3 Review of existing estimates

3.1 Incidence

Problems arise when trying to compare incidence estimates from different studies, as operational definitions and study methodologies can affect estimates. Differences between estimates may reflect both real variations in the rate of brain injury between regions and over time, and differences in methodology.

Existing estimates of the incidence of ABI are presented in Table 3.1 (overseas estimates) and Table 3.2 (Australian estimates). Most studies use rates of hospitalisation as indicative of incidence (although factors other than incidence affect hospitalisation rates). Among hospital-based studies, operational definitions and methodologies can differ in terms of:

- whether data were from a single hospital or from multiple hospitals in a region—this is likely to affect sample size, catchment area, and the heterogeneity of the sample in terms of demographic and socioeconomic factors;
- methods of identifying cases of brain injury—both the source of data (e.g. coded summary data; individual medical records containing uncoded information), and the medical criteria used (e.g. specific diagnoses; symptoms);
- whether principal diagnoses or all diagnoses were used to identify cases of ABI from coded summary data sources;
- the population age range included;
- whether or not deaths before hospital admission and / or in hospital were included in the estimate of brain injury incidence; and
- whether or not non-residents (i.e. people who reside outside the study area) and repeat admissions were included.

Important aspects of study methodology are summarised in Tables 3.1 and 3.2, where this information is provided in the published sources (see also Table 2.3 for more detailed analysis of operational definitions). Most of the estimates reviewed in this section relate to traumatic brain injury, rather than ABI more broadly.

Overseas estimates

Table 3.1 presents 15 estimates of ABI incidence overseas. All are expressed as number of incident cases of head / brain injury per 100,000 per year. The earliest estimate of incidence is for Minnesota, USA, during the decade 1965–1974, and the most recent is for Colorado in the 1990s.

Table 3.1: Overseas estimates of annual ABI incidence rates [a]

Rate (/100,000)	Study location and data collection period	Data sources and methods	Criteria for inclusion[b]	Population	Source
55	China 1982	Population survey, with review of available medical records	History of head trauma with loss of consciousness, post-traumatic amnesia, or clinical evidence of subsequent focal brain dysfunction. Deaths included	People living in households in six large cities	Wang et al. (1986)
281	Aquitaine, France 1986	Hospital admissions and death certificates—used sample approach rather than census	Contusion, laceration, skull fracture, or brain injury, and/or loss of consciousness following injury caused by external mechanical force. Residents of Aquitaine. Pre-hospital deaths included	Total population	Tiret et al. (1990)
372	Ravenna, Italy 1984–85	Hospital admissions—single hospital servicing study area	Examination by neurologist revealed indication of head injury (including loss of consciousness or post-traumatic amnesia). Non-residents included	Total population	Servadei et al. (1988)
228	New Zealand 1988	Hospital discharge data (all public hospitals)—excluding deaths	ICD–9 codes. Only people alive at discharge included. Transfers excluded	Total population	Caradoc-Davies & Dixon (1995)
236	Akershus County, Norway 1974	Hospital admissions and pre-hospital deaths	Trauma to face, head or neck with skull or neck fracture, or specified symptoms. Pre-hospital deaths included where head injury recorded (whether or not recorded as cause of death)	Total population	Nestvold et al. (1988)
316	Johannesburg, South Africa 1986–87	Hospital admissions—used sample approach rather than census	'Included' ICD–9 code, or 'case–finding' ICD–9 code with clinical symptoms of brain injury. Resident of Johannesburg. Only first admissions included	Age 15 and over	Nell & Brown (1991)
91	Cantabria, Spain 1988	Hospital admissions—single hospital servicing study area	Loss of consciousness, skull fracture or neurological findings attributable to head injury. Resident of Cantabria. Contacted hospital within 24 hours of injury	Total population	Vazquez-Barquero et al. (1992)

(continued)

Table 3.1 (continued): Overseas estimates of annual ABI incidence rates [a]

Rate (/100,000)	Study location and data collection period	Data sources and methods	Criteria for inclusion[b]	Population	Source
249	Umea district, Sweden 1984–85	Hospital admissions and pre-hospital deaths	Physician or autopsy diagnosed traumatic brain injury—implied symptoms of impaired brain function due to trauma. Surveyed ICD codes listed. Repeat brain injuries within study period excluded. Pre-hospital deaths included	16–60	Johansson et al. (1991)
160	Cambridge, UK 1982	Hospital admissions	Admissions with diagnosis of head injury	Total population	Johnson & Gleave (1987)
270 (males) 116 (females)	Olmsted County, Minnesota, USA 1965–74	Hospital admissions, emergency room visits, out-patient examinations, home visits, and death certificates (i.e. any medical attention)	Concussion with loss of consciousness, post-traumatic amnesia, neurologic signs of brain injury, and skull fracture (with or without altered consciousness). Pre-hospital deaths included	Total population (age-adjusted to 1970 USA population)	Annegers et al. (1980)
200	USA 1974	Sample survey of hospital data	'Included' ICD–8 code, or 'case–finding' ICD–8 code with clinical symptoms of brain injury	Total population	Kalsbeek et al. (1980) see also Anderson et al. (1980)
249	Bronx, New York, USA 1980–81	Hospital admissions, emergency room attendances, and Medical Examiner's reports—sample of hospitals serving the area	Loss of consciousness >10 min, skull fracture, post-traumatic seizure, or neurological findings attributable to head injury (ICD–9 codes used for some hospitals). Resident of Bronx. Identified within 24 hours of injury. Only first admissions included. Pre-hospital deaths included	Total population	Cooper et al. (1983)
152	Rhode Island, USA 1979–80	Hospital admissions— all hospitals in Rhode Island	ICD–9 codes	Total population	Fife et al. (1986)
160	San Diego, USA 1981	Hospital admissions—all hospitals in San Diego	ICD–9 codes used to flag cases, but only people with physician-diagnosed brain injury included	Total population	Kraus et al. (1984)
101	Colorado, USA 1990s	Traumatic Brain Injury Surveillance System. Hospital admission or death due to TBI	Skull fracture or intracranial injury	Total population	Brooks et al. (1997)

(a) See Table 2.3 for more detailed information on operational definitions used in incidence studies.
(b) Unless otherwise indicated it is assumed, based on information provided in the cited sources, that all estimates include deaths before discharge from hospital, but not deaths prior to admission.

Only two of the estimates were not based on hospital data. The lowest estimate (55, China 1982) was based on a population survey in six large cities, and the definition used was quite narrow, requiring clinical evidence of focal brain dysfunction. The estimates of brain injury incidence in Minnesota included all people who had 'head injury with evidence of presumed brain involvement', identified from hospital admissions, emergency room visits, out-patient examinations, home visits and death certificates. Although information was gathered from a wide range of sources, the overall estimate (which would lie somewhere between the reported 119 for females and 274 for males), is well within the range of estimates obtained from hospital data only (see Table 3.1).

Overseas estimates based on hospital data range from 91 to 372. It is difficult to assess the likely effect of definitional and methodological differences on the estimates obtained. Presumably studies that look at a number of hospitals, rather than a single hospital, and use a census rather than a sample approach to data collection, produce more reliable and representative data. Variation in the age structure of populations that use given hospitals is likely to be reflected in rates of hospitalisation for ABI (Moen & Batey 1986). Thus studies that combine data from a number of hospitals produce estimates that are effectively averaged across differences in demographic factors.

Many studies use a list of ICD codes to identify cases of brain injury. Some lists are longer and more inclusive than others (see Table 2.4). However, the extent to which the inclusion or exclusion of specific ICD codes will affect estimated incidence will depend on the distribution of cases between codes, and on hospital coding practices. Some codes may be used very infrequently, so their inclusion or exclusion will have little effect on the estimates arrived at. Unless the number of separations for each diagnosis code is reported it is difficult to assess the magnitude of the effect of including or excluding specific codes.

It has been argued that definitions that identify cases of brain injury solely on the basis of diagnosis codes recorded in summary data can lead to over-estimation of incidence (Willer et al. 1990). However, the extent of overestimation will depend on the particular codes used, and coding practice.

Estimates in Table 3.1 for New Zealand in 1988, the USA in 1974, Rhode Island in 1979–80, and Johannesburg in 1986–87 were based on operational definitions that used diagnosis codes to identify cases from coded summary data—these estimates range from 152 to 316. Estimates based on operational definitions that are arguably more rigorous, in that they require the presence of specific symptoms to identify brain injury (Minnesota 1965–74, Cantabria 1988, New York 1980–81, Ravenna 1984–85, Aquitaine 1986, Akershus County 1974 and Rhode Island 1981), range from 91 to 372.

Thus, based on the studies reported in Table 3.1, there does not seem to be any general tendency for what appear to be more restrictive definitions to produce lower estimates. This does not suggest that differences in operational definitions are not important in influencing estimates. Rather, there is such variation, due to other methodological differences and real differences in incidence rates, that any effect cannot be clearly detected.

