatham garrison were invited to attend field trials: ‘One of them, who was stationed at least 50 yards up wind from the point of burst, immediately left the trench showing every sign of great mental disturbance and stating that he felt very ill.’58 It was established that he could not have inhaled any of the vapour and yet had been deeply affected by the experience.

Douglas observed that, although not primarily designed to inspire terror, the ‘violent irritant or choking sensations’ of chlorine and phosgene had the capacity to undermine the resolve of all but the most resolute soldier.59 Specialist medical officers increasingly recognized the importance of gas as a psychological weapon. Captain H.W. Barber, who treated mustard gas cases at No. 25 General Hospital, argued that the ‘sudden shock’ of being gassed often caused as many symptoms as ‘any toxic property of the gas itself’.60 Writing in spring 1917, Lieutenant Colonel S.L. Cummins, adviser in pathology to the British armies in France, concluded that any division subjected to a series of gas attacks in close succession was likely to exhibit a significant drop in morale,61 while Charles Wilson, a regimental

medical officer, believed that ‘the majority of men who left the front line in 1917 “gassed” were frankly frightened’.62

The capacity of poison gas to inspire strong emotion led to a range of unwanted outcomes: panic even when protected by a respirator, the misinterpretation of harmless sounds and smells and taking evasive action, soldiers reporting sick when actually well, and doctors referring mild or transient cases of gassing for lengthy treatment in base hospitals. Panic is defined as precipitate and unreasoning behaviour not likely to serve the interests of the subject; it often involves actual or attempted physical flight.63 Captain A.J. Waugh, medical officer to the 1st North Staffordshires, reported such a case when his battalion was exposed to cloud gas in May 1916: ‘a few men lost their heads, took off their [anti-gas] helmets and ran back, being severely gassed in consequence’.64

Examples of troops misinterpreting harmless visual and olfactory stimuli were common and revealed the power of gas to disrupt military routine and discipline. Lieutenant G.L. Grant, medical officer of the London Scottish, treated large numbers of officers and men in September 1915 who believed they had been gassed. None had any objective signs of toxic exposure and all responded miraculously to a placebo.65 In February 1918 a soldier in a working party of 1/22nd London Regiment felt swelling and soreness of his throat and reported that he had been gassed. Although no shelling had taken place and no one had observed any signs of gas, fear swept through the unit and within a few hours 67 of the 105 men had been evacuated to an advanced dressing station as casualties,66 where some ‘acted as though they had temporarily lost their reason’.67 No organic cause could be discovered and the fact that no officer reported any ill effects suggested that this was an example of contagious anxiety. Similarly, a group of US machine-gunners became convinced that their food had been contaminated by toxins from a shell that had exploded nearby. Presenting to a nearby aid post, they complained of stomach pains, and some had even vomited. Doctors could find no evidence of exposure to gas and they were treated with bicarbonate of soda.68

Diphenyl chlorarsine (‘blue cross’), a nasal irritant and vomiting agent first used against the British in July 1917, caused short-term incapacity: sometimes ‘the pain and discomfort is sufficient to cause a man to lose his mental control for a short time’.69 Although the toxic effects were temporary, servicemen often continued to experience symptoms after the poison had worn off. Five soldiers were examined by Captain C.D. Christie three days after they had been exposed to chlorarsine gas. They complained of ‘extreme weakness and inability to use all of their extremities’. Christie observed that ‘it is very hard to reconcile the bizarre nature and distribution of the neurological findings on any anatomical or physiological basis’, though he believed the symptoms to be ‘genuine’,70 which suggested an unconscious mechanism rather than malingering.

