

sidered. The indications for this are failure to achieve adequate oxygenation or persisting inability to cough up secretions because of stupor or exhaustion. When reliable information indicates that during the months before the episode the patient had already reached an intolerable stage of disability despite all available treatment it may not be kind to embark on IPPV but it should be started whenever doubt exists. Oxygen in whatever concentration is necessary should not be withheld in the short period between the decision and intubation and it must be continuous. When the patient is restless intubation should be undertaken only by the skilled, but when deep coma has supervened intubation will be easier and should be carried out without delay.

The aims of IPPV are to secure adequate oxygenation, to remove secretions by tracheal and bronchial suction, and sometimes to rest an exhausted patient. Vigorous ventilation is undesirable, and there is no need to lower the PaCO_2 below about 8 kPa (60 mm Hg) initially as this may induce circulatory collapse and at a later stage lead to difficulty in withdrawing IPPV. Most patients will need IPPV until at least the morning after intubation, allowing the whole day for assessment after withdrawal. Further attempts at withdrawal are made daily. Tracheostomy is seldom needed. IPPV is usually appropriate when some reversible event such as pneumonia, abdominal surgery, or inappropriate sedation is a contributory factor, and is sometimes needed when the patient is admitted in coma or there is inadequate information about the earlier history. When oxygen is used sparingly and thoughtfully, however, and vigorous measures to stimulate breathing and coughing have been

performed, relatively few patients with a prospect of recovery will need IPPV.

ARRESTED IMPROVEMENT

Some patients relapse after initial improvement and remain somnolent and ill with a high PaCO_2 of 10.5 kPa (80 mm Hg) or so, apparently needing oxygen from time to time. In these circumstances it may help (a) to have the patient sitting out of bed; (b) to give a large dose of frusemide; (c) to add dichlorophenamide 50 mg 12-hourly; (d) to stop oxygen treatment altogether and remove the mask; and (e) to consider venesection if the packed cell volume is over 53%.

Aftercare

After the patient has recovered from the illness several steps should be taken to improve his respiratory condition. He should be persuaded to stop smoking and, if obese, to lose weight, and sedatives should be avoided. Exacerbations should be treated promptly with antibiotics, and the patient and his family should be taught to recognise features of recurring respiratory failure promptly. The possibility of an asthmatic component must be investigated and treatment given if necessary. Finally, the patient should be encouraged to exercise regularly within the limits imposed by dyspnoea.

Medical History

Schumann's hand injury

R A HENSON, H URICH

British Medical Journal, 1978, 1, 900-903

The tragic story of the life and death of Robert Schumann has fascinated both musicologists and medical men. The history of the mental illness that afflicted him from the age of 18 until his death at 46 has been amply recorded. For some 24 years this illness was characterised by fluctuations of mood, with many episodes of depression and identifiable, though less frequent, spells of elation. The composer recovered between these bouts of affective disorder. Around 1852, when Schumann was 42, symptoms of organic brain disease appeared, and in 1854 he entered a mental hospital after attempted suicide in the Rhine. There he died with terminal convulsions in 1856.

Slater and Meyer¹ looked at the history of these illnesses and reviewed previous diagnoses. They concluded that the true psychiatric diagnosis was manic-depressive psychosis with

ultimate dementia paralytica, thus siding with Gruhle² against Möbius,³ who believed that the diagnosis was dementia praecox, or schizophrenia.

Schumann complained of many neurological and psychiatric symptoms over the years, most of which appear to have been due to the affective disorder or later organic nervous disease. Slater and Meyer¹ attributed his giddiness and tinnitus (apparently without notable deafness) to a peripheral process, probably syphilitic. In fact, the known evidence does not indicate whether the causal lesion was central or peripheral. In February 1854 auditory hallucinations occurred, which can be ascribed to organic brain disease.

Exploration of neurological complaints in historical figures, before the introduction of systematic clinical examination and diagnostic laboratory investigations, is commonly unsatisfactory. The conclusions are generally tentative or speculative. Nevertheless, we were moved to review the evidence on Schumann's hand injury by reading Sams's two articles in *The Musical Times*.^{4,5} The second article contained new material from the Leipzig City Archives.⁶ By courtesy of Dr Sams we have been able to examine a copy of Rothe's report and discussion of the correspondence found at Leipzig. While further information may emerge in the future, the time seems ripe for a neurological study of this one aspect of Schumann's medical history. Our main sources have been Sams's two communications, Rothe's work, and Walker's biographical chapter in *Schumann, The Man*

Neurological Department, The London Hospital (Whitechapel), London E1 1BB

R A HENSON, MD, FRCP, physician in charge (also chairman, section of neurological sciences)

H URICH, MD, FRCP, professor of neuropathology, University of London

and his Music.⁷ Further useful information has come from the composer's letters; his diary proved less helpful, though admittedly our search was limited to the years when symptoms first appeared.