Only two estimates in Table 3.1 are restricted to certain age groups within the population. The estimate for Johannesburg in 1986–87 (316) is for the population aged 15 and over, and the estimate for Umea District in 1984–85 (249) is for people aged 16 to 60. Both these estimates are relatively high. The effect of excluding certain age groups from the calculation of overall incidence rate will depend on age-specific rates of incidence, and the age structure of the population.

Some estimates include people who have died before hospital admission, where brain injury has been recorded in the coroner's report. These people may account for a substantial

proportion of all brain injuries (e.g. 12%, Kraus et al. 1984; 17%, Willer, cited in Honey 1995a; 3.8%, Selecki et al. 1981). People who die in hospital tend to make up a smaller proportion of the overall estimate (e.g. 7%, Willer, cited in Honey 1995a; 6%, Kraus et al. 1984; 2.3%, Selecki et al. 1981), but this group is more commonly included in estimates of incidence. It is important to consider deaths due to brain injury when discussing the overall impact of brain injury in a society. However, from a disability perspective, we are interested in those people who survive the critical phase and return to the community.

Australian estimates

Table 3.2 presents 11 estimates of incidence of brain injury in Australia. The estimates are all expressed as number of incident cases of head / brain injury per 100,000 per year. They range from 57 to 377—similar to the range for overseas estimates reported in Table 3.1 (55–372). Nine of the estimates are based on hospital admissions data but, as for the overseas estimates, operational definitions and methodologies differ substantially between studies.

The estimate of 160 for New South Wales in 1990 (Lyle et al. 1990) was derived by direct application of the incidence rate reported for San Diego (USA) in 1981 (see Table 3.1, Kraus et al. 1984). The objective of Lyle et al. (1990) was to predict the extent of brain injury in New South Wales by applying severity and outcome rates obtained in the San Diego study, as comparable local data were not available. The authors based their work on the assumption that brain injury rates in New South Wales and San Diego were similar. This assumption was supported by a detailed comparison of data collected by Selecki et al. (1981) in New South Wales with data from San Diego.

The estimate of 128 for Victoria in 1992 was derived using a formula developed by Willer (cited in Honey 1995a), based on the Canadian Health and Activity Limitation Survey. The methods by which the formula was devised are not reported by Honey (1995a).

Caution must be exercised if estimates and formulae based on studies conducted elsewhere are to be used to answer questions about rates of brain injury in Australia. Estimates can be affected by a range of factors, as well as differences in operational definitions and study methodology, including population sex and age structure and socioeconomic and cultural factors (such as levels of interpersonal violence), geography, traffic safety policy, and hospital admission practices. These factors are likely to differ between countries, and between regions within countries. Lyle et al. (1990) stated in their paper that 'reliance on these estimates for service development should be seen only as a short-term solution…Access to local information is essential for aetiological research, evaluation and monitoring'.

Of the nine estimates based on hospital data, six apply to the total population. The estimates for Victoria of 116 in 1987–88 and 104 in 1990 are limited to people aged 0–65, and the estimate of 57 for Western Australia in 1988–92 applies only to people aged 16–65. It is difficult to know what effect these restrictions have on the overall estimate.

The very low Western Australian estimate was based on a definition of brain injury that included traumatic head injury, ruptured aneurysm, neoplasms / tumour, post-infectious brain damage and anoxia. However, people were excluded if they did not stay in hospital for more than a day, were not resident in Western Australia or died in hospital, and 'duplicate' records were removed. These factors might partially explain the low estimate, particularly the exclusion of people who did not stay in hospital for more than a day. A high

Table 3.2: Australian estimates of annual ABI incidence rates [a]

Rate (/100,000)	Study location and data collection period	Data sources and methods	Criteria for inclusion[b]	Population	Source
377	NSW 1977	Admissions to all public hospitals in 1977, plus 50% sample of private hospitals in the first 6 months of 1978	Principal diagnosis only. List of ICD–8 codes all included, plus list of ICD–8 codes to be included if accompanying external cause code indicates trauma	Total population	Selecki et al. (1981)
100	NSW, North Coast Health Region 1988	Admissions to all 22 hospitals in the region	ICD–9 codes used to flag cases of possible brain injury in some hospitals. Diagnosis of brain injury, defined as documentation of a definitive period of alteration of the conscious state. Transfers excluded	Total population	Tate et al. (1998)
160	NSW 1990	Incidence rate for San Diego, 1981 (based on hospital admissions) applied to NSW without adjustment	ICD–9 codes used to flag cases, but only people with physician-diagnosed brain injury included	Total population	Lyle et al. (1990) Kraus et al. (1984)
116[c]	Vic 1987–88	Admissions to public hospitals—Patient Reporting System	Recorded as due to head injury	Age under 65	Health Department Victoria et al. (1991)
104	Vic 1990	Victorian Inpatient MDS (admissions to all public hospitals)	ICD–9–CM codes associated with head injury	Age 0–65	Honey (1995a)
128	Vic 1992	Formula developed from results of 1986 Canadian Health and Activity Limitation Survey applied to 1992 Victorian population data	Unclear whether or not pre-hospital deaths included	Total population	Willer, cited in Honey (1995a:29)

(continued)

Table 3.2 (continued): Australian estimates of annual ABI incidence rates [a]

Rate (/100,000)	Study location and data collection period	Data sources and methods	Criteria for inclusion[b]	Population	Source
200–300	Qld	Hospital admissions	No details given	Total population	Queensland Department of Family Services and Aboriginal and Islander Affairs (1994)
57[d]	WA 1988–92	Hospital admissions	List of conditions (specific ICD–9 codes not given); length of stay >1 day; of WA origin; duplicates removed. Only people alive at discharge included	Age 16–65	Stanton et al. (1994)
250	SA (one metropolitan region) 1984	Admission through accident and emergency department of one major hospital	Problem presented or diagnosis given indicated head injury. Deaths after admission included	Total population	Badcock (1988)
322	SA 1987	Hospital separations, all public and private hospitals in SA	Records flagged by ICD–9 codes, then a subset checked in detail against a clinical definition involving certain critical symptoms. Unclear whether deaths after admission included	Total population	Hillier et al. (1997)
232[e]	ACT 1977	Hospital admissions		Total population	Selecki et al. (1981)

(a) See Table 2.3 for more detailed information on operational definitions used in incidence studies.
(b) Unless otherwise indicated, it is assumed, based on information provided in the cited sources, that all estimates include deaths before discharge from hospital, but not deaths prior to admission.
(c) This figure was calculated using the annual number of incident cases (4,970) published in the source and ABS population data for Victoria as at 30 June 1988.
(d) This figure was calculated using the annual number of incident cases (600) and the population data provided in the source.
(e) This figure was calculated using information in the source on the number of ACT residents hospitalised for head injury (495) and ABS population data for the ACT as at 30 June 1977.

proportion of people who are admitted to hospital with head injury are discharged after one day. For example, in the hospital-based study of Selecki et al. (1981) 46% of patients admitted with head injury remained in hospital for one day or less.

For several of the hospital-based estimates there is little information on the 'criteria for inclusion' used. The three studies for which ICD–9 codes were specified (Hillier et al. 1997; Honey 1995a; Tate et al. 1998) differ slightly in the range of codes selected (Table 2.4). All three included codes for skull fracture (800, 801, 803 and 804) and intracranial injury (850–854). In addition, Hillier et al. (1997) included anoxic brain damage, and Tate et al. (1998) included non-psychotic mental disorders due to organic brain damage and 'late effects' of fracture of the skull and face bones and intracranial injury. Selecki et al. (1981) used a short list of ICD–8 codes (including birth trauma), plus an additional longer list of codes that were included if an accompanying external cause code indicating trauma was recorded. In practice, this definition is likely to have been quite broad, which might explain the high incidence estimate obtained for New South Wales (377) (although the estimate of 232 for the Australian Capital Territory, based on the same operational definition, was not so high). It should be noted that Selecki et al. (1981) and Tate et al. (1998) only included people for whom one of the selected ICD codes was recorded as principal diagnosis.

The 1990 Victorian study (Honey 1995a) included all records identified by the listed ICD codes. In the South Australian study, individual medical records for a sub-sample of cases identified using the ICD codes were checked against a 'clinical definition'. This required subjects to have been admitted to hospital with 'a presenting history of trauma to the head' resulting in any of a number of specified symptoms or conditions (Hillier et al. 1997). All records reviewed conformed to the clinical definition, so the overall estimate was effectively based on ICD–9 codes alone. In the North Coast study, records identified by the ICD diagnosis codes were excluded if there was no mention of 'a definitive period of alteration of the conscious state'. In contrast to the South Australian study, 846 of the 1,259 cases identified by ICD–9 codes were excluded because they did not meet this criterion. Had the clinical definition not been applied in the New South Wales North Coast study the estimate of 'incidence' would have been similar to that produced by the South Australian study.