III Adaptation and Protective Measures

Although gas masks saved lives by offering reliable protection against the inhalation of toxins, in themselves they were also a source of anxiety for both wearers and onlookers. Captain G. Donaldson of the 2/7th Royal Warwickshire Regiment, writing home in July 1916, observed: ‘We had our gas helmets on. It was like an appalling night-mare as you look

like some horrible kind of demon or goblin in these masks.'71 When 'smoke and tube helmets' were first introduced in autumn 1915, some units discarded them, having misinterpreted the smell of neutralizing chemical with which they were impregnated for that of gas; the practice helmets that they had worn earlier had little or no odour.72

Habituation and the adoption of coping strategies were hampered by continual refinements in chemical weapon technology. Each toxin had specific properties, demanding different protective measures and different forms of treatment. Knowledge, even among the medical corps, remained inconsistent. As late as April 1918 Douglas observed, 'I really believe that nearly all medical officers are terrified of the mere mention of gas poisoning,'73 and a month earlier had acknowledged that 'the majority of medical officers' in France could not accurately diagnose which gas a patient had been exposed to on the basis of its physical effects.74 Captain W.J. Forshaw, based at No. 2 Australian General Hospital, wrote of mustard gas in 1917: 'many regimental medical officers have no knowledge of the after effects and receive no information and scanty supplies of material for treatment'.75 Doctors untrained in the effects of gas practised defensive medicine and referred patients to base hospitals whether or not this caution was needed.

Knowledge can, but does not necessarily, serve as an effective defence against irrational fear. 76 However, mastering protective measures and diagnosing the differences between different types of gas took considerable training.77 The use of phosgene against British troops on 19 December 1915 prompted the setting up of anti-gas schools to prepare soldiers for the hazards of chemical warfare, and from March 1916 instruction in anti-gas measures to recently arrived drafts was given at Étaples, Rouen, and Havre. In September an anti-gas school also opened at Calais and in the following year others at Boulogne and Abbeville.78 Soldiers were exposed to an hour of cloud gas to give them confidence in their mask, and then exposed to 30 seconds of tear gas to give them a fright.79 Contemporary reports conflicted on the effectiveness of this training. Some servicemen reported a harsh realism. Private Frank Bass of the 1st Battalion, Cambridgeshire Regiment, wrote of his time at Étaples in September 1916: 'Lecture on gas. Officer lecturing had been two years here and through two gas attacks. Callousness of lecturers shocks us.'80 Many gas instruction officers felt devalued and, despite the dangers of chemical weapons, found soldiers unmotivated by the subject. A.E. Hodgkin, chemical adviser to the Fifth Army, recalled that many slept through his lectures unless compelled by the cold to stay awake: 'never, never will the mystery of gas warfare penetrate the brain of the regular soldier'.81 More effective, perhaps, was the experience of seeing fellow soldiers poisoned by gas. Ernst Jünger recalled that sight of comrades 'groaning and retching while their eyes watered' taught him 'never to go anywhere without my gas mask, having previously, incredibly foolishly, often left it behind in my dugout, and used its case – like a botanist – as a container for sandwiches'.82

However, contemporary accounts suggested that well-disciplined and experienced troops sustained lower rates of gas casualties than battalions newly arrived at the front.83 Douglas observed in March 1918:

> We have admittedly to deal with a large psychic element in very mild cases of gas poisoning, and this feature is naturally more prominent in the case of troops who

> have only a limited experience of gas shelling. Even with such experienced troops of our own, we have to contend with this difficulty – hence instructions to detain doubtful cases of gas poisoning in medical units within the army area until diagnosis is certain.84

The official history reported that when exposed to chlorine at Bellewaarde Ridge on 24 May 1915, soldiers of the 5th Border Regiment, attached to 10th Brigade, showed no outward concern. Many were miners accustomed to the dangers of gas, and their example did much to 'fortify the confidence of other troops'.85 Such variations in responses to chemical attacks confirmed that some units habituated to the threats; the question remains whether those battalions that accommodated more readily had been better prepared in terms of training and selection for the demands of trench warfare. No studies were undertaken to test the value of anti-gas training and the impact of actual exposure on subsequent performance.

