The Hillsborough tragedy

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On 15 April 1989 Liverpool and Nottingham Forest met at Hillsborough football stadium for an FA Cup semifinal match. The ensuing crush disaster, in which 95 spectators were killed, has been studied in an official report.[1]

In the arrangements for spectators in the west stand of the ground (fig 1) the area was divided by five radial metal barriers, with further metal barriers capped by backwards turned bars to prevent invasion of the pitch. The pens also had longitudinal barriers to control crowd surges. The effect of this design was that the central pens, 3 and 4, lying immediately behind the Liverpool goal, became confined spaces, leaving as the only free outlets the tunnel along which the spectators had entered and one small gate for each pen in the perimeter and side fences (1-12; fig 1).

A sudden late influx of an additional 2000 spectators into the central pens led to a severe crush, which was exacerbated by surges in the crowd when the match started. One of the horizontal crush barriers (124a; fig 1) in pen 3 collapsed and large numbers of spectators were thrown forwards under a weight of falling bodies (fig 2). At 1506 a police officer ran on to the pitch and the match was stopped.

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Actions at scene

Gates 3 and 4 were opened and the police and spectators worked quickly to move the critically injured and dead on to the pitch. Others were evacuated through the tunnel. First aid was administered by police and St John Ambulance personnel together with seven or eight doctors and a few nurses who had been spectators. The resuscitation equipment available was very limited and included only a single oxygen cylinder. The nine available stretchers were quickly used up, and spectators resorted to using advertising hoardings to carry bodies to the gymnasium, which served as a makeshift mortuary.

Help was not summoned over the public address system until 1530. By then some doctors had left the ground thinking that the match had been abandoned because of hooliganism. At about 1520 Radio Sheffield made an urgent appeal for local doctors to go to the ground. Between 1530 and 1545, 20 doctors arrived in the sports hall. Nearly all were general practitioners carrying equipment that they would normally take on a home visit. These supplies were quickly exhausted. By 1615 most of the injured had been removed to hospital.

The greatest difficulty for doctors at the stadium was the overwhelming number of casualties requiring immediate attention. Unusually for a major incident, the dead and seriously injured had no wounds or obvious sign of injury. Most were very young. This led to a dilemma in differentiating those who should benefit from resuscitation from those who could be pronounced dead. The paucity of equipment for resuscitation resulted in frustration and feelings of inadequacy.

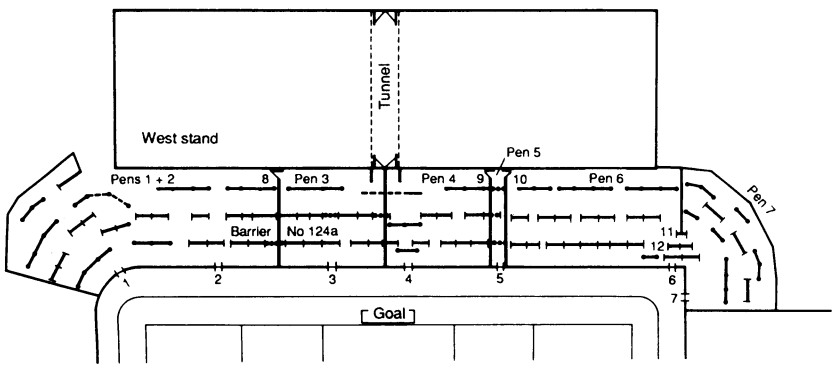

FIG 1—*Arrangement of west terraces. Barrier 124a in pen 3 was barrier which collapsed. Gate 3 was 82 cm wide, gate 4 was 79 cm wide*

Hospital accident and emergency departments

The accident and emergency departments of the Northern General and Royal Hallamshire Hospitals received a message from the police at 1520 to say they were "on casualty standby." The agreed and correct procedure, however, was to telephone the hospital switchboard with the message, "Implement the major disaster plan." No such message was received.