Can we identify a 'reasonable range' for incidence estimates?

Having reviewed a number of estimated 'incidence' rates it is necessary to make some comment as to what might be considered a 'reasonable' estimate or range of estimates.

Looking first at the overseas estimates reviewed (Table 3.1), two were from studies based on non-hospital data (Wang et al. 1986; Annegers et al. 1980) and, because of the very different methodologies they used, it is difficult to make a meaningful comparison between these and the estimates based on hospital data.

However, methodologies also varied among the hospital-based studies. Two of the hospital-based studies were restricted to certain age groups (Nell & Brown 1991; Johansson et al. 1991). Given the very different levels of risk of brain injury (particularly TBI) associated with different age groups, these two studies will be excluded for the purpose of deciding on a 'reasonable' estimate for the total population.

Of the remaining estimates, those based on data from a single hospital might, in general, be less reliable as indicators of incidence for a variety of reasons, such as differences in demographic factors (e.g. socioeconomic status), admission policies and coding practices. Estimates based on data from a number of hospitals in a region are likely to be more reliable because they average across these differences.

Focusing on studies that used data from a number of hospitals, estimates ranged from 101 to 281 per 100,000 per year. Though operational definitions varied slightly between these studies, all were framed fairly narrowly to identify cases of head injury / traumatic brain injury. Two of the estimates (Cooper et al. 1983; Tiret et al. 1990) included deaths prior to hospitalisation. Exclusion of pre-hospital deaths would have reduced these estimates to about 270 per 100,000 in the case of the French study (Tiret et al. 1990) and 228 per 100,000 in the case of the New York study (Cooper et al. 1983).

Thus, based on overseas studies, a 'reasonable' estimate of the rate of hospitalisation due to traumatic brain injury would seem to lie within the range of 100 to 270 per 100,000 per year.

Similar reasoning can be followed to arrive at a 'reasonable' range of estimates based on Australian studies (Table 3.2). Excluding estimates not based on Australian hospital data (Lyle et al. 1990; Willer, cited in Honey 1995a), restricted to certain age groups (Health Department Victoria et al. 1991; Honey 1995a; Stanton et al. 1994) or based on data from a single hospital (Badcock 1988), the range of estimates is between 100 and 377 per 100,000 per year. Only one of these estimates is under 200 per 100,000 (Tate et al. 1998). This range of estimates is likely to be too broad for many applications.

Cumulative incidence

Annegers et al. (1980) estimated a lifetime cumulative incidence for traumatic brain injury (to age 75) of 20% for males and 8% for females. However, this is an inflated estimate of individual risk, because it was based on annual incidence rates and there was no adjustment for the fact that an individual may experience repeat brain injury events. In fact, the study found that people who had experienced one head injury were at increased risk of repeat head injury. Of the 3,587 head injury episodes in that study, 7% were not the first head injury experienced by the individual. It was calculated that, after one head injury a person was at three times the risk of a subsequent head injury, and after a second head injury this increased to eight times the risk of the general population. However, medical staff might be more likely to use technology such as CT scan or MRI to investigate a potential brain injury in a person who has a history of brain injury, leading to a greater proportion of subsequent brain injuries being confirmed and recorded.

3.2 Proportion of incident cases leading to long-term disability

If we have reliable information on the proportion of people who go on to experience long-term disability as a result of their brain injury, incidence data can potentially be used to calculate the number of people in the community needing ongoing support. However, studies that look at incidence rates of brain injury, and related incidence rates of resulting impairment or disability, are quite rare (van Balen et al. 1996).

Table 3.3 lists several studies that have provided estimates of the proportions of people who have brain injuries (mostly TBI) who go on to experience longer-term problems. The studies vary in terms of the definition of ABI used, what severity levels of ABI are included in the sample, time elapsed between injury and follow-up assessment, sample size, age groups considered, and measure of outcome. The proportions, as presented in Table 3.3, are calculated as a percentage of the sample of survivors assessed. In many of the studies more than one measure of outcome is used. As the outcome categories are generally not mutually exclusive, percentages should not be summed.

The first six studies presented in Table 3.3 included all new cases of head / brain injury, regardless of severity, admitted to a certain hospital (or hospitals) within a specified period. Three of these six only provide information collected at discharge from hospital. This information is of limited use in predicting the proportion of people who will experience long-term disability, as significant improvement commonly occurs in the first few months after brain injury (Jennett & Teasdale 1981).

What stands out looking at these first six studies is the variation in terms of how and when 'outcome' was measured. Some studies used more than one measure, producing quite different estimates of the proportion of people with ongoing problems or needs.

Kraus et al. (1984) used three measures of 'outcome' at discharge. They reported that 12% of people discharged alive were in need of ongoing care (primary care, rehabilitation, outpatient or home care). Using the Glasgow Outcome Scale, 5% of patients had moderate or severe disability or were in a persistent vegetative state. Seven per cent of patients had physician-diagnosed neurologic deficit or limitation.

Hillier et al. (1997) reported that 40% of people hospitalised for ABI had 'residual difficulties' on discharge, most commonly physical difficulties (experienced by 23% of people) and headaches (experienced by 21%). Eight percent of people needed some sort of physical assistance, particularly with mobility.

Five studies presented data on outcome for people who had 'severe' ABI (Cuff & Donald 1987; Johansson et al. 1991; Johnson & Gleave 1987; Tate et al. 1989a, b; Tennant et al. 1995). However, the definition of 'severe' differed between studies. Tennant et al. (1995) found that 16% of people experienced disability of moderate or greater severity (GOS). Taking an alternative approach to assessing outcome, based on the 1980 ICIDH concept of Occupational Handicap (WHO 1980), they found that 36% of people were unable to occupy their time in employment, education or homemaking. Ability to occupy time may be a useful measure of quality of life.

The last three studies in Table 3.3 deal with moderate brain injury (Rimel et al. 1982) and minor brain injury (Powell et al. 1996; Rimel et al. 1981). The two definitions of minor brain injury are very similar, and the follow-up time was 3 months after discharge in all three studies. In both studies of minor brain injury persistent symptoms were reported for around 85% of people. Powell et al. (1996) reported that the most common symptoms were headache (experienced by 46% of people) and tiredness (37% of people). Rimel et al. (1981) reported that 78% of people with minor ABI experienced persistent headaches and 59% had memory deficits. Of people with moderate ABI, 93% experienced headaches, 90% had memory deficits, and 87% had difficulties with activities of daily living (Rimel et al. 1982).

The three studies also looked at the percentage of people who were unemployed 3 months after discharge. A large proportion of people with moderate ABI who had been employed before injury were unemployed. The difference in the percentage of people unemployed in the two studies of minor brain injury (Powell et al. 1996; Rimel et al. 1981) might be explained by demographic differences between the two samples. Rimel et al. (1982) observed that factors such as education and socioeconomic status can significantly influence outcome after minor brain injury, while these factors are less important in determining outcome after moderate or severe brain injury, as their influence is overwhelmed by the severity of the injury itself.

Table 3.3: Estimates of the proportion of people who suffer adverse outcomes as a result of ABI [(a)]

Region and date	Measure of outcome[(b)(c)]	Proportion with specified outcome	Level of severity[(d)]	Time of assessment (relative to time of injury)	Sample size	Source
Rhode Island, USA, 1979–80	Discharged to chronic care institution:	4%	All levels	At discharge	2,870	Fife et al. (1986)
San Diego, USA, 1981	GOS moderate disability or worse:	5%	All levels	At discharge	2,972	Kraus et al. (1984)
	Need for continuing care/rehab on discharge:	12%				
	Neurologic deficits or disability:	7%				
South Australia, 1987	Discharge to rehab. care:	15%	All levels	At discharge	177	Hillier et al. (1997)
	Residual difficulties on discharge:	40%				
Cantabria, Spain, 1988	GOS moderate disability or worse:	3%	All levels	1 year	477	Vazquez-Barquero et al. (1992)
Umea, Sweden, 1984–85	Self-reported impairment:	35%	All levels	1.5–3 years	162 (aged 16–60)	Johansson et al. (1991)
	Self-reported disability:	15%				
Ravenna, Italy, 1984–85	GOS moderate disability or worse:	4%	All levels	3 months	370	Servadei et al. (1988)

(continued)

Table 3.3 (continued): Estimates of the proportion of people who suffer adverse outcomes as a result of ABI [(a)]