Schumann's hand injury—the myth

Schumann's hand disability has been ascribed to using a mechanical finger strengthener, which led to injury of the tendons of the third and fourth, or fourth and fifth, digits of the right hand. The source of this idea appears to be Wieck's *Clavier und Gesang*,⁸ in which Schumann's disaffected teacher and father-in-law wrote of a famous pupil who used a self-invented "finger tormentor," thus leading to the "righteous outrage" of his third and fourth fingers. Spitta⁹ told the story briefly: "Schumann devised a contrivance by which the greatest possible dexterity of the finger was to be attained in the shortest time. By means of this ingenious appliance the third finger was drawn back and kept still, while the other fingers had to practise exercises. But the result was that the tendons of the third finger were overstrained, the finger was crippled, and for some time the whole right hand was injured. This most serious condition was alleviated by medical treatment. Schumann recovered the use of his hand, and could, when needful, even play the piano; but the third finger was useless, so that he was for ever precluded from the career of a virtuoso."

This explanation of Schumann's hand injury is suspect according to Sams⁴ and Walker.⁷ Firstly, Wieck⁸ did not mention Schumann by name and, secondly, the composer's available diaries and letters do not mention the use of a mechanical device. Schumann simply wrote in his biographical notes (1831), "Overdone technical studies. Laming of my right hand." Referring to his difficulty in 1839 he said, "Some fingers (no doubt because of too much writing and playing in earlier years) have become quite weak, so that I can hardly use them." There were many finger strengtheners on the market at the time, and Bötticher wrote that Schumann ordered one in 1837.⁴

In a medical affidavit of 1841, translated below, Reuter said that Schumann used a machine because his second and third fingers were less strong and agile than the others, and this led to a paralytic condition. The evidence indicates that if Schumann used a finger strengthener he did not do so until after the hand condition appeared. Clara Schumann's only clear (and later) recollection was that the device did not cause the injury,⁴ but Niecks¹⁰ quoted her as having remembered that the cause was practice on a stiff, dummy keyboard.

Sams⁴ ridiculed the notion that any mechanical device, "short of a thumbscrew," could have such a profound and crippling effect. The idea that anyone would use and tolerate a finger-strengthening device so powerful that it caused permanent tendon damage, presumably by traction, seems preposterous. Sams⁴ concluded that the textual evidence indicated gradually progressive hand trouble of unknown cause. He suggested that the condition was a paralysis of the extensor muscles of the fingers and that the cause was mercurial poisoning.

Clinical history of the hand disability

The trouble started in 1830 or 1831. Friedrich Wieck accepted Schumann for training as a virtuoso pianist in 1830. While it may seem unlikely that he would have done so had Schumann been notably handicapped by the condition of his hand at the time, Walker⁷ stated that Schumann recorded "weakness in my right hand" as early as 1830. The following brief extracts from the composer's letters portray the development of the disability: "About October 1831 paralysis (*Lähmung*) of my right hand." In a letter to his mother, dated 14 June 1832, he wrote of his "strange misfortune." "The disease has spread from a paralysed, broken down finger on to the whole hand" (5 April 1833). "Don't worry about my finger, I can compose

without it. . . . I would hardly be any happier as a travelling virtuoso" (19 March 1834). To Clara Wieck, his future wife, "unhappy . . . on account of my suffering hand . . . it is getting steadily worse . . . I can only bring out music with difficulty, this is frightening and has caused me much distress. Now, you are my right hand" (3 December 1838). There is no doubt that the right hand was affected but contemporary observers differ in their accounts of the side affected and the distribution of the paralysis.

