Chronic subdural hematoma: surgical management in 133 patients

RICHARD G. ROBINSON, CH.M., F.R.C.S.

Department of Surgery, University of Otago Medical School, and Neurosurgical Unit, Dunedin Hospital, Dunedin, New Zealand

One hundred and thirty-three patients with chronic subdural hematoma were treated surgically between 1943 and 1980. The patients, aged 5 to 84 years, were graded retrospectively according to the Bender scale; 28% were in Grades 3 and 4. There were 107 unilateral and 26 bilateral hematomas. The clots were removed mostly via burr-holes without drainage. The treatment of 121 patients included an active policy of brain expansion at operation and the postoperative management of intracranial hypotension by lumbar injection. Two patients died, for a mortality rate of 1.5%. The patients who died were 54 and 59 years old, both from among the 26 cases with bilateral lesions; 107 unilateral lesions were treated, with no deaths. None of 51 patients who were aged 61 years and over died. The mean postoperative stay was 17.2 days, and at 3 weeks 77% had been discharged home. Fifteen percent of survivors had permanent disabilities. The common residual deficits were personality and memory disorders, and there was hemiparesis in Grade 4 cases. The high-risk groups of chronic subdural hematoma were those in Grades 3 and 4, bilateral hematomas, and the elderly. These seemed to be benefited by brain inflation and lumbar injections for intracranial hypotension.

KEY WORDS chronic subdural hematoma □ clinical grading □ outcome □ brain inflation □ intracranial hypotension □ high-risk groups

No neurosurgical condition has such potential for cure as chronic subdural hematoma, yet only too often the results have left much to be desired. Many of the difficulties have been due to delay in diagnosis, but even after operation some patients have not done well or have deteriorated and died. Apart from brain-stem hemorrhages, one well recognized problem has been the failure of the compressed brain to expand and for this to persist, so that distortion of the brain stem and its dysfunction would be responsible for the patient's poor progress. This could be compounded by intracranial hypotension which itself can produce stupor. It was to combat these that LaLonde and Gardner introduced to the English-speaking world in 1948 the advantages of the lumbar or ventricular injection of saline solution at the time of operation and afterward. Munro of Boston adopted this technique, and one of the purposes of the formulation of artificial cerebrospinal fluid (CSF) in 1950 was for use in chronic subdural hematoma. In 1952, we instituted a policy of brain inflation when indicated at the time of operation and treated intracranial hypotension actively in the postoperative period. This series reports the total experience of a neurosurgical unit in handling 133 patients with chronic subdural hematoma, most of whom were managed with this policy.

Summary of Cases

Review Methods

The records of all patients with chronic subdural hematoma, aged 5 years and over, who were admitted to the Neurosurgical Unit, Dunedin Hospital, from its opening in May, 1943, to August 31, 1980, were examined. Those referred for secondary treatment were excluded. A chronic lesion was defined as having an outer and inner membrane, however thin, on inspection at operation. From the beginning, a standard form for recording clinical data was used for all admissions. The Unit is a reference center covering a wide area, and many patients came from hospitals 370 km away or more.

The patients were graded retrospectively according to the criteria of Bender (Table 1). All were followed after discharge from the hospital at peripheral clinics until either they had returned to their previous occupations or their condition had stabilized.
counted for 28% of the patients.

Management

The practice of the Unit was to deal with the hematoma through burr-holes. At first these were performed under local anesthesia but in the last 15 years, neuroleptanalgesia given by an anesthetist was usual. At first the classic four-burr-hole technique was used but latterly two burr-holes sufficed. With a unilateral hematoma, the contralateral burr-hole was used for ventricular puncture when indicated. The subdural space was routinely washed out with copious amounts of warm Ringer’s solution, administered through a soft No. 8F catheter inserted in all directions, until the washings were clear. Since 1952, if the brain did not expand to within about 1 cm of the periphery, Ringer’s solution was injected into a convenient anterior horn of the ventricle until this had been achieved, which might require up to 150 ml of fluid. In the first few patients, the inflation was performed by lumbar puncture while the burr-holes were open, but this meant repositioning the patient.34 The subdural space was drained very rarely. Most of the operations were carried out by the staff neurosurgeons.