Region and date	Measure of outcome[(b)(c)]	Proportion with specified outcome	Level of severity[(d)]	Time of assessment (relative to time of injury)	Sample size	Source
Cambridge, UK, 1980–82	Unemployable:	19%	Severe: post-traumatic amnesia >24 hours	Min. 2 years	68	Johnson & Gleave (1987)
North West Region, UK, 1974–83	GOS moderate disability or worse:	16%	Severe: length of stay in neurosurgery > 1 week	2–13 years	176 (aged 16–50)	Tennant et al. (1995)
	Unable to occupy time:	36%				
Canton St Gallen, Switzerland, 1987	GOS moderate disability or worse:	33%	Severe: intracranial lesions detected by CT scan	3 years	45	Annoni et al. (1992)
	Capacity for work reduced:	55%				
Western Metropolitan Health Region of Sydney	GOS moderate disability or worse:	48%	Severe: sufficient severity to necessitate inpatient rehabilitation after acute medical management	3–10 years (average 6 years)	87 (aged 15–45 at admission)	Tate et al. (1989a) Tate et al. (1989b)
	Neurophysical and/or neuro-psychological Impairment:	91%				
	'Substantially limited' or 'poor' psychosocial outcome:	76%				
NSW	GOS moderate and severe disability:	30%	Severe	?	?	Cuff & Donald (1987)

(continued)

Table 3.3 (continued): Estimates of the proportion of people who suffer adverse outcomes as a result of ABI [(a)]

Region and date	Measure of outcome[(b)(c)]	Proportion with specified outcome	Level of severity[(d)]	Time of assessment (relative to time of injury)	Sample size	Source
Virginia, USA, 1977–79	GOS moderate disability or worse: Persistent symptoms: Unemployed (% of those employed before injury):	22% 84% 34%	Minor: loss of consciousness ≤20 minutes, GCS ∫13 and length of stay ≤48 hours	3 months	424	Rimel et al. (1981) Rimel et al. (1982)
Virginia, USA, 1977–79	GOS moderate disability or worse: Persistent symptoms: Unemployed (% of those employed before injury):	61% 96% 69%	Moderate: GCS 9–12	3 months	170	Rimel et al. (1982)
Berkshire, UK, 1992–93	Persisting symptoms: Not returned to work:	86% 5%	Minor: loss of consciousness ≤20 minutes, GCS ∫13, post-traumatic amnesia ≤24 hours, and no complications	3 months	46	Powell et al. (1996)

(a) The definitions of head/brain injury used in many of these studies can be found in Table 2.3.
(b) GOS=Glasgow Outcome Scale.
(c) In many of the studies more than one measure of outcome is used. As the outcome categories are not mutually exclusive, percentages should not be summed,
(d) GCS=Glasgow Coma Scale.

The Glasgow Outcome Scale (GOS) is used as a measure of outcome in a number of the studies cited. An outcome of 'moderate disability or worse' includes moderate disability, severe disability and persistent vegetative state (see Section 2.4). If assessment is made soon after injury many patients might be expected to recover sufficiently over subsequent months to reach a better outcome category on the scale. Assessments made a year or more after injury are more likely to reflect the ultimate level of long-term disability that will be experienced (Jennett & Teasdale 1981).

The three studies that used the GOS to assess people with all levels of initial injury severity gave similar estimates of the proportion of people with moderate disability or worse (3–5%), although the time at which assessment was made varied between studies (Kraus et al. 1984; Servadei et al. 1988; Vazquez-Barquero et al. 1992). Studies that include all levels of injury severity might be more readily comparable with one another than studies that focus on brain injuries of a particular severity. Differing definitions of severity level can add an extra source of variation.

The studies focusing on severe brain injury produced varying estimates of the proportion of people with moderate disability or worse, ranging from 16% to 48% (Annoni et al. 1992; Cuff & Donald 1987; Tate et al. 1989b; Tennant et al. 1995). Surprisingly, in the study reported by Rimel et al. (1981, 1982), 22% of people with mild brain injury and 61% of people with moderate brain injury had moderate disability or worse at the 3 month follow-up. These proportions are substantially higher than those reported for samples that included brain injury of all severities, and even some of the studies looking at severe injury only. These differences suggest that, even using a widely accepted scale for assessing outcome (the GCS) it can be difficult to make valid comparisons between studies.

3.3 Prevalence

There are relatively few existing estimates of the prevalence of long-term disability attributable to ABI, either in Australia or overseas. Information about the incidence of ABI is sometimes used, in conjunction with other information, to calculate prevalence estimates. However, such estimates usually rely on a series of assumptions that are not easily verified. Also, these estimates are subject to the same limitations as the incidence data on which they are based. Prevalence estimates based on population surveys may be more reliable indicators of real prevalence rates. However, operational definitions and methodologies vary between surveys so, as with estimates of incidence, caution should be exercised when comparing estimates from different studies.

Overseas estimates

Table 3.4 presents five overseas estimates of the prevalence of disability attributable to ABI, varying between 62 and 783 per 100,000. Three of the estimates are based on population surveys, and are limited to people living in households. However, the survey methodologies and operational definitions used vary.

The 1983 estimate for China (783) was based on a door-to-door survey in six large cities. People who gave responses indicating unconsciousness following head injury or past or present evidence of focal brain dysfunction resulting from head injury were asked to be examined by a neurosurgeon, who gave a diagnosis based on examination and review of

Table 3.4: Overseas estimates of prevalence rates of disability attributable to ABI [(a)]

Rate (/100,000)	Study location and data collection period	Data sources and methods	Criteria for inclusion	Population	Source
783	China 1983	Population survey, with review of available medical records	History of head trauma with loss of consciousness, post-traumatic amnesia, or clinical evidence of subsequent focal brain dysfunction	People living in households in six large cities	Wang et al. (1986)
62	Canada 1986	Health and Activity Limitation Survey—survey of individuals with disability identified through census question	Limited in normal daily activity; ongoing problems with ability to remember and learn due to injury to brain acquired after birth	People living in households, age 15 and over	Steger Moscato et al. (1994)
100	UK 1982	Population survey	Disabled or handicapped in own or family's eyes; disability caused by head injury	People living in households	Bryden (1989)
100	UK 1988	Cited in a report by the Medical Disability Society	Disabled survivors of brain injury	Total population (unclear in source)	Tennant et al. (1995)
439	USA 1974	Sample survey of hospital data 1970–74, with patient follow-up in 1974	People first hospitalised for head injury during 1970–74 and still alive in 1974 and having received health services treatment related to head injury during last 6 months of 1973 or during 1974[(b)]	Total population	Kalsbeek et al. (1980) (see also Anderson et al. 1980)

(a) See Table 2.5 for more detailed information on operational definitions used in prevalence studies.
(b) The US National Head and Spinal Cord Injury Survey provided an estimate of the 'frequency'. The count included people who were hospitalised for head injury during 1974 (whether or not they suffered ongoing problems as a result) and people who had been hospitalised for head injury during the period 1970–73, were still alive at follow-up in 1974 and were not deemed to have 'recovered'. Recovery was defined as not having received treatment or services associated with head injury from any provider of health care within the past 6 months. The rate was obtained by dividing this count by the average population of the USA in 1974.

available medical records. The operational definition used may have included people who once had a brain injury, but who did not have ongoing sequelae (Wang et al. 1986).

In the Canadian Survey people were initially identified as having a disability through a question in the Canadian census about whether they were limited in 'normal daily living'. The follow-up Health and Activity Limitation Survey then identified people as having ABI if they reported 'ongoing problems with ability to remember and learn', due to injury to the brain acquired after birth. It seems that brain injury due to stroke, disease (e.g. brain tumour, Alzheimer's disease), ageing or developmental delay was excluded (Dawson & Chipman 1995). This fairly narrow definition may in part explain the low estimate. Also, the estimate did not include people with ABI living in establishments. The fact that the estimate was limited to people aged 15 and over should not have resulted in a lower rate, as the prevalence of disability attributable to TBI tends to be highest in the middle adult years (Steger Moscato et al. 1994).

The 1982 estimate for the UK was based on a household population survey carried out in a particularly socioeconomically disadvantaged region in Scotland. The survey identified people with a disability or handicap caused by head injury and was therefore, presumably, limited to traumatic brain injury. Disability or handicap was identified by the question: 'Is there anyone living here whose everyday life is affected by illness, disability or injury, either physical or mental, or by problems due to age (e.g. arthritis, rheumatism or heart trouble), injury, or defect of sight, hearing or mobility?' (Bryden 1989).

The National Head and Spinal Cord Injury Survey in the USA (Kalsbeek et al. 1980) used a very different approach to estimating the 'frequency' of head injury. The data were taken from a sample of hospital records drawn from a sample of hospitals throughout the contiguous United States. The estimate included all people hospitalised for head injury during 1974, plus people who had been hospitalised for head injury during the period 1970–73, were still alive at follow-up in 1974 and were not deemed to have 'recovered'. Recovery was defined as not having received treatment or services associated with head injury from any provider of health care within the past 6 months. Thus, people with ongoing disability who were not accessing health services would have been excluded. People with disability due to a head injury sustained prior to 1970 were also not included. These factors would tend to lead to an underestimate. However, people hospitalised in 1974 were included regardless of whether they experienced ongoing problems as a result of head injury. This would tend to lead to an overestimate, if the rate was considered as a measure of prevalence.