NORTHERN GENERAL HOSPITAL

After the police message at 1520 key staff at the Northern General Hospital, including the consultant in accident and emergency medicine and two assistant matrons, were informed that the hospital should expect casualties. At 1525 a message was received from the South Yorkshire Ambulance Service control to "expect a child with a cardiac arrest." There was no reference to Hillsborough or indication that a major incident was taking place.

Shortly afterwards three patients receiving cardiopulmonary resuscitation arrived, and only then, after conversation with the ambulance crew, did the charge nurse in the accident and emergency department implement the proper major disaster procedure.

Failure to implement the major incident alert earlier meant that only a few staff had to cope with a large clinical workload in addition to implementing the major incident plan. The consultant physician on call was not included in the first line call of the major disaster plan but had been alerted by seeing the BBC television broadcast at 1525. He arrived at 1555, when his presence was important in coordinating the efforts of medical and nursing staff dealing with the immediate resuscitation and treatment of the patients with crush asphyxia. Within five minutes many more staff had arrived, and two doctors were dispatched to the stadium with the major accident equipment.

Presentation and disposal of patients

Most of the seriously injured arrived at the hospital within the first 30 minutes. Thirteen patients were

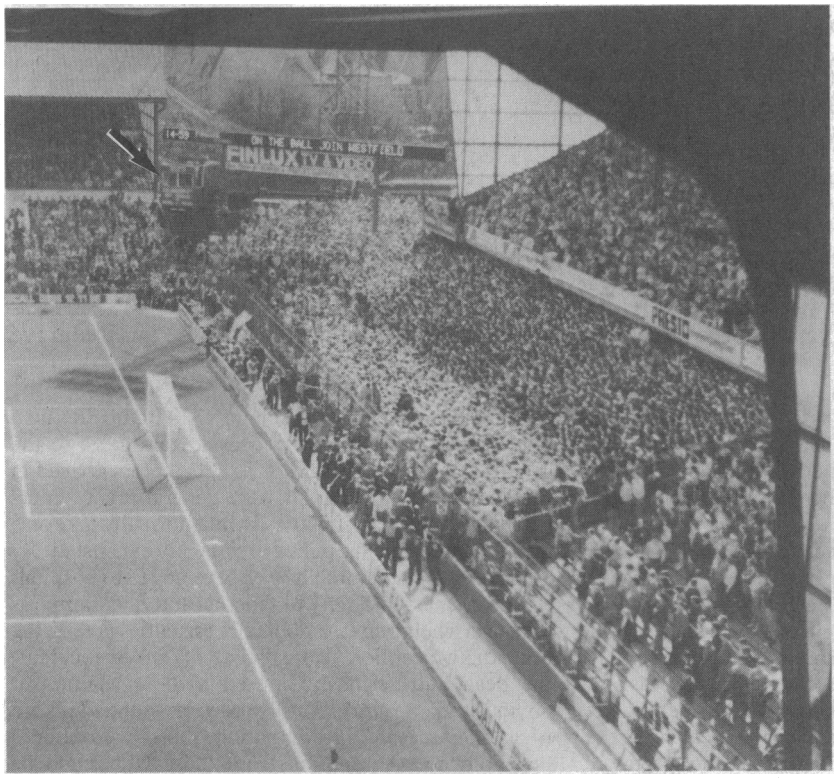

FIG 2—*Photograph of west terraces at 1459 showing overcrowding in pens 3 and 4. Police observation post arrowed*

receiving basic cardiopulmonary resuscitation. Initially these patients were treated in the resuscitation areas, and cardiac output was restored in three of them. As more patients requiring resuscitation arrived those patients arriving in cardiac arrest were triaged to the mortuary. Two of the patients resuscitated from cardiac arrest reached intensive care but subsequently died.

Fifteen other patients had severe neurological problems. Nine were admitted unconscious or convulsing. A further six were confused on arrival and subsequently deteriorated, and all six began having severe convulsions requiring resuscitation, intubation, and ventilation in the accident and emergency department or on the receiving ward. An additional 15 patients lost consciousness in the crush. Two of these were confused on admission but rapidly improved. One subsequently developed cortical blindness. Figure 3 summarises the status of patients on arrival and their subsequent management and disposal.