Postoperatively, the patients were cared for in a special-care unit, often nursed flat and kept bedfast until clinical recovery was assured. A generous fluid intake of least 2500 ml in adults was maintained by mouth or by nasogastric tube. Steroids were not used consistently even when they became available and no comment can be made as to their value. All complicating diseases were treated by methods standard at the time.

Electrolytes were monitored when necessary, and after the mid-1960’s, hyponatremia was managed by fluid restriction. If the expected clinical recovery was not maintained or the patient deteriorated, and we had a high index of suspicion, a lumbar puncture was done. When the CSF pressure was below 90 mm H2O, enough warm Ringer’s solution was injected to give a pressure of 200 mm H2O before the needle was withdrawn. If clinical progress was not satisfactory, lumbar injections were continued daily until the patient’s recovery seemed certain. When the surgeon thought that there might be a reaccumulation of the hematoma, the burr-holes were reopened under local anesthesia. Guidance might be sought from carotid arteriography.

Results

Patient Analysis. There were 133 consecutive patients with chronic subdural hematoma, 108 were male and 25 female, with the proportion of women rising with age (after the age of 70 years there was a 1:1 ratio). The ages ranged from 5 to 84 years: three were aged 5 to 10 years; four were 11 to 21 years; 16 were 21 to 30 years; 22 were 41 to 50 years; 30 were 51 to 60 years; 32 were 61 to 70 years; 17 were 71 to 80 years; and two were over 80 years.

Table 1 shows the retrospective clinical grading using the Bender scale. Twenty-eight percent (35) were very drowsy or worse before operation in Grades 3 and 4. There were 107 unilateral hematomas, 55 on the right and 52 on the left. Twenty-six (19.5%) of the hematomas were bilateral. All were surface lesions in the frontoparietal area. There were no differences in grading between the bilateral and unilateral cases.

Treatment Analysis. Burr-holes were used in 123 patients, of whom 44 had three or more holes placed. There were 10 primary bone flaps, only three were made after 1960, and two of these were for clotted lesions.

There were 121 patients managed after the introduction of operative inflation, and this procedure was done on 87 occasions (71.9%). Rather more inflations were performed in cases with bilateral hematomas, 20 of 24, and of the four patients without operative inflation one died. Inflation caused complications in two patients: in one patient there was a temporary worsening of the state of consciousness, and the other developed a transient hemiparesis. Both recovered fully. In both patients, complications were due to displacement of the needle at the time of injection after the ventricle had been located.

Because of unsatisfactory progress and when any degree of intracranial hypotension was found at lumbar puncture, spinal injection was performed in 24 patients (19.8%) of the 121 managed after the inflation technique had been introduced. All except one had received brain inflation at the time of surgery. A greater proportion of those aged 50 years and over had a spinal injection. There were two categories of intracranial hypotension: 11 patients had a small reduction of pressure but did not need further injection; and 13 patients (10.7%) had severe intracranial hypotension with drowsiness and CSF pressures hovering around 0 mm Hg for 3 to 10 days. Six patients with severe intracranial hypotension had bilateral hematomas. Preoperative grading was no guide to the occurrence of severe intracranial hypotension as eight were in Grade 2, four in Grade 3, and one in Grade 4. Their ages ranged from 35 to 72 years, and eight were over the age of 60 years.