Given the very different definitions and methodologies used it is difficult to draw any conclusions about how 'reasonable' the various estimates presented in Table 3.4 might be. It is likely that real rates of prevalence differ between the countries represented, due to factors such as different levels of interpersonal violence, traffic safety standards, and the quality and availability of acute care and rehabilitation.

Australian estimates

Table 3.5 presents nine existing estimates of ABI prevalence within Australia. Methodologies vary, with six of the estimates based on data from the 1993 ABS disability survey. Four of the estimates (Western Australia 1991, South Australia 1996–97 and the two estimates for Australia 1993) are markedly higher than any of the overseas estimates presented in Table 3.4.

The first estimate of prevalence for the whole of Australia based on the 1993 ABS disability survey includes all people who answered positively to the screening question about long-

term effects of head injury, stroke or other brain damage (ABS 1996b). The second, slightly higher, estimate for Australia (Madden et al. 1995) was arrived at by identifying people who answered positively to the screening question on long-term effects of head injury, stroke or other brain damage and / or reported an ABI-related disabling condition, and reported a limitation, restriction or need for help. (This method is equivalent to the approach based on 'all disabling conditions plus activity limitation' explained in Section 4.3.)

Estimates for Western Australia and the Australian Capital Territory based on the ABS disability survey include only people who reported head injury, stroke or other brain damage as their main disabling condition. People who had long-term effects of head injury, stroke or other brain damage, but reported some other condition as their 'main disabling condition' were not included in these estimates. It should also be noted that these estimates are based on small sample sizes and are subject to relative standard errors of between 25% and 50%.

The Western Australian estimate of 1,696 per 100,000 (Stanton et al. 1994) included only people aged between 16 and 65 and was obtained using a population modelling approach and incidence rates based on hospital data. While it is not perfectly clear from the information published, it would seem to be an estimate of the number of people in the community who have had a brain injury at some point, whether or not they have ongoing disability as a result.

The other two estimates for Western Australia are based on the 1993 ABS disability survey. Rook (1994) stated that 2,700 people in Western Australia with a disability had a main disabling condition of 'head injury / stroke / any other brain damage' (Rook 1994:9). Applying ABS population figures for Western Australia in 1993 gives a rate of 161 per 100,000. This is substantially lower than the '0.4% of all Western Australians' who reported ABI as the main cause of their disability (Alessandri et al. 1996:9). Without further information on the methodology used it is not possible to explain the difference between these two estimates.

The estimate of the prevalence of disability attributable to ABI obtained using data from the South Australian Survey of Disability Prevalence was 1,740 per 100,000. Both the South Australian Survey and the 1993 ABS Survey of Disability, Ageing and Carers used fairly broad definitions, in that ABI was not limited to brain injury resulting from particular causes, and stroke was explicitly included in both surveys. The South Australian Survey definition was perhaps narrower, as it specified brain injury 'resulting in a substantial behavioural change and / or significant memory loss', while the ABS survey did not specify the type of 'long-term effects'. However, the ABS survey did specify that 'long-term effects' meant effects that had lasted or were expected to last 6 months or more, while the South Australian survey definition did not impose a minimum duration requirement. The South Australian estimate is limited to people living in households, while the estimates based on the ABS survey data also include people living in establishments.

3.4 Non-traumatic ABI

Most of the estimates reviewed above, particularly the estimates of incidence, focus primarily on traumatic brain injury. Estimates of the incidence and prevalence of other subgroups of ABI are less easily found in the literature. Below, we briefly review definitions and existing incidence and prevalence estimates for two subgroups of ABI—stroke and alcohol-related brain injury—and mention some other causes of ABI.

Table 3.5: Australian estimates of prevalence rates of disability attributable to ABI

Rate (/100,000)	Study location and data collection period	Data sources and methods	Criteria for inclusion	Population	Source
294[(a)]	Vic 1993	1993 ABS Survey of Disability, Ageing and Carers	Unclear from information published in source	Total population	Honey (1995a)
240–290	Vic	Based on a 'realistic interpretation' of estimates derived from various data sources	Long-term moderate or severe disability	Aged under 65 (unclear in source)	Health Department Victoria et al. (1991)
1,696[(b)]	WA 1991	Hospital admissions data used to determine incidence, then demographic model of WA population used to calculate prevalence based on incidence	List of conditions; alive on discharge; length of stay >1 day; of WA origin	Age 16–65	Stanton et al. (1994)
161[(c)]	WA 1993	1993 ABS Survey of Disability, Ageing and Carers	People with a disability (as defined in the survey) who reported ABI as their main disabling condition	Total population	Rook (1994)
400	WA 1993	1993 ABS Survey of Disability, Ageing and Carers	People with a disability (as defined in the survey) who reported ABI as their main disabling condition	Total population	Alessandri et al. (1996)

(continued)

Table 3.5 (continued): Australian estimates of prevalence rates of disability attributable to ABI

Rate (/100,000)	Study location and data collection period	Data sources and methods	Criteria for inclusion	Population	Source
1,740	SA 1996–97	South Australian Survey of Disability Prevalence—telephone survey	Brain injury caused by e.g. drowning, asphyxiation, stroke or illness resulting in a substantial behavioural change and/or significant memory loss	People living in households	South Australian Health Commission (1998)
134[(d)]	ACT 1993	1993 ABS Survey of Disability, Ageing and Carers	People with a disability (as defined in the survey) who reported ABI as their main disabling condition	Total population	Gilbert (1997)
1,400	Australia 1993	1993 ABS Survey of Disability, Ageing and Carers	People with a disability (as defined in the survey) who gave a positive response to the screening question on long-term effects of head injury, stroke, or any other brain damage	Total population	Australian Bureau of Statistics (ABS) (1996a)
1,920[(e)]	Australia 1993	1993 ABS Survey of Disability, Ageing and Carers	AIHW method: disability (as defined in the survey), plus positive response to relevant screening question and/or reported relevant ICD code, plus 'filter' based on restrictions and limitations	Total population	Madden et al. (1995)

(a) This figure was calculated using the estimated number of people (13,100) published in the source and ABS population data for Victoria as at March 1993.
(b) This figure was calculated using the estimated number of people (17,843) and population data for WA in 991 published in the source.
(c) This figure was calculated using the estimated number of people (2,700) published in the source and ABS population data for WA as at March 1993.
(d) This figure was calculated using the estimated number of people (400) published in the source and ABS population data for the ACT as at March 1993.
(e) This figure was calculated using the estimated number of people (338,469) published in the source and ABS population data for Australia as at March 1993.

Stroke

Stroke is the second most common cause of death in Australia, after coronary heart disease, and is an important cause of disability (AIHW 1999b). While stroke is a cause of death and disability in people of all ages, it occurs most commonly in the later years of life. In older people disability tends to result from multiple causes. Therefore, it can be difficult to distinguish disability caused by stroke from disability caused by other conditions, both in individuals and in epidemiological studies (Campbell et al. 1994).

Definitions

The definition of stroke used by the World Health Organization (WHO) is 'sudden onset of clinical signs of focal or global disturbance of cerebral function lasting more than 24 hours (except in cases of sudden death or if the development of symptoms is interrupted by a surgical intervention) with no apparent cause other than vascular' (The WHO Monica Project 1990, cited in Sarti et al. 1994). Many studies of stroke epidemiology have used the WHO definition, or definitions based closely on it (e.g. Christie 1982; Wolfe et al. 1993).

Table 3.6: ICD–9 diagnosis codes for cerebrovascular conditions

3-digit code	Description
430	Subarachnoid haemorrhage
431	Intracerebral haemorrhage
432	Other and unspecified intracranial haemorrhage
433	Occlusion and stenosis of precerebral arteries
434	Occlusion of cerebral arteries
435	Transient cerebral ischaemia
436	Acute, but ill-defined, cerebrovascular disease
437	Other and ill-defined cerebrovascular disease
438	Late effects of cerebrovascular disease

Source: National Coding Centre (1995)

In some studies ICD–9 codes are used to identify stroke in coded hospital morbidity and mortality data (see Table 3.6). However, the range of codes included differs between studies. Codes 430–438 cover all cerebrovascular disease and its after-effects (Bennett et al. 1994), but a smaller subset can be used to identify acute 'stroke' events. An Australian study looking at rehabilitation after stroke used ICD–9 codes 430–436, excluding 437 and 438 (Shah et al. 1991). Tuomilehto et al. (1993) defined stroke using ICD–9 codes 430–434, and 436, excluding 435, 437 and 438. A transient ischaemic attack (TIA—included within code 435) is an episode of acute neurological deficit that resolves completely within 24 hours (Toole 1994), and therefore does not come within the WHO definition of stroke. However, mild but definite cognitive deficits have been documented in some TIA patients (Toole 1994).