IV Management Strategies

The management of gas casualties (that is, systems introduced to direct the flow of patients from the battlefield through various treatment processes to duty or discharge from the forces) evolved during the conflict as a small number of specialist physicians acquired technical understanding. C.G. Douglas was a key player in the BEF's strategy to combat the effects of gas.86 A physiologist and fellow of St John's College, Oxford, he had volunteered for military service in 1914 and was sent to France, where, in May 1915 at the suggestion of J.S. Haldane,87 he was transferred from general medical duties to investigate the immediate effects of chlorine on front-line soldiers.88 Douglas had conducted research into respiratory physiology under Haldane's guidance and was the obvious person to study the pathology of chemical weapons. He visited the sites of gas attacks and studied casualties in detail, building up a comprehensive understanding of their diagnosis and treatment. Indeed, medical officers in command of CCSs were ordered to inform Douglas whenever severely gassed cases were admitted to their units so that he could make an immediate examination. 89 Awarded a Military Cross in 1916 and four mentions in dispatches, Douglas rose to the rank of lieutenant colonel in 1918 and was responsible for drafting the British army's official policy on the effects of gas and their treatment. On being demobilized from the forces, he returned to his Oxford laboratory and a distinguished career of academic research in which he collaborated with J.G. Priestley to write a textbook, *Human Physiology*.90 His precision, attention to detail, and personal courage were in no small measure responsible for the accretion of reliable data on which to base practice and policy.

After the first major use of gas in 1915, and without an obvious treatment apart from bed rest, the British medical service in France adopted a defensive policy: the transfer of gas casualties to the UK as quickly as possible.91 This management approach became deeply embedded in RAMC culture, despite mounting evidence to suggest that it was often inappropriate and inefficient.92 The general medical strategy of 1915–16 was to build up a network of CCSs with a range of expertise to take the pressure off base hospitals during offensives.93 In terms of gas casualties, the CCSs were designed to filter out mild cases to prevent them overcrowding treatment facilities in the rear, but because doctors in these units

had only rudimentary knowledge of chemical weapons, they referred the vast majority of such patients to base hospitals.

By dramatically increasing the number of casualties, the use of mustard gas in July 1917 (Table 2) forced the British to re-evaluate the way that servicemen were treated. Early in 1918 Douglas discovered that 58 per cent of gas casualties admitted to all base hospitals in France between 1 July and 31 October 1917 had been evacuated to the UK. Of those that had remained in France, only 23.3 per cent had returned to active duty by the beginning of November, while 17.5 per cent were still being treated in base hospitals or convalescent depots.94 The management of shell shock provided a model that could be adapted for gassed patients. In December 1916 forward psychiatric units had been set up in CCSs to offer rapid treatment and prevent evacuation to the rear or the UK.95 Faced with a flood of mustard gas cases, the BEF adopted a similar strategy. Plans to open a specialist gas hospital for each of the armies in France were abandoned, ‘as the object is to bring cases under treatment as early as possible’.96 As with shell shock, specialist officers were deployed to undertake diagnosis, and regimental medical officers were instructed to label gassed soldiers as ‘Not Yet Diagnosed Gas’ to pre-empt premature evacuation to base hospitals.

In practice the new policy proved difficult to implement. As late as March 1918, Douglas bemoaned ‘the tendency to send gas casualties, even when they are very slight, over to England, and this of course implies very heavy wastage’.97 In September 1918 Colonel Elliott was horrified to discover that mustard gas cases were still being transferred to the UK, and only 11 per cent had returned to duty with the BEF after five months of treatment. 98 Not only did this policy extend the period that soldiers remained patients, clinical studies conducted towards the end of 1916 showed that excessive periods of treatment could arrest a natural recovery process as new clusters of functional symptoms developed. By autumn 1918, surveys had shown that 70 per cent of mustard gas casualties treated in CCSs and other lines of communication units could be returned to duty within eight weeks of exposure.99

V Treatment Tactics

The first chlorine attacks put doctors in a difficult position. Few had any knowledge of how to treat the toxin, and medicine could offer little to counteract severe pulmonary damage.100 As a result, military physicians took an exceptionally precautionary approach to any case of poisoning. Great emphasis was placed on rest, and Sir Arthur Sloggett, the director general of army medical services, ordered that for a minimum of two days ‘no casualty should be allowed to leave his bed or stretcher for any purpose whatever’.101 Severe cases were retained at CCSs for at least four days and transferred to base hospitals lying down.102 A report compiled in autumn 1916 showed that cases of moderate exposure were commonly held in medical units for two to three months ‘and very possibly longer’.103 While this cautious approach had merit in spring 1915, when knowledge was incomplete, its value soon expired. For the majority of casualties, who proved to be mild cases, this management instilled or reinforced the idea that they were suffering from a potent and irreversible affliction.104 Rather than promote an atmosphere of recovery and health, extended stay in hospital created fertile conditions for the elaboration of symptoms and chronic invalidity.105