The accident and emergency department of the Northern General Hospital is designed to cope with large numbers of patients requiring resuscitation. There are four permanent and eight prepacked resuscitation points. But even these facilities were not sufficient, and each point was used more than once as patients were moved on either as a result of being declared dead or being removed to a support ward.

ROYAL HALLAMSHIRE HOSPITAL

At the Royal Hallamshire Hospital the accident and emergency consultant and senior registrar arrived together at 1537. The nursing staff had already begun to prepare the department to receive large numbers of casualties. When two police officers arrived and confirmed that a major disaster was taking place the hospital implemented the major disaster plan.

The first two patients arrived at 1540. One was dead and the other required intubation and was transferred to the intensive care unit. Being further from the stadium, the hospital then had a quiet period, when the departmental plans could be organised. Between 1600 and 1700 the department received a further 69 patients, 24 of whom required admission. Figure 3 shows the disposal of patients. Forty five were discharged after treatment.

Intensive care units

Seventeen patients were admitted to intensive care at the Northern General Hospital and one at the Royal Hallamshire Hospital. Six of those initially admitted to the Northern General were subsequently transferred to the Royal Hallamshire for computed tomography and to relieve pressure on staff at the Northern General.

On admission to intensive care all patients had arterial lines, central venous pressure lines, urinary catheters, and nasogastric tubes inserted. Care was aimed at good oxygenation, preventing further seizures, and treating presumed cerebral oedema. All patients were hyperventilated to achieve an arterial carbon dioxide pressure of 3·5-4·0 kPa and an arterial oxygen pressure of 15-20 kPa, aimed at reducing intracranial pressure while maintaining good cerebral perfusion and oxygenation.[2] Ventilation was continued for one to 15 days. One patient admitted to intensive care at the Northern General Hospital died soon

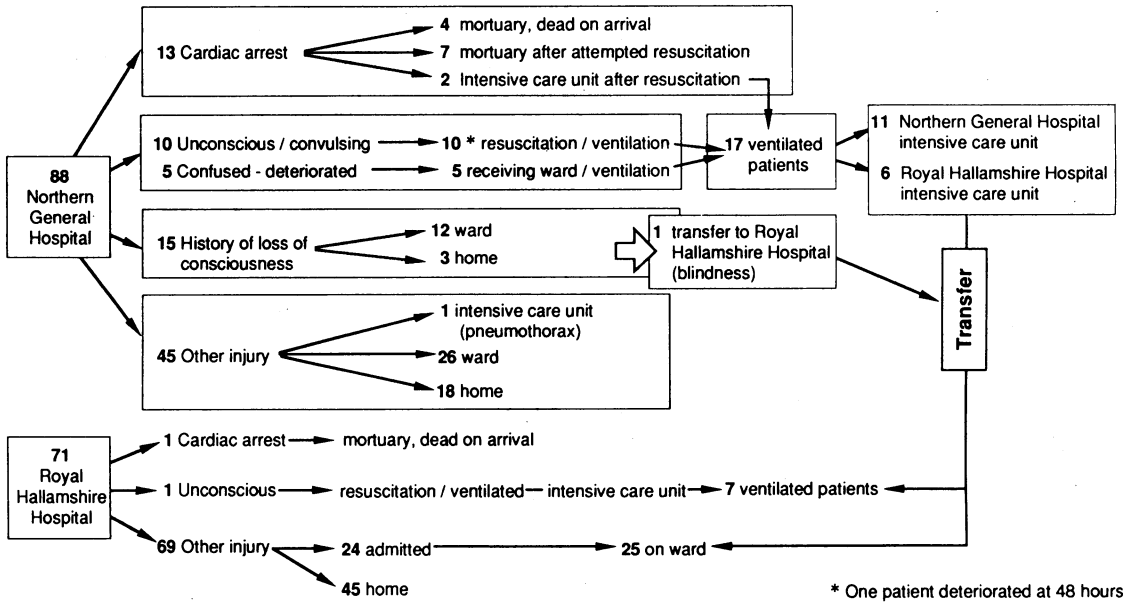

FIG 3—*Flow of patients through accident and emergency departments of Northern General and Royal Hallamshire Hospitals. Three other patients, with minor injuries, taken to Barnsley District General Hospital*