Epidemiology of stroke and stroke-related disability

Incidence

Incidence of stroke can be affected by the age, sex and race mix of a population. Estimates of incidence can also vary depending on whether only first-ever-in-a-lifetime strokes or all strokes are counted, whether transient ischaemic attacks are included, and what diagnostic criteria are used (US Department of Health and Human Services 1995).

Estimates of stroke incidence based solely on hospital admissions are likely to be underestimates, as not all people who have a stroke are admitted to hospital (Bonita et al. 1984). The Perth Community Stroke Study found that 20% of all stroke events were managed entirely outside hospital, and that the likelihood of being admitted to hospital after a stroke decreased with increasing age (Anderson et al. 1993). In developed countries hospital admission may reflect a person's living arrangements and the extent to which help is available to them at home, as well as the severity of their stroke (Poungvarin 1998). Many studies, therefore, use information from a variety of sources (e.g. GPs, hospitals, nursing homes, health centres) so that the majority of stroke events occurring within a community are identified.

In Table 3.7 overseas and Australian estimates of stroke incidence rates are given. In all cases where it was specified, the working definition of stroke used was the WHO definition, or a definition based closely on it. However, methodology and the population age range considered differed between studies, so comparisons should be made with caution.

In addition to the estimates presented in Table 3.7, Aho et al. (1980) presented estimates of stroke incidence for 14 countries, based on a study coordinated by the WHO in which data were collected in the early 1970s through registers set up at local hospitals or health centres. Age-standardised estimates of incidence (all attacks) ranged from 189 per 100,000 per year in Sri Lanka to 1,344 per 100,000 in Japan. Warlow (1998), in a review of stroke epidemiology, gave an annual incidence estimate of 200 per 100,000 first-ever-in-a-lifetime strokes, based on studies in 'various white populations'. In general, first-ever strokes account for 70–80% of all stroke events (Anderson et al. 1993; Bonita et al. 1984, 1994; Sarti et al. 1994).

Of the three Australian estimates, the Melbourne figure of 380 per 100,000 is the highest for incidence for all stroke events (Christie 1981). However, the Melbourne study included only people aged over 25, while the Sydney and Perth studies included people of all ages (Fisher et al. 1979; Anderson et al. 1993). Including people aged 25 and under would be expected to produce a lower overall estimate, as few cases of stroke are likely to occur in this age group—their inclusion will increase the denominator without a proportionate increase in the numerator. The three studies also differed in terms of the number of data sources used (GPs, hospitals, nursing homes, etc.), which may also have contributed to differences in estimated incidence.

In both the Melbourne and Perth studies data were collected over a period of 18 months. As some studies have found that stroke incidence varies with season (e.g. Giroud et al. 1989), collecting data over periods that are not multiples of 12 months may bias annual incidence rates.

Several studies have revealed substantially higher rates of stroke in men than in women (Anderson et al. 1993). Data from the Finland study indicated a male to female rate ratio of around 1.8 for both 'first-ever' and all strokes (Sarti et al. 1994). Aho et al. (1980) reported male to female rate ratios for first-ever stroke ranging from 1.0 in Israel to 2.0 in Japan. In

Table 3.7: Overseas and Australian estimates of stroke incidence

Incidence (/100,000 per year)		Study location and data collection period	Data sources and methodology	Early mortality	Population	Source
All strokes	**First ever**					
Overseas estimates						
346 (m) 193 (f)	269 (m) 154 (f)	Finland 1983–85	Data collected from hospital admission/discharge diagnoses and death certificates	30% (m) 26% (f) (4 weeks, % all strokes)	People aged 25–74	Sarti et al. (1994)
326 (m) 180 (f)	252 (m) 143 (f)	Finland 1987–89	Data collected from hospital admission/discharge diagnoses and death certificates	21% (m) 24% (f) (4 weeks, % all strokes)	People aged 25–74	Sarti et al. (1994)
145		Dijon, France 1985–87	Stroke registry of Dijon—data from hospitals and GPs	12.5%—first week 21.5%—first month 30%—first year	Total population	Giroud et al. (1989)
224	170	New Zealand, 1981	Data collected from hospitals, GPs, death certificates, rest homes, locum and emergency services	33.5% (1 month) 43.5% (6 months) 48.5% (1 year) (% of first events during study period)	People aged 15 and over	Bonita et al. (1984)
195	142	New Zealand, 1991	Data collected from GPs, hospital medical staff, private physicians and supervisors of hostels and nursing homes	All strokes: 24% at 4 weeks	Age-standardised to world population aged $\geq$ 15	Bonita et al. 1994
	330	Taiwan, 1990	Population study, with annual follow-up over 4 years. Stroke confirmed by physician	17.3% (1 month, % first strokes)	People aged > 35	Hu et al. (1992)

(continued)

Table 3.7 (continued): Overseas and Australian estimates of stroke incidence

Incidence (/100,000 per year)		Study location and data collection period	Data sources and methodology	Early mortality	Population	Source
All strokes	**First ever**					
	160	Oxfordshire, UK 1981–86	Data collected from GPs and hospitals, and patients were assessed by neurosurgeon to confirm stroke	19% (1 month) 31% (1 year)	Total population	Bamford et al. (1990), Bamford et al. (1988)
	6.3 (age <45) 35 (age 45–54) 149 (age 55–64) 397 (age 65–74)	England 1989–90	Data collected from GPs, district nursing and rehab services, hospital admissions and death certificates	26% (3 weeks, % first strokes)	People aged under 75	Wolfe et al. (1993)
	200		Review of incidence rates in white populations	20% (1 month, % first strokes)	Total population	Warlow (1998)
Australian estimates						
380		Melbourne, Victoria 1978–79 (18 months)	Data collected from GPs, hospitals and ambulance calls	24% (3 weeks, % all strokes)	People aged over 25	Christie (1981)
205	160	Sydney, NSW 1979–80	Data collected from GPs and hospital records	33% (12 weeks, % all strokes)	Total population	Fisher et al. (1979)
258	178	Perth, WA 1989–90 (18 months)	Data collected from GPs, hospital medical staff, private physicians and supervisors of hostels and nursing homes	All strokes: 24% at 4 weeks 39% at 1 year First ever strokes: 23% at 4 weeks 36% at 1 year	Total population	Anderson et al. (1993)

Australia, while men have higher rates of stroke, greater numbers of women are affected by stroke because more women than men live into old age (AIHW 1999b).

Stroke incidence rates also increase dramatically with age (Aho et al. 1980; Bamford et al. 1988; Bonita et al. 1994; Giroud et al. 1989; Wolfe et al. 1993). A projection study conducted by the National Health and Medical Research Council (NHMRC) in 1996 suggested that, '[a]ssuming stable incidence rates and patterns of care, the changing age / sex structure of the population is expected to result in a 69% increase in the number of new cases of stroke per year' (National Stroke Foundation 1997).

Prevalence

Prevalence of stroke survivors in a community depends on rates of incidence and mortality. As with ABI generally, a distinction must be drawn between the prevalence of people who have ever had a stroke, and the prevalence of people who have some ongoing disability resulting from stroke. Measuring the prevalence of stroke-related disability is difficult because co-morbidities, such as osteoarthritis and dementia, are common in older people and make it difficult to establish the extent to which stroke contributes to the overall level of disability (Warlow 1998).

Estimates of the prevalence of people who have ever experienced a stroke vary considerably (Table 3.8). Again, this may partly reflect the different age ranges to which the estimates apply. The estimate range for India (90–222 per 100,000) is substantially lower than the others given in Table 3.8. The estimate for Taiwan (1,642) is substantially higher, but it relates to an older population (people aged over 35) than any of the other estimates. The estimates reported by Wade (1988) for the UK indicate that 50% of people in the community who have ever had a stroke have 'significant problems' as a result. The New Zealand study found that slightly over half of all people who have ever had a stroke make an 'incomplete recovery', and about 20% require assistance in at least one area of self-care (Bonita et al. 1997). The data also indicated that, among stroke survivors, more women (27%) were dependent on others for self-care activities than men (16%) (Bonita et al. 1997).

The three Australian prevalence estimates fall within the range of the overseas estimates presented. The estimate of 990 per 100,000 was based on the 1995 National Health Survey, a 5 yearly population survey that collects self-reported information on the health status of Australians. The National Health Survey does not provide information on people living in establishments (i.e. nursing homes, etc.). The estimate covers all health conditions coded to ICD–9 codes 430–438, including 435 (transient cerebral ischaemia)—a more inclusive definition than those used in most of the other studies presented in Table 3.8.