Schumann became liable for service in the local militia under a law of 1840. He claimed exemption and submitted medical evidence, in the form of an affidavit, prepared by his friend, Dr M E Reuter, who had known him since 1833. We have translated this affidavit and the medical reports quoted here from Rothe's paper.⁶ The doctor wrote on 26 January 1841: "In his youth he first noticed that the second and third fingers were less strong and agile than the others. Prolonged use of a machine with which the fingers were forcibly dorsiflexed led to a paralytic state of these fingers in which there was only weak sensation and their movement was not subject to voluntary control. He was compelled to give up a virtuoso career, but the fingers have remained in the same paralytic condition despite repeated attempts at treatment, so that in playing the piano he cannot use the middle finger at all and the index finger only partially. He is incapable of holding objects in his hand. . . ." Dr E W Güntz examined for the authorities on 19 July 1841, the delay being caused by the Schumanns' late honeymoon: peculiarly, he did not report until 31 December 1841. He confirmed that Schumann could not use "the index and third fingers of his right hand forcefully and voluntarily owing to partial paralysis. Therefore he cannot carry out arms drill reliably. . . ." Schumann was called up despite these reports. He appealed, and Dr Reuter reported again on 18 February 1842: "He can use the right index imperfectly and the right middle finger not at all in grasping and holding an object. These fingers have been paralysed more than ten years. . . ." The physician instructed by the authorities for their second examination was Dr R D Brachman, who reported on 16 March 1842, "The paralysis of the index and middle fingers of the right hand is only partial and does not prevent him from playing the piano, as is generally known. By the same token it should prevent him even less from handling a gun. . . ." He advised that the composer should be excused exercises, partly for other medical reasons that are irrelevant to this discussion.

In summary, it appears that Schumann suffered progressive paralysis of his right hand beginning at the age of 20 or 21 (1830 or 1831). The period of progression is not clear, but the condition had probably stabilised by the time of Dr Reuter's first report. We have no certain knowledge of the subsequent course. The paralysis was incomplete, and the right index and middle fingers were affected. As we have noted, Wieck⁸ wrote that the third and fourth fingers were "outraged," and the traditional story has the fourth and fifth⁷; but if these other digits were affected the doctors did not note this in 1841 and 1842. Presumably any disability present in other fingers was slight. We do not know for certain which movements were weakened or lost. The pattern of the paralysis and the use of an appliance for extending the offending members, as Dr Reuter witnessed, suggest that the extensors of the fingers were at fault.

Site of the lesion

There seems little doubt that the condition was neurologically determined. If the loss of movement had stemmed from direct injury or joint disease, rather than neurological disorder, this should have been obvious from the history and examination. Ehrenfechter¹¹ wrote that "several young students at Stuttgart lost the use of the third finger through overstraining it," as a result of excessive tension caused by lifting the flexed digits high when playing the piano. An injury of this type could not possibly have caused permanent paralysis of the index and

middle fingers, as described by all three Leipzig doctors in 1841 and 1842.

There is no neurological lesion that produces isolated weakness of flexion of the index and middle fingers. Anterior interosseous nerve palsy causes weakness or paralysis of flexion of the thumb and index finger, and we exclude a lesion of that nerve on this anatomical ground.¹² Furthermore, there is no neurological lesion that affects flexion and extension of the index and middle fingers alone. A lesion of the right posterior interosseous nerve, however, produces isolated weakness of extension in these digits, with serious effects for a pianist. While formal finger flexion is not directly weakened by posterior interosseous paralysis, patients find difficulty in grasping objects.^{13 14}

The persistently localised nature of Schumann's progressive disability excludes spinal cord disease and points to peripheral nerve disorder. A lesion of the seventh cervical nerve root or of the radial nerve, in arm or axilla, would have caused weakness of elbow and wrist extension, signs that the patient or examiner would have noticed.

The main objection to our tentative localisation is the "weakness of sensation" in the affected fingers mentioned by Dr Reuter, but not by Drs Güntz and Brachman. Unfortunately, Dr Reuter did not describe the distribution of the sensory weakness, nor did he indicate its nature. Paralysis can be felt as numbness, and this is a possible though not compelling explanation. As the other doctors made no mention of sensory disorder doubt may be cast on this part of Reuter's testimony. Despite this reservation, damage to the posterior interosseous nerve provides the most likely explanation of the neurological condition, for the clinical picture is more in accord with a lesion here than at any other point in the nervous system. The condition was apparently painless, which would fit the idea of posterior interosseous nerve involvement.