Diagnostic difficulties created by the use of novel toxins were compounded because the symptoms of acute anxiety often mimicked the physical effects of mild exposure to gas: palpitations, chest pain, shortness of breath, fatigue, and weakness could all be produced by fear and worry,106 a similarity sometimes exploited by front-line troops seeking a medical exit from the battlefield.107 Having been invalided to a place of safety, the anxious soldier often made a recovery only to relapse when faced with the prospect of discharge from hospital and return to the trenches.

A survey conducted at No. 25 General Hospital at Hardelot, near Boulogne (which had a specialist gas ward), of 496 chlorine and phosgene casualties admitted between 8 July and 12 September 1916 produced disappointing findings.108 Only 118 patients (23.7 per cent) were discharged directly to base details and from there to active duty, while a further 132 (26.5 per cent) were referred to No. 1 and No. 5 Convalescent Depots at Boulogne. By the end of September only 42 (8.5 per cent) of these convalescent servicemen had returned to duty. In total 179 (36 per cent) gassed soldiers had been evacuated to the UK for further treatment. Although the study had shown that two-thirds of cases could ‘be satisfactorily treated in France’, it also demonstrated that doctors had yet to identify clinical regimes that efficiently returned servicemen to active duty.109

By May 1916 the threat to manpower presented by chemical weapons had become so serious that Sloggett set up a ‘committee on treatment of gas cases’.110 With Cummins as its secretary, it comprised a group of specialist physicians: Elliott, Douglas, Sir Wilmot Herringham, and Sir Almroth Wright. The last, a bacteriologist, had worked with the British army to develop a vaccine against typhoid, and was in France to conduct research into wound infections.111 In an era before antibiotics, gassed soldiers were also vulnerable to infection. At the very outset the committee made a crucial error: ‘in view of the fact that the cases arriving at base hospitals during the recent attacks have been, for the most part, slight or convalescent, it was decided that no evidence need be taken on the lines of communication’.112 By concentrating the study at base hospitals and excluding CCSs and other intermediate treatment centres, the committee failed to observe the development of symptoms and identify opportunities for early intervention. Not until 1917 was the oversight corrected.

Research into patterns of illness following exposure to gas revealed a complex picture. A study of convalescent depots in France by Captain Riddell in summer 1916 identified the features of disordered action of the heart (DAH) in gassed soldiers ‘under protracted’ treatment.113 DAH was characterized by shortness of breath, palpitations, chest pain, and fatigue after any form of exercise. No organic cause could be found and yet the disorder could prevent a soldier from returning to active duty. Furthermore, a study conducted in May and June 1916 of five convalescent depots receiving gassed casualties revealed extended treatment times: of 676 admissions, 480 (71 per cent) were retained for an average of nine weeks before being found ‘fit for duty’.114

Further research at Mount Vernon Military Heart Hospital in Hampstead and at No. 25 General Hospital in France revealed that ‘cardiac disability’ was the ‘chief weakness which invades all these [chlorine and phosgene] casualties’.115 At first, because of the mortality

associated with severe exposure, doctors were misled by these symptoms. In June 1916 T.R. Elliott examined a selection of gassed DAH patients and concluded that they had either been prematurely encouraged to undertake programmes of graduated exercise or they had inhaled greater concentrations of gas than realized. Accordingly, Elliott recommended an extended period of recuperation and advised that any patient who exhibited an irregular heartbeat in the third week of admission should be transferred to the UK ‘for rest’.116