afterwards. The 10 patients who remained at that hospital were also treated with mannitol, fluid restriction, and frusemide to ensure a slightly negative fluid balance while ensuring an adequate cardiac output. All 10 patients also received high dosage dexamethasone (1 mg/kg). Four patients who remained at the Northern General Hospital and four admitted to the Royal Hallamshire Hospital had refractory seizures which were controlled with intravenous anticonvulsants, two patients requiring thiopentone.

In five patients there was evidence of inhalation of gastric contents (observed at intubation, in aspirate from endotracheal tubes, or in chest radiographs). All these patients received prophylactic metronidazole and cefuroxime and none developed chest infection. Five patients not given prophylactic antibiotics developed chest infections. In these patients a potential cause additional to inhalation may have been prolonged ventilation from four to 11 days. One patient who died was confirmed to have inhalational pneumonia at necropsy.

Crush asphyxia

The disaster at Hillsborough resulted in a gradual and prolonged crush affecting large numbers of previously fit young people. The patients presented with little evidence of the classic signs of traumatic asphyxia —facial oedema; cyanosis of face, neck, shoulders, and upper chest; subconjunctival haemorrhages; scattered petechiae over the upper part of the body[3-6]—yet many developed serious neurological complications, usually said to be rare.[3,5,7]

All the asphyxiated patients had craniocervical cyanosis, which cleared completely within two hours of the trauma. Only two patients had petechial haemorrhages (chest and face) and one oedema of the retinas and optic discs. Figure 4 summarises the neurological complications and subsequent outcome. Altogether 22 patients suffered neurological injury due to asphyxia. Their ages ranged from 14 to 41 years (median 19), and all had been unconscious at the ground. Two died two and 48 hours after admission without regaining consciousness.

EARLY RECOVERY

Six ventilated patients (including four who had secondary deterioration) recovered within 24 hours and were extubated. Four patients required ventilation for up to 72 hours and six for up to 14 days. Each of these six patients had a severe neurological deficit at one month.

Five patients deteriorated within two hours of their initial recovery. Without warning, each started to scream, developed myoclonus and a deteriorating level of consciousness, and went into status epilepticus. These patients, two of whom had already been moved to medical wards, required urgent resuscitation, including ventilation and treatment of seizures. A sixth patient, who seemed to be recovering, deteriorated after 48 hours. Five of the six made a good physical recovery. A seventh patient developed cortical blindness after six hours but subsequently made a good neurological recovery.

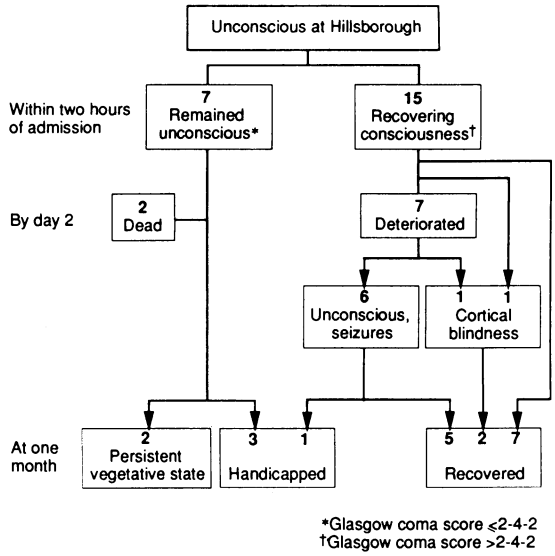

FIG 4—*Neurological injuries and outcome at 48 hours and one month*

OUTCOME AFTER NEUROLOGICAL INJURY

Figure 4 shows the outcome at one month in patients with neurological injury. Two patients died in hospital. The two patients in a vegetative state remained so one year after injury. Four patients had major neurological and cognitive handicaps, including one patient who was cortically blind. The table gives the details of seven representative cases, together with the outcomes at six months.