Clinical features of lesions of the posterior interosseous nerve

Paralysis of the posterior interosseous nerve can lead to finger drop, without wrist drop, from loss of power in the finger extensors with preserved radial wrist extension. When the causal lesion is progressive the digits are not necessarily weakened equally. Goldman *et al*¹⁵ reviewed the different patterns of paralysis encountered. We have found that weakness of index finger extension is often noticed first, the disability spreading sequentially to the middle, ring, and little fingers, but this pattern is by no means invariable. Thumb extension and abduction are ultimately affected. Lesions of the nerve are commonly but not invariably painless¹⁶; there is no accompanying sensory loss or paraesthesiae. Weakness of wrist extension may or may not be evident, depending on the site of the lesion.

Anatomy and pathology of the posterior interosseous nerve^{14 17}

The posterior interosseous nerve is the larger terminal branch of the radial nerve. Given off in front of the lateral epicondyle of the humerus, it passes in a fascial tunnel, sending branches to the supinator and extensor carpi radialis muscles, before passing between the two lamellae of the supinator muscle to reach the back of the forearm. On leaving the supinator, the nerve courses down the back of the forearm, supplying the common extensor muscle of the fingers, the extensor of the little finger, and the ulnar extensor of the wrist; further down it innervates the long abductor and both extensors of the thumb and extensor of the index finger. The anatomy of the nerve is variable—for example, its branching is complicated and inconsistent—and in some people the supinator heads are not continuous at their insertion, so that the nerve is in direct contact with the radius.

The nerve may be compressed within the supinator and at entry or exit. Such compression occurs most often at the point of entry, either from the fibrous margin of the muscle or from a fibrous band, passing from the common extensor origin to merge with the border of the supinator and the fascia that invests the nerve. Indeed, compression and elongation of the nerve can be seen at this site with pronation and supination of the forearm.¹⁴ Compression injury can lead to localised nerve swelling with distal atrophy if the complaint goes unrelieved.^{18 19} The nerve can be damaged by repeated hyperextension of the elbow.²⁰

Posterior interosseous palsy is a complication of fractures of the upper third of the radius or dislocation of the radioulnar joint, and the prognosis is usually good in such cases. Missile wounds and lacerations are other causes.

Spontaneous palsies have two common causes—lipoma and other benign tumours, and compression or traction injury at one or other of the sites mentioned above. While lipomas accounted for 12 of the 18 cases reviewed by Goldman *et al*,¹⁵ nerve compression, visible at operation, from the normal structures mentioned above (especially the margin of the supinator muscle) has been reported.^{13 14 18 19 21}

Examples of posterior interosseous paralysis have occurred after a long swim,²² competitive rowing,²³ and snowballing and hammering.²⁰ In such cases, where the lesion can be confidently ascribed to vigorous pronation and supination of the forearm and flexion and extension of the elbow, the onset is commonly acute. With slowly progressive palsies, not due to compression from tumour, surgical exploration shows the point of nerve damage, but does not always indicate any individual muscular activity as the cause.

Pathological diagnosis in Schumann's case

We can confidently assert that neurosyphilis did not cause Schumann's condition. Firstly, acquired tertiary syphilis would hardly be manifest at the age of 20 or 21; and, secondly, although syphilitic amyotrophy may present with wrist and finger drop in the early stages, the paralysis does not remain limited to the extensor muscle group.²⁴

The idea of a toxic neuropathy was introduced by Sams,⁴ the suggested cause being mercurial treatment of a syphilitic infection. This solution does not commend itself to us in a case of apparently chronic, partial mononeuropathy. Neither Gowers²⁵ nor Kinnier Wilson²⁶ mentioned peripheral nerve lesions in their chapters on mercurial intoxication. Both organic and inorganic mercurial compounds can damage peripheral nerves; but there are remarkably few documented cases²⁷ apart from epidemic series of organic mercurial poisoning^{28 29} (and LeQuesne *et al*³⁰ questioned whether peripheral neuropathy was responsible for symptom production in their patients). With inorganic mercury poisoning the usual pattern is a symmetrical polyneuropathy, though asymmetrical lesions affecting one or more limbs have been ascribed to this cause.³¹ Mercurial neuropathy was so rare during antisyphilitic treatment as to raise doubts on the causal relationship.³² We have been unable to trace a recorded example of isolated mononeuropathy due to mercury poisoning. Admittedly lead intoxication may present with unilateral paralysis of the wrist and finger extensors, but the condition usually reaches a considerable degree in a few days or one or two weeks; the complaint is usually, if not always, ultimately bilateral, other muscle groups may be affected, and the prognosis is usually good.^{25 26} We see no reason to invoke lead poisoning here.