Yet the solution proposed by Elliott was soon shown to exacerbate the ongoing invalidity. During 1917 further study of gassed servicemen found that the acute effects of gas could pass only to be replaced by a range of psychosomatic symptoms (irregular heartbeat, chest pain, and shortness of breath), aggravated when asked to perform any form of physical effort. Specialist gas doctors largely agreed that this was not a toxic effect. Lieutenant Colonel W.E. Hume, who had studied mustard gas patients at No. 25 General Hospital and No. 1 Convalescent Depot at Boulogne, argued, ‘the fact that there is such a discrepancy between the fast [heart] rate in all conditions of the body awake and the slow rate asleep in the majority of DAH patients seems to be proof that the tachycardia is of psychical origin’. 117 In a report for the Medical Research Committee, J.S. Haldane, J.C. Meakins, and J.G. Priestley observed that ‘it is difficult in many cases to distinguish the chronic gas cases from those suffering from irritable heart, shell-shock, or neurasthenia’.118 Similarly, Soltau, who examined the files of 150 gas pensioners, concluded that 30 per cent reported a range of symptoms that could equally well meet the criteria for neurasthenia or shell shock, while a further 25 per cent could be reclassified as DAH, formerly known as soldier’s heart.119

Working at No. 15 Canadian General Hospital in Taplow, Buckinghamshire, Lieutenant Colonel John C. Meakins and Captain T.W. Walker studied chronic cases of gassing who had been invalided to the UK. The key symptom, they identified, was shallow breathing which appeared to prevent the patient from increasing the volume of their respirations beyond a limited extent.120 This, in turn, restricted any physical activity that they could undertake. Dyspnoea, or shortness of breath, was often accompanied by rapid heartbeat, dizziness, and fatigue. These symptoms were not necessarily correlated with severity of exposure to toxin and, in their view, reflected ‘a distinct neurotic element’. The ‘mental effect of gas poisoning’, Meakins and Walker believed, was heightened by ‘the delayed action of certain gases and frightful consequences of high concentrations … deeply impressed upon the minds of the soldiers by the observation of their more unfortunate comrades’.121 A follow-up study conducted three months after discharge showed that only 16 (9.8 per cent) of 163 mustard gas cases returned to duty with the BEF.122

Concerned by lengthy stays in medical units and the need to return as many soldiers as possible to fighting units, a group of RAMC doctors (C.G. Douglas, T.R. Elliott, and A.B. Soltau) decided to monitor admissions to find ways of making treatments more effective. They studied the progress of gassed patients as they passed from field ambulance to CCS, base hospital, and convalescent depot to find out more about lengths of admission and outcomes. Key points in treatment were identified when so-called ‘neurasthenic’ symptoms might develop.123 ‘The neurasthenic element’, Douglas argued, was

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> the important feature in all gas cases, and it was the recognition of the true part played by this that contributed to the results. Firm control of patients from the start and the careful restriction of the period of detention in hospital to the minimum, prevented cases from falling into a morbid condition and developing those functional symptoms which so often delay convalescence and are exaggerated by prolonged hospitalization.124

The doctors discovered considerable variation in the efficacy of treatment: only 19.4 per cent of gassed patients treated at No. 25 General Hospital, with its specialist gas ward, were referred to the UK, whereas at Étaples, which lacked such expertise, 62.1 per cent were invalided across the Channel.125 From his study of No. 7 Stationary Hospital, Boulogne, Elliott concluded that ‘no casualties need be invalided for a longer period than three months and that the majority are soon fit for duty’.126

Following these investigations, strategies were developed to maintain the momentum of recovery and to distract patients from their symptoms. For example, men wearing dark glasses to combat the effects of photophobia were ordered to remove them as soon as their pupils had returned to a normal size and colour.127 When the acute effects of gassing had passed, great emphasis was placed on fresh air, exercise, and soldierly activities to maintain the progression to active duty.128 Programmes of graduated exercise were devised, based on measurements of pulse and respiration. The guidelines were:

> four days after the patient is allowed out of bed, he walks half a mile and, if this is not found excessive, he walks one mile on the next day; and if this again is not too much, it is repeated, and on the following day he walks three miles at the rate of three miles an hour.129

It continued to be believed that if a soldier were started on a programme of ‘muscular exercise’ too early or too forcibly, then tachycardia and dyspnoea were aggravated and the condition of DAH was ‘liable to be established and to persist for a very long time’.130 Nevertheless, by mid-1918 Douglas estimated that the average treatment time for 80 per cent of gas admissions had been cut from around three months to eight weeks.131