Seizures

Seizures occurred in 16 patients, mostly as multiple tonic-clonic events associated with myoclonus. Four patients were known to have had seizures before reaching hospital, 12 had seizures in the accident room, and five continued to have seizures despite large doses of conventional antiepileptic drugs. No patient who made a complete physical recovery had had any further seizures by one year of follow up.

Other neurological injuries

Six patients had neuropraxia, of whom three had brachial plexus injuries, and one each had sciatic nerve, lateral cutaneous nerve of thigh, and posterior interosseous nerve injuries.

Other injuries

Five patients suffered a pneumothorax, bilateral in one, which in four cases required drainage. One patient was noted at initial intubation to have severe laryngeal oedema, which persisted and required a tracheostomy.

Nine patients had evidence of right heart strain in the initial electrocardiogram (four of these cases have been reported[8]). Three patients had pericardial

Representative case histories of patients with neurological injury

Case No	Sex and age (years)	Time and status on admission	Progress	Outcome at six months
1	M 41	1700. Agitated, responsive to pain	1745 Developed status epilepticus. Sedated and ventilated for 24 hours	No neurological deficit
2	F 26	1600. Unconscious	1730 Developed seizures and myoclonus. Sedated and ventilated for 24 hours. Several episodes of upward deviation of eyes	No formal follow up. Reportedly in good health
3	M 21	1630. Unconscious	1800 Improved and alert, then suddenly restless and irritable with deterioration in conscious level. Spasms. Treated with chlormethiazole and sodium valproate. Ventilated overnight	Minor behavioural problems
4	M 17	1618. Unconscious	Improved but at 1700 became agitated and had seizure. Treated with benzodiazepines and ventilated overnight	Difficulty in concentrating. Mental impairment on psychological testing
5	M 20	1547. Already intubated, unconscious	1625 Recovered consciousness and extubated. 1730 Became obtunded and had seizures. Given benzodiazepines and ventilated overnight	No neurological or psychological impairment
6	M 16	1630. Unconscious	1700 Had first seizure. Given thiopentone, sodium valproate, and phenytoin and ventilated for 48 hours. Became able to obey commands. Relapsed with further seizures. Ventilated for seven more days	Spastic weakness, cognitive handicap
7	M 23	1640. Responded to questions	2230 Complained of poor vision, confused, confabulating. Tests showed perception of light only. Gradual improvement over 10 days	Needed inpatient psychiatric care. Severe memory loss and damage to right temporal lobe

effusions. Cardiac ultrasonography showed transient impairment of right ventricular function in seven patients. Hypotension was noted in two patients. One patient had laparotomy for a rigid abdomen and was found to have gastric dilatation.

There were large numbers of soft tissue injuries to the chest, back, and pelvic area—especially in patients who had been crushed against the metal barriers—but surprisingly few fractures. One patient had a fractured skull but no serious neurological damage, four patients had rib fractures, and one patient each had fractures of radius and ulna, styloid process, and ankle. One patient had arch aortography because of a widened mediastinum suspected on chest radiography, but the result was normal. In seven patients changes compatible with aspiration were seen in chest *x* ray films.

Discussion

We have used the term "crush asphyxia" to describe the main neurological injury in these patients. Although there were similarities to traumatic asphyxia, the conditions differ in mechanism of injury, clinical findings, main complications, and outcome.