The common cause of spontaneous isolated peripheral nerve lesion is compression, and we have already discussed the ways in which the posterior interosseous nerve can be damaged in this way. Compression from a benign tumour, particularly lipoma, can be safely excluded in Schumann's case because we know from the medical reports of 1841 and 1842 that the lesion

was incomplete some ten years after onset, two fingers only being noticeably affected. This leaves us with the possibilities that the nerve was compressed either before entry into the supinator muscle, within the muscle, at the points of entry and exit, or anterior to the elbow joint. The most likely site would have been the entrance into the supinator; but the point of exit could have been inculpated if the muscle heads were not continuous at their insertion in Schumann's radius, so that the nerve was in direct contact with the bone and therefore more exposed to damage from pronation and supination of the forearm. Guillain and Courtellemont³³ believed that the latter explanation was correct in their conductor patient with partial posterior interosseous palsy and tenderness over the point of the nerve's emergence from the supinator. The movements endangering the nerve at either site, however, would have been pronation and supination.

Schumann indicated at least twice that he thought excessive practice caused his condition. Is this feasible? He began his training as a keyboard virtuoso at a time when technique had undergone radical, necessary changes through the replacement of clavichord and harpsichord by the piano; Chopin (b 1810) and Liszt (b 1811) were his contemporaries.

We find it difficult to imagine the degree of elbow extension demanded by practice or performance causing posterior interosseous damage in front of the elbow joint. Might supination and pronation have been responsible? Gerig³⁴ has published an account of the writings of the great keyboard teachers, including those of Schumann's day; but it is difficult for the inexpert reader to discern how far rotation of the forearm was allowed or encouraged at that time. Czerny³⁵ warned against "unnatural twisting or shaking of the arms or elbows. . . ." Ortmann³⁶ observed that pronation and supination, forearm rotation, are particularly engaged in octave tremolo; these movements also operate when the little finger is passed over the thumb. While the degree of forearm rotation used in play may lead to posterior interosseous compression, we think this explanation unlikely unless there was a minor anatomical deviation in Schumann's case. This could have taken the form mentioned above; but the presence of a fibrous band or unduly prominent aponeurotic border of the supinator would have had the same ultimate effect, rendering the nerve unnaturally liable to compression during muscular activity.

We are unaware of any reports of posterior interosseous palsy in professional pianists. On a less exalted plane, case 2 of Woltman and Learmonth¹⁶ was a girl pianist; the authors advised her to cease playing, which she did, but the disability slowly progressed over many years. We have heard of another example, a boy pianist aged 16, through the courtesy of Professor Ian Macdonald.

The apparent absence of reported cases among the large body of professional pianists leads us to conclude that either Schumann's condition was related to piano playing because of a minor anatomical variation, or its cause lay in other activities. One objection to our solution lies in the apparent arrest of the condition when only two fingers were affected, an unusual course for a compression palsy of the type we suggest. In fact, this objection may lend force to the idea that piano playing was the source of Schumann's trouble: the enforced restriction of play deriving from his weakened fingers might have saved him from further injury.

Conclusion

We have leaned heavily on the evidence of Drs Reuter, Güntz, and Brachman in reaching this speculative solution to the problem of the paralysis of Schumann's hand for they report hard clinical information, though this is admittedly incomplete. Further documents may yet emerge and shed further light. Meanwhile we end with the thought that music may have been enriched by Schumann's enforced retirement from his career as a pianist and his consequent concentration on composition.

We are grateful to Dr Eric Sams for permitting access to H-J Rothe's *Neue Dokumente zur Schumann*, and for his interest. Professor W Ian Macdonald and Mr Basil Helal kindly read the initial manuscript and made helpful comments.