The Victorian estimate was from a community-based stroke study in which people who had a stroke were followed up at intervals for two years. Survival rates over the 2 year period were used to calculate the estimated community prevalence of 792 per 100,000. This, along with information about disability experienced by survivors, was used to produce an estimate of the prevalence of disability attributable to stroke—slightly over 200 per 100,000. Disability was defined as 'not being independent in all [activities of daily living] and / or not being able to walk at least 100 metres unaided' (Christie 1981).

The Perth estimate of 1,200 per 100,000 was based on the Perth Community Stroke Study. Methods of estimation were not detailed in the source (NHMRC 1997).

Table 3.8: Overseas and Australian estimates of the prevalence of stroke and disability attributable to stroke

Prevalence (/100,000)					
Ever had stroke	**Disability**	**Location and date**	**Data sources and methodology**	**Population**	**Source**
Overseas estimates					
820	623	Finland, 1973–76	Follow-up examination—part of prospective population study (only stroke treated in hospitals, health centres and homes for the elderly detected)	People aged 20 and over	Aho et al. (1986)
90–222		India	Not specified	Total population	Cited in Poungvarin (1998)
833	461 (incomplete recovery) 173 (ADL[(a)] disability)	Auckland, New Zealand, 1992	Estimates derived from two population-based incidence studies (1981–82 and 1991–92) using an actuarial model	People aged ≥ 15	Bonita et al. (1997)
1,642	540 (ADL[(a)])	Taiwan, 1986	Population-based study	People aged >35	Hu et al. (1989)
690		Bangkok, Thailand	Population survey with professional medical examination to confirm stroke	People aged >20	Viriyavejakul et al. (1983)

(continued)

Table 3.8 (continued): Overseas and Australian estimates of the prevalence of stroke and disability attributable to stroke

Prevalence (/100,000)					
Ever had stroke	**Disability**	**Location and date**	**Data sources and methodology**	**Population**	**Source**
Overseas estimates (continued)					
831		UK, 1985	Office of Population Censuses and Surveys disability survey data	Total population	Clark & Opit (1994)
600	300	UK	Not specified	Total population	Wade (1988)
Australian estimates					
792	200	Victoria, 1979	Incidence data collected from GPs, hospitals and ambulance calls. Follow-up of patients at intervals to 2 years post-stroke used to calculate prevalence	People aged >25	Christie (1981)
1,200		Perth, 1990	Based on Perth Community Stroke Study—methods not detailed in source	Total population	NHMRC (1997)
990		Australia, 1995	National Health Survey—stroke and other cerebrovascular disease reported by respondents and classified and coded by ABS as ICD–9 codes 430–438	People aged 25 and over	AIHW analysis of 1995 National Health Survey data

(a) ADL = activities of daily living.

Alcohol-related brain injury

Alcohol-related brain injury (ARBI) is 'physical injury sustained by a part or parts of the brain, as a result of excessive consumption of alcohol' (ARBIAS 1996). Although reliable estimates of the incidence and prevalence of ARBI are particularly difficult to obtain, there is some evidence to suggest that rates of ARBI are higher in Australia than in other comparable countries (Connelly 1993; Harper et al. 1989). ARBI is recognised to be a major cause of ABI-related disability, particularly in the middle-adult years (Honey 1995b).

As well as being a primary cause of ABI, alcohol can be a risk factor for traumatic brain injury (TBI). The association of alcohol with TBI (particularly due to road accidents) has been well documented (Kraus 1987). Rimel et al. (1981, 1982) found that the proportion of brain-injured patients who had positive blood alcohol levels at admission to hospital increased with injury severity—43% of patients with mild injury, 73% of patients with moderate injury, and 84% of patients with severe injury. Mean blood alcohol level also increased with injury severity.

Intoxication can interfere with diagnosis and severity assessment after TBI, as alcohol tends to depress consciousness, as measured using the Glasgow Coma Scale (Kraus 1987). This may hamper physicians in prescribing appropriate management for brain-injured patients during the critical phase of care. There is also a suggestion that alcohol abuse can result in more severe damage and poorer outcome in the event of TBI (Levin 1989; R nty et al. 1993).

Characteristics of ARBI

The mechanisms by which excessive alcohol consumption brings about brain damage are likely to be several, and are not yet properly understood. Both the direct neurotoxic effect of alcohol and thiamine (Vitamin B1) malnutrition that commonly accompanies alcoholism are thought to contribute to alcohol-related brain damage (Tuck & Jackson 1991). Some types of neurological damage caused by alcohol abuse seem to be at least partially reversible (Oscar-Berman et al. 1997).

In its 'Guide to general practitioners and health professionals', the ARBI services and support organisation ARBIAS identifies six disorders commonly associated with ARBI: cerebellar atrophy, peripheral neuropathy, hepatic encephalopathy, frontal lobe dysfunction, Wernicke's encephalopathy and Korsakoff's amnestic syndrome (ARBIAS 1996). Wernicke's encephalopathy, an acute neurological illness caused by severe thiamine deficiency, and Korsakoff's amnestic syndrome, a chronic disorder of cognitive function, seem to be associated conditions, though the relationship between them is not fully understood. The term 'Wernicke–Korsakoff syndrome' (WKS) is frequently used in the literature, though its specific meaning is somewhat unclear.

ARBI is similar to other forms of ABI in terms of the types of impairment that commonly result (Honey 1995b). Alcoholics tend to exhibit fairly circumscribed patterns of cognitive deficit, rather than global impairment. Characteristic types of impairment include disturbances of executive function (e.g. difficulties with planning, problem solving), memory impairment, disorders of awareness (e.g. denial, lack of motivation), and emotional problems (e.g. confusion and anger) (ARBIAS 1996).

Estimates of incidence and prevalence

Obtaining estimates of the proportion of the population affected by ARBI is especially challenging because of underdiagnosis. The particular stigma attached to ARBI may affect the willingness of individuals to identify as having ARBI. Also, relatively few cases of ARBI are treated in the acute care system, and there is no other common point for collection of data. Even when individuals do come into contact with the health care system their condition may not be recognised, as diagnosis involves comprehensive neuropsychological and neurological assessment—there is no easily administered screening tool (Honey 1995b). The earliest signs of cognitive impairment are subtle and doctors are unlikely to suspect brain damage until it is well established (Tuck & Jackson 1991).

However, hospital-based studies have been used to obtain estimates of incidence and / or prevalence. Table 3.9 presents some estimates of rates of ARBI in Australia and overseas. The methods used for obtaining estimates vary between studies. Other estimates not reported here are referred to in the sources cited.

Overseas estimates

Thompson et al. (1988) estimated the proportion of the population of England and Wales affected by alcohol-related brain injury at 2% (Table 3.9). This figure was based on evidence from previous studies that about 50% of heavy drinkers showed signs of cognitive impairment, and that 4% of the adult population of England and Wales were heavy drinkers (Thomson et al. 1988).

Victor and Laureno (1978) provided two estimates of the prevalence of Wernicke–Korsakoff syndrome in the USA based on hospital admissions—cases were identified by the presence of a combination of characteristic clinical symptoms (e.g. ataxia of gait, mental confusion). The estimates of 130 per 100,000 (Boston) and 50 per 100,000 (Massachusetts) represent the proportion of all hospital admissions. The estimates of 2,200 per 100,000 (for Wernicke's encephalopathy) and 4,100 per 100,000 (for cerebellar atrophy) are proportions of all autopsies conducted at a major hospital in Cleveland over a 13 ½ year period.

The estimates of 800 per 100,000 for Wernicke's encephalopathy and 1,700 per 100,000 for cerebellar atrophy in Norway also represent the proportion of all autopsies conducted (Torvik et al. 1982). The authors noted that alcohol consumption in Norway is considerably lower than in most other western countries.

Australian estimates

In a retrospective study of medical records in 17 public general hospitals in Sydney between 1978 and 1993, cases of Wernicke's encephalopathy, Korsakoff's psychosis, and WKS were identified using ICD–9 codes and specified diagnostic criteria. A total of 1,267 cases were identified, including 10 non-alcoholic patients with WKS. Over the 16-year period, rates of WKS (calculated as a proportion of all hospital admissions) decreased from about 32 per 100,000 in 1978 to 23 per 100,000 in 1993. Thiamine enrichment of bread flour (mandatory in Australia since 1991) and a decrease in national alcohol consumption were discussed as possible contributors to the decrease (Ma & Truswell 1995).

Gold et al. (1986) conducted a 12-month hospital surveillance program in an inner urban area of Sydney. They produced an estimate for the 'attack rate' (presumably meaning 'incidence') of cerebral alcohol syndrome of 38 per 100,000 per year. Cerebral alcohol syndrome was used as a blanket term to mean 'the spectrum of cognitive dysfunction associated with chronic alcohol abuse'.