Traumatic asphyxia is usually caused by a heavy weight falling on the chest or a violent crush between heavy objects, such as motor vehicles. Often the victim has warning that he or she is about to be crushed, and the natural reflex is to suddenly inhale and then close the glottis.[3 5] It has been suggested that this results in massive increase in pressure in the superior vena cava, explaining the classic presentation of traumatic asphyxia, when petechial haemorrhages in the superior vena caval distribution and subconjunctival haemorrhages are almost always found.[9 10] Any neurological complications do not usually lead to permanent disability. If the patient survives the initial crush, then a full recovery usually ensues.[3 5-7]

By contrast, crush asphyxia is caused by a gradually increasing and sustained pressure on the chest, so preventing inspiration and making it more likely that the glottis will remain open. This may lessen the increase in pressure in the superior vena cava, while the sustained nature of the crush may be important in the development of serious neurological complications.

In this series a major contribution to the eventual success of resuscitation was that 24 anaesthetists arrived at the accident and emergency department of the Northern General Hospital, making it possible to allocate an individual anaesthetist to every unconscious patient.

Syndromes caused by acute cerebral anoxia were reviewed by Plum and Posner and include early delayed post-anoxic encephalopathy and post-anoxic myoclonus.[11] In our series 30% of patients (6/20) remained seriously disabled at six months. Two deaths which occurred early in hospital are included because they added substantially to the management burden. The delayed neurological deterioration was unexpected in patients who had seemed to be recovering and were already being moved to medical wards away from intensive monitoring and resuscitation facilities. The deterioration occurred between two and 48 hours after the anoxia had passed, and all but one patient seemed to make an uneventful recovery. Detailed neuropsychological assessments, however, subsequently showed two patients to have cognitive defects.

There was no extrinsic reason for the neurological deterioration and we can offer no explanation or mechanism. We presume that it was another response of the young brain to prolonged hypoxia. No pathological material was available for examination. The deterioration did not resemble delayed post-anoxic encephalopathy[12] or delayed post-anoxic myoclonus[13]; the syndrome developed much earlier and there were no extrapyramidal features. In retrospect, computed tomography and electroencephalography, undertaken because of uncertainty about the nature of the acute brain injury, added nothing to patient management or to understanding the mechanism of the deterioration. Dexamethasone reduces oedema around tumours but its place in the management of hypoxic oedema is less certain. It was used in these patients on the basis that it might be beneficial and was unlikely to do harm.

Inhalation of gastric contents was a problem in six patients; severe external pressure on the abdomen, a full stomach, and unconsciousness are potent conditions for inhalational pneumonia. Our experience suggests that prophylactic antibiotics helped reduce the risk of chest infection in these patients.

IMPLICATIONS FOR DISASTER PLANNING

Communication difficulties are an important feature in most major disasters reported.[14 15] In our series neither of the two main hospitals received proper warning that a major disaster was taking place, which delayed implementing the major disaster plans. Fortunately, many staff arrived because of the media coverage, but in other circumstances—particularly at night—this might have resulted in important delays. We strongly advise that the standard message recommended in the recent government guidelines, "Major incident declared—activate plan," should be used to alert hospitals of a major incident.[16] This task should be undertaken by the ambulance service, and other emergency service communications to hospitals should be relayed via the ambulance service.

Our previous major disaster plan expected the junior staff, on receiving the disaster call, to contact their seniors in the cascade system. This proved impracticable because of the large numbers of patients requiring their immediate services for resuscitation. We have altered our plan to delegate this responsibility to administrative personnel, and we suggest that other hospitals should modify their plans also. The initial impression was that a large number of patients with head injuries were to be expected, and a surgically directed major disaster plan was implemented. Medical skill proved to be important in the early management of the neurological cases, and we recommend that a consultant physician should be included with the anaesthetic and surgical teams in the first line call for all major disaster plans. Though neurologists are not usually included in major disaster plans, they may have useful advice to give about the management of cerebral anoxia and seizures.

The victims at Hillsborough were similar in age, mainly wore T shirts and jeans, and carried no identification. Identifying unconscious and dead people was therefore a major problem and the uncertainties and process of identification very distressing to relatives. The dead at the stadium were photographed, which helped identification. We recommend using a Polaroid camera for this and now include a medical photographer in our major incident plan.