References

- Slater, E, and Meyer, A, *Confinia Psychiatrica*, 1959, **2**, 65.
- Grühle, W, *Zentralblatt für Nervenheilkunde*, 1906, **29**, 805.
- Möbius, P J, *Über Robert Schumanns Krankheit*. Halle, Marhold, 1906.
- Sams, E, *The Musical Times*, December 1971, p 1156.
- Sams, E, *The Musical Times*, May 1972, p 456.
- Rothe, H-J, *Neue Dokumente zur Schumann*. Leipzig, Forschung im Stadtarchiv, 1967.
- Walker, A, in *Robert Schumann, The Man and His Music*. London, Barrie and Jenkins, 1972.
- Wieck, F, *Clavier und Gesang*, p 44, 1853, quoted by E Sams.⁴
- Spitta, P, in *Grove's Dictionary of Music and Musicians*, ed H S Colles, 3rd edn, vol 4, p 652. London, Macmillan, 1929.
- Niecks, F, *Robert Schumann*, p 102, 1925. Quoted by Spitta.⁹
- Ehrenfechter, C A, *Technical Studies in the Art of Piano Playing*, p 34. London, William Reeves, 1891.
- Nakano, K K, Lundergan, C, and Okihiro, M M, *Archives of Neurology*, 1977, **34**, 477.
- Hustead, A P, Mulder, D W, and McCarthy, C S, *Archives of Neurology and Psychiatry*, 1958, **79**, 269.
- Henson, R A. Personal observation.
- Goldman, S, et al, *Archives of Neurology*, 1969, **21**, 435.
- Woltman, H W, and Learmonth, J R, *Brain*, 1934, **57**, 25.
- Sunderland, S, *Nerves and Nerve Injuries*. Edinburgh and London, Livingstone, 1968.
- Lallemant, R C, and Weller, R O, *Journal of Neurology, Neurosurgery and Psychiatry*, 1973, **36**, 991.
- Ochoa, J, and Neary, D, *Lancet*, 1975, **1**, 632.
- Sharrard, W J W, *Journal of Bone and Joint Surgery*, 1966, **48B**, 777.
- Whiteley, W H, and Alpers, B J, *Archives of Neurology*, 1959, **1**, 226.
- Kruse, F, *Neurology*, 1958, **8**, 307.
- Harvey, P K, 1977. Personal communication.
- Heathfield, K W G, and Turner, J W A, *Lancet*, 1951, **2**, 566.
- Gowers, W R, *Manual of Diseases of the Nervous System*. London, Churchill, 1886.
- Kinnier Wilson, S A, *Neurology*, ed A N Bruce, 1st edn. London, Edward Arnold, 1940.
- Cohen, M M, in *Handbook of Clinical Neurology*, ed P J Vincken and G W Bruyn, vol 7, p 514. Amsterdam, North Holland Publishing Co, 1970.
- McAlpine, D, and Araki, S, *Lancet*, 1958, **2**, 629.
- Rustam, H, and Hamdi, T, *Brain*, 1974, **97**, 499.
- LeQuesne, P M, Damluji, S F, and Rustam, H, *Journal of Neurology, Neurosurgery and Psychiatry*, 1974, **37**, 333.
- Crouzon, O, and Delafontaine, P, *Revue Neurologique*, 1926, **33**, 642.
- Kulkow, A E, *Zentralblatt für die gesamte Neurologie und Psychiatrie*, 1927, **111**, 274.
- Guillain, G, and Courtellemont, M, *Presse Médicale*, 1905, **13**, 50.
- Gerig, R R, *Famous Pianists and their Technique*. Newton Abbott, David and Charles, 1976.
- Czerny, C, 1839, quoted by Gerig,³⁴ p 112.
- Ortmann, O, *The Physiological Mechanics of Piano Technique*. New York, E P Dutton, 1929.

(Accepted 18 January 1978)

ONE HUNDRED YEARS AGO Attention has recently been called in Glasgow, in a very striking manner, to the milk-supply, by the outbreak of typhoid fever which, as we stated a short time since, was most conclusively shown by the medical officer of health for the city to have been due to the use of polluted milk. The epidemic is now subsiding, no new case having appeared for a fortnight; this result being undoubtedly due to the energetic measures taken by the authorities. Last week, a largely attended meeting of influential persons was held, under the presidency of the Lord Provost, to consider what steps should be taken to secure a supply of pure milk for the city and suburbs. It was resolved, "That it is desirable that a general measure of legislation should be passed with regard to the regulation of milk supply." In the view of the meeting, it was desirable that all dairies which directly or indirectly supply milk to towns, should be bound to register themselves with the local authority, and that shops for the retail selling of milk should be required to take out licenses. These dairies and shops should be regularly and frequently inspected. A committee was appointed to carry out the object of the resolutions. (*British Medical Journal*, 1878.)