Table 3.9: Estimates of the proportion of the population affected by ARBI, overseas and in Australia

Rate (per 100,000)	Location, date and population	Data sources and methods	Source
Overseas			
2,000	England and Wales 1980s (adult population)	Information on the prevalence of cognitive impairment in alcoholics combined with information on the proportion of adults who drink heavily	Thomson et al. (1988)
130	USA	Admissions to single major general hospital with Wernicke–Korsakoff syndrome	cited in Victor & Laureno (1978)
50	USA	Admissions to single major general hospital with Wernicke–Korsakoff syndrome	cited in Victor & Laureno (1978)
Autopsy studies (calculated as proportion of autopsies performed)			
800 (Wernicke's encephalopathy) 1,700 (cerebellar atrophy)	Oslo, Norway 1975–79	Autopsies from hospitals in the Oslo area	Torvik et al. 1982
2,200 (Wernicke's encephalopathy) 4,100 (cerebellar degeneration)	USA, 1963–76	Post-mortem material from single major general hospital	Victor & Laureno (1978)
Australia			
38 ('cerebral alcohol syndrome')	NSW, 1981 (age >15)	Condition identified in hospitalised patients, confirmed by neuropsychological screening. Rates calculated as proportion of population aged >15	Gold et al. (1986)
23 (Wernicke's encephalopathy and/or Korsakoff's psychosis)	NSW, 1993	Condition identified through hospital records. Rates calculated as proportion of all admissions	Ma & Truswell (1995)
Autopsy studies (calculated as proportion of autopsies performed)			
2,800 (Wernicke's encephalopathy)	WA, 1973–81 (age > 20)	Autopsy study (131 cases identified)—forensic and hospital autopsies	Harper (1983)
2,100 (Wernicke–Korsakoff syndrome)	NSW, ? (age >15))	Autopsy study (6 cases identified)—forensic and hospital autopsies	Harper et al. (1989)
1,100 (Wernicke–Korsakoff syndrome)	NSW, 1996–97 (age >15)	Autopsy study (25 cases identified)—forensic autopsies only	Harper et al. (1998)

The remaining three Australian estimates presented are based on autopsy studies. A study of 4,677 brains of patients aged over 20 between 1973 and 1981 in Western Australia revealed 2.8% (2,800 per 100,000) with Wernicke's encephalopathy. This 2.8% was described as 'incidence'. The rate was substantially higher among coroners' necropsies (4.7%) than among necropsies performed at the Royal Perth Hospital (1.7%). For the combined sample, incidence was highest for people aged in their fifties, and 75% of the cases identified were in males. A review of medical records indicated that in at least 90% of the cases alcoholism was the cause. Only 20% of the cases had been clinically diagnosed (Harper 1983).

Two similar autopsy studies were conducted in Sydney (Harper et al. 1989; Harper et al. 1998). In these, the rates obtained were reported as 'prevalence' rather than 'incidence'—probably a more accurate description. In the 1996–97 study, based on autopsies from the New South Wales Institute of Forensic Medicine, 25 cases of WKS were identified among the 2,212 brains examined—a prevalence of 1.1% (Harper et al. 1998). This rate was compared with the rate of 4.7% for coroners' necropsies from the 1973–81 Western Australian study (no comparison with the previous New South Wales study was made, possibly because of the small sample size in that study). The authors suggested that the significant reduction in prevalence might have been due to the fortification of bread flour with thiamine.

Because of the different methodologies used, a direct comparison of estimates from clinical studies and autopsy studies is not appropriate. As well as different approaches to the identification of ARBI cases, rates were calculated in quite different ways. In the three autopsy studies rates of WKS were calculated as a proportion of all autopsies. The two clinical studies used very different approaches to calculating rates—Ma and Truswell (1995) reported WKS as a proportion of all hospital admissions, while Gold et al. (1986) used the population of the study area as the denominator. Thus the populations being considered in these various studies were at very different levels of risk for WKS.

Nonetheless, it is somewhat surprising that the two clinical studies produced rates so similar in magnitude, and so much lower than the rates from the autopsy studies. One explanation for this may be the high level of under-diagnosis of Wernicke–Korsakoff syndrome in living patients (Harper 1983). Also, the clinical studies included a cross-section of people at different stages of life. Many people included may not have had ARBI at the time of the study, but may have developed it in later life. In contrast, the autopsy studies included only people at the end of life, and thus provide what is essentially a cumulative prevalence estimate, or a measure of the lifetime 'risk' of acquiring ARBI.

Other causes of ABI

Neurological diseases such as multiple sclerosis can cause ABI, although, as mentioned in Section 2.1, disability resulting from such causes may be grouped as neurological disability rather than ABI. Jacobson et al. (1997) used incidence and prevalence rates from numerous studies conducted in western countries and published between 1965 and 1995 to estimate the population burden of multiple sclerosis. They calculated a pooled (weighted mean) prevalence rate of 58.3 per 100,000, and a pooled (weighted mean) incidence rate of 3.2 per 100,000. Their review suggested that there had been an increase in the prevalence of multiple sclerosis over the 30-year period.

McLeod et al. (1994) reported results of a 1981 study looking at the prevalence of multiple sclerosis in Australia. Age-standardised prevalence varied markedly across Australia between 11.8 per 100,000 in tropical Queensland and 75.6 per 100,000 in Hobart—these results supported a previously noted increase in prevalence with increasing latitude. Females were two to three times more likely to have multiple sclerosis than males. The

authors did not report on levels of disability. In Australia age of onset for multiple sclerosis is generally between 20 and 50 years, and the mean duration of the disease (from onset to death) is more than 25 years (Hammond et al. 1988).

Diseases such as dementia (e.g. Alzheimer's disease) and Parkinson's disease are important causes of ABI, particularly in older people. It would seem that stroke survivors are at particularly high risk of developing dementia. Results from an Italian population survey found dementia present in about 8% of people aged 65 years or over. However, dementia was much more common in people who had a history of stroke (30%) than in people who had never had a stroke (6%) (Prencipe et al. 1997). Similarly, a survey of people aged 75 years and older in Stockholm found that rates of dementia were three times higher among people with a history of stroke (Zhu et al. 1998). Given its high prevalence in older people dementia is a substantial contributor to the overall impact of ABI.

Volatile substance abuse is another cause of brain injury, particularly among young people. Substances inhaled include petrol, glue and other hydrocarbon products such as liquid correction fluid. Petrol sniffing is recognised as an important cause of sickness and death in some Aboriginal communities in Australia (Brady 1992). Goodheart and Dunne (1994) conducted a study of 25 patients admitted to the Royal Perth Hospital between 1984 and 1991 with a diagnosis of intentional petrol sniffing. Eight of the 20 'chronic' petrol sniffers died while in hospital. Results of neuropathological examination of these eight patients showed abnormalities in all cases. Of the survivors, only one was functionally independent at discharge. While the short-term effects of inhalation may be attributable to several constituents of petrol, the long-term effects are thought to be largely a result of organic lead poisoning. The 11 patients with particularly high blood lead levels had very poor outcomes—eight died in hospital and the remaining three were left with moderate to severe handicap.

There are other important causes of ABI which we are, unfortunately, unable to review here, due to space limitations and the general lack of information available in the published literature. Honey (1995a) has reviewed data available on ABI caused by tumour, hypoxia and infection, and has discussed some of the difficulties associated with gathering data on these subgroups of ABI.

3.5 Summary of estimates reviewed

Most of the estimates of ABI incidence and prevalence reviewed in this chapter relate to traumatic brain injury. Though some estimates of rates of other types of ABI are available in the literature, differences in definition and methodology make it impossible to construct a picture of the overall impact of ABI in Australia or overseas. Even for TBI, on which numerous studies have been conducted, it is very difficult to put forward a 'best estimate' for incidence or prevalence.

Hospital separation data, while they do not provide information on incidence as such, are collected in a systematic manner across Australia. These data may provide a reasonable basis for monitoring the level and demographic pattern of demand for acute care services associated with traumatic brain injury, and perhaps other ABI subgroups. There is patchy information on the proportion of people who experience a traumatic brain injury or stroke who go on to have long-term disabilities. However, definitional problems are compounded here by questions about when and how to measure 'outcome' and what constitutes disability.

The 1993 ABS Survey of Disability, Ageing and Carers is the most commonly used source of data for estimating the prevalence of ABI, although estimates vary depending on the operational definition employed (e.g. whether 'main disabling condition' or 'all disabling conditions' is used—Table 3.5). Despite its limitations (discussed further in Section 4.3) the ABS survey is the best source of disability prevalence data currently available in Australia.

More detailed information about the nature and level of support needed by people with ABI, at the population level, is likely to rely on first establishing reliable estimates of incidence and prevalence.