Doctors at the scene had little equipment to help with the resuscitation of large numbers of casualties. but, given the little likelihood of a recurrence of such a disaster, it seems improbable that any sporting venue would be equipped with such facilities. Nevertheless, large stadiums and similar venues should ensure that the personnel and equipment necessary for advanced life support are available to deal with smaller scale emergencies, such as a cardiac arrest. It has been recommended that there should be increased provision of resuscitation equipment at large events, and in future ambulance services will deploy their major incident vehicles near stadiums where crowds in excess

of 25 000 are expected.[1] New legislation may be needed.

This report was compiled on behalf of the staff at the Northern General Hospital, Sheffield, the staff at the Royal Hallamshire Hospital, Sheffield, and the many people who helped at the scene. The efforts of hundreds of professionals and volunteers were vital and greatly appreciated. We thank Neil Appleyard, David Edbrooke, David Dawson, Stuart Yates, Ian Winston, John Duncan, G A Baker, Charlie Cooper, Kath Sherry, Tim Shaw, and A Moss for access to the neuropsychological reports on some of the survivors.

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Doctor-patient communication: the Toronto consensus statement

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Effective communication between doctor and patient is a central clinical function that cannot be delegated. Most of the essential diagnostic information arises from the interview, and the physician's interpersonal skills also largely determine the patient's satisfaction and compliance and positively influence health outcomes.[1-3] Such skills, including active listening to patients' concerns, are among the qualities of a physician most desired by patients.[4] Increasing public dissatisfaction with the medical profession is, in good part, related to deficiencies in clinical communication. Studies in many countries have confirmed that serious communication problems are common in clinical practice.

This consensus statement addresses three issues: What are the most important facts we already know about doctor-patient communication? What are the most important things that could be done now to improve the situation? and, What are the most important unanswered questions?

What are the most important facts we already know about doctor-patient communication?

PROBLEMS IN PRACTICE

Communication problems in medical practice are both important and common. For example, 54% of patient complaints and 45% of patient concerns are not elicited by physicians.[5] Psychosocial and psychiatric problems are common in general medical practice, but these diagnoses are missed in up to 50% of cases.[6 7] In 50% of visits the patient and the doctor do not agree on the nature of the main presenting problem.[8 9] In one study patients were interrupted by physicians so soon after they began describing their presenting problems (on average within 18 seconds) that they failed to disclose other significant concerns.[10] Most complaints by the public about physicians deal not with clinical competency problems, but with communication problems,[11] and the majority of malpractice allegations arise from communication errors.[12] Residents or trainees[13] and practising physicians[14] have shown substantial deficiencies when studied. Only a low proportion of visits with doctors include any patient education,[15] and a surprisingly high proportion of patients do not understand or remember what their physicians tell them about diagnosis and treatment.[16] Cultural differences also impede the work with patients.[17 18]

Patient anxiety and dissatisfaction is related to uncertainty and lack of information, explanation, and feedback from the doctor. Yet doctors often misperceive the amount and type of information patients want. The language doctors use is often unclear, both as regards the use of jargon and in relation to a lack of the expected shared meanings of relatively common terms.[20-23]

COMMUNICATION PRACTICES IN RELATION TO OUTCOMES

The quality of clinical communication is related to positive health outcomes.[24 25] Reduction in blood pressure was significantly greater in patients who, during visits to the doctor, had been allowed to express their health concerns without interruptions.[26] Concordance between physician and patient in identifying the nature and seriousness of the clinical problem is related to improving or resolving the problem.[5 9 27]

Explaining and understanding patient concerns, even when they cannot be resolved, results in a significant fall in anxiety.[28] Greater participation by the patient in the encounter improves satisfaction and compliance[1] and outcome of treatment (for example, control of diabetes and hypertension).[24] The level of psychological distress in patients with serious illness is less when they perceive themselves to have received adequate information.[29 30]

Beneficial clinical communication is feasible routinely in clinical practice and can be achieved during normal clinical encounters, without unduly prolonging them, provided that the clinician has learned the relevant techniques.[31 32]

EDUCATIONAL ISSUES

To become effective communicators, physicians