Casualties, of whatever nature, invalided to hospitals in Britain were less likely to return to front-line battalions than those treated in France.132 This was not simply because they included the most severe cases but also because soldiers were understandably reluctant to surrender a place of safety for the dangers and privations of trench warfare. As a result, considerable resources were directed towards treating the wounded and sick in France, and specialist units for shell shock, functional heart disorders, and gassed servicemen were set up. In October 1917, for example, a field ambulance serving the 47th Division was designated a corps gas centre not only to provide expert diagnosis and treatment but also to reduce the flow of casualties across the Channel.133 When, in August 1917, Douglas investigated the returns for all British base hospitals in France, he found that 47 per cent of gassed patients had been invalided to the UK for further treatment.134 Table 3 shows that the proportion evacuated home fell progressively from 22 per cent in summer 1918 to 5 per cent by the autumn. In addition, the specialist gas unit opened at No. 7 Stationary Hospital, Boulogne, under Major Wilson and Captain McIntosh steadily reduced the length of time

spent in hospital. ‘The experience of this hospital showed very clearly the value of special knowledge of gas poisoning on the part of the medical officer in order that he can be confident and firm in dealing with gassed soldiers.’135

Table 3 also shows that convalescent depots opened on the French coast were increasingly used to prepare soldiers for return to active duty. With shorter times in hospital, they were designed, as Major General Sir Wilmot Herringham recalled, ‘to give the men a cheerful and enjoyable time, while strengthening their bodies by regular and at the same time interesting exercise’.136 However, evidence from the convalescent depots suggests that it was far from easy to encourage soldiers to leave the relative comforts of these camps for the privations of the front line.137 In summer 1917 Douglas conducted a study of convalescent cases in France and found that after eight weeks only 64.7 per cent had discharged to full duty.138 Continued training of medical officers in diagnosis, together with stricter limits on periods of convalescence, progressively reduced the time that gassed soldiers remained as invalids. By September 1918, Colonel T.R. Elliott argued, this system of specialist hospital treatment combined with formal convalescence enabled the military to return 70 per cent of men exposed to mustard gas to infantry bases within eight weeks of exposure.139

A study of ex-servicemen who had been awarded a war pension for gassing showed that, though they had recovered from the physical effects, many continued to suffer from medically unexplained symptoms and psychological effects.140 Many of these pensioners were reclassified as suffering from DAH because cardiac symptoms (palpitations, chest pain, and shortness of breath) were prominent. Much effort was devoted to their treatment, largely involving programmes of graduated exercise. Although oxygen therapy saved the lives of many with severe exposure to gas,141 experiments showed that it had no lasting or therapeutic effect on chronic cases reclassified as DAH.142 Because of popular sympathy and the fact that many were unable to work consistently, many received a war pension.

VI Conclusions

Basil Liddell Hart, who had himself been gassed at the Somme in July 1916, wrote, ‘the chlorine gas originally used was undeniably cruel, but no worse than the frequent effect of shell or bayonet, and when it was succeeded by improved forms of gas both experience and statistics proved it the least inhumane of modern weapons’.143 Although his classification of chemical weapons did not gain general currency in the post-war period, his observation about their capacity to kill or wound requires discussion. While poets such as Wilfred Owen emphasized the trauma of soldiers dying from gas, their suffering was not significantly different from a terminal stomach wound or shrapnel damage to the head and face. This raises the question whether gas had a particular capacity to inspire terror, or whether the initial novelty and the continual refinement of toxins and delivery systems were responsible for its enduring psychological impact. During the period before the issue of effective respirators, Charles Cruttwell, an infantry officer, believed that gas undermined a basic survival mechanism. A serviceman subjected to artillery bombardment had few, if any, defensive options, and trusted to luck. However, when he was exposed to cloud gas, Cruttwell argued, it was impossible to evoke the protection of chance – ‘if the very air which he breathes is poison, his chance is gone: he is merely a destined victim for the slaughter’.

144 By contrast, shrapnel was tangible. It could be removed from a wounded soldier's body by a surgical procedure, but no physician could decontaminate a man's lungs, and it was popularly believed that, once toxins had been metabolized, the respiratory system remained damaged for ever.145

While the impact of cloud gas could be assessed because of its very novelty, the subsequent development of chemical weapons in the form of shells and mortar bombs made it more difficult to disaggregate their impact on morale from the wider effects of artillery bombardment.146 Nevertheless, the Allied armies invested heavily in the production of chemical weapons, and had the war continued into 1919 output was planned to increase significantly,147 which suggested that commanders had identified a particular casualty-producing power in gas. While seasoned infantry battalions habituated to gas, it appears to have unnerved units poorly prepared for the rigours of trench warfare. What was not discovered was whether gas could undermine morale at a faster rate than an artillery barrage. Because gas shells were mixed with high-explosive ordnance, it was difficult to compare the psychological impact of these various weapons on front-line troops.

In addition to the deliberate exploitation of surprise and uncertainty, fears evoked by gas owed much to broad cultural themes. Some toxic chemicals, like phosgene, could not be readily detected through the senses and triggered powerful vestigial fears of mysterious threatening forces.148 They touched on a deep human concern about the risk of being invaded by a potent and unseen force. Chemical weapons were unfamiliar, which created opportunity for rumour and exaggeration. Beliefs about gas often inspired strong emotions that could disrupt the rational evaluation of evidence and the formation of coping mechanisms. Fears may have been intensified because gas was a product of science and cutting-edge technology. Man-made disasters have generally been experienced as more troubling than natural ones.149 The novelty and scale of chemical weapons during the First World War were such that they had an enduring impact beyond the veteran population and respiratory medicine. Indeed, in 1928 the US Army physician, Colonel H.L. Gilchrist, wrote, 'the blame for every conceivable sort of ailment has been placed on gas; in fact, there is scarcely a functioning organ of the body whose disturbed action either during or since its participation in [the war] but has had the blame for its erratic performance laid to the door of poison gas'.150

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Table 1

British casualties from cloud gas (chlorine and phosgene).

	19 December 1915	27–9 April 1916	30 April 1916	17 June 1916	8 August 1916
Admissions to CCSs	1013	987	474	533	540
Deaths	36	64	50	64	106
Deaths per 100 casualties	3.6	6.5	10.5	12.0	19.6

Source: The National Archives, Kew, WO 142/100, C.G. Douglas, ‘Note of the Total Casualties Caused in the British Forces by Gas Warfare’, typescript, 17 January 1919, p. 5.

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Table 2

British casualties as a result of chemical weapons, 1915–18.

	Total casualties	deaths deaths	Deaths per 100 casualties
Type of gas			
Cylinder gas (chlorine)	*c.*7,000	*c.*350	Not known
April and May 1915			
Lachrymator gas shell	Not known	Nil	Nil
May 1915 – June 1916			
Cylinder gas (chlorine and phosgene)	4,207	1,013	24.0
December 1915 – August 1916			
Gas shell (diphosgene)	8,806	532	6.0
July 1916 – July 1917			
Gas shell (mustard gas, phosgene, and chlorarsines)	160,526	4,086	2.5
July 1917 – November 1918			
Projector attacks (phosgene)	444	81	18.2
December 1917 – May 1918			
Total	180,983	6,062	3.3

Source: The National Archives, Kew, WO 142/109, C.G. Douglas, ‘Casualties Caused in British Forces by Gas Warfare’; WO 158/123, ‘British Gas Casualties 1915 to 1918’; T.R. Elliott and A.B. Soltau, ‘Invalidism from Gas Poisoning’, in W.G. Macpherson, W.P. Herringham, T.R. Elliott and A. Balfour, eds, *Medical Services: Diseases of the War*, vol. 1, History of the Great War, based on Official Documents (London, HMSO, 1923), p. 517.

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Table 3

Mustard gas cases treated at No. 7 Stationary Hospital, Boulogne, May to October 1918.

	May to July		August		September and October	
	No.	%	No.	%	No.	%
Total admissions	254	100	438	100	559	100
Deaths	8	3.1	10	2.3	5	0.9
Evacuated to UK	57	22.4	70	16	27	4.8
To convalescent depot in France	36	14	138	31.5	342	61
Remaining in hospital	153	59.5	220	50.2	185	33.1

Source: The National Archives, Kew, WO 142/101, ‘Report on the Disposal of Gas Casualties on Lines of Communication’, n.d.