TABLE 1

Retrospective clinical grading of chronic subdural hematoma on admission in 133 patients*

<table>
<thead>
<tr>
<th>Clinical Grade</th>
<th>Description</th>
<th>No. of Cases</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>fully alert and conscious; normal mental function; few or no focal neurological signs</td>
<td>59</td>
</tr>
<tr>
<td>2</td>
<td>drowsy or lethargic; organic mental syndrome; focal neurological signs</td>
<td>39</td>
</tr>
<tr>
<td>3</td>
<td>very drowsy or stuporous; conspicuous organic mental symptoms; pronounced focal neurological signs</td>
<td>15</td>
</tr>
<tr>
<td>4</td>
<td>coma or signs of hernia</td>
<td>20</td>
</tr>
</tbody>
</table>

*Grading according to the Bender scale. Grades 3 and 4 accounted for 28% of the patients.
There was one exceptional patient of 49 years with bilateral hematomas in whom the state of consciousness could not be stabilized over 10 days. In that patient a fine indwelling catheter was inserted into the lumbar subarachnoid space and repeated injections of Ringer's solution were needed for another 10 days. Ultimate recovery was excellent. No adverse clinical effects were noted in any patient during or after the lumbar injections.

Twelve patients were reexplored for the possibility of a recurrent hematoma. In 10 patients the burr-holes were reopened and in two the bone flaps were raised. Although some degree of brain collapse was found in most patients, none had a frank hematoma and the subdural fluid was serosanguinous.

Other complications were few. One patient developed a subdural infection that was treated successfully by drainage and antibiotic therapy, and one with an infected secondary bone flap had the bone removed.

The duration of hospital stay from the day of the operation to discharge home was as follows: eight were hospitalized for 7 days or less; 63 for 8 to 14 days; 30 for 15 to 21 days; 16 for 22 to 28 days; and 14 for over 28 days. The mean stay was 17.2 days. Patients were discharged home within 2 weeks of surgery in 54% of cases and by 3 weeks in 77%.

Two of the 133 patients died in the hospital, a mortality rate of 1.5% (Table 2), but no deaths were associated with the 107 unilateral lesions. The mortality rate for bilateral hematomas was 7.7% (two of 26 patients). One of these was aged 59 and in Grade 4. This patient was in a terminal state at operation, and brain-stem hemorrhages were found at autopsy. The other patient, who was aged 54 years and in Grade 3, continued to deteriorate and died 3 days later; no autopsy was performed. The 12 patients treated before 1952 made good recoveries.

The long-term follow-up review of the 131 survivors showed that 20 (15%) had neurological disabilities of a permanent nature which were judged to have been due to the hematoma and not to coincidental disease (Tables 2 and 3). No patient survived into a chronic vegetative state. The type of disability, age, and preoperative grading are given in Table 3. Memory and personality defects were the most common residual deficits. Hemiparesis was an additional problem for those in Grade 4. Those aged 60 years and over and those in Grades 3 and 4 were more likely to have disabilities. There were two deaths during the first year (both in a mental hospital); one non-disabled survivor, an alcoholic, committed suicide 9 months after leaving the hospital.

Discussion

Comparisons between the reported series of chronic subdural hematoma are difficult to make as outcome is influenced by the proportion of patients who are drowsy or comatose, elderly, or alcoholic. Bender's grading of clinical state2,3 (Table 1) was a valuable contribution to this problem, in the same way that grading has been for subarachnoid hemorrhage and head injury. This scale merits adoption, and some other reports have made use of it.16,18,25,39,42 Valid comparisons should be possible when grading, age, and bilaterality are known. It is not thought that the retrospective grading of our patients has distorted unduly the overall results.

The mortality of chronic subdural hematoma treated by burr-holes, as reported in the earlier literature, ranged from 6% to 25%.22 The 212 cases reported by McKissock, et al.,26 in 1960 had a mortality rate of 6.1%, and 18.4% were stuporous or in coma; this report was a benchmark for the time, and has been improved upon only recently. Better diagnostic methods and improved management of ill patients have evolved since then. Table 4 presents a summary of the series reported since 1972, both before and after the introduction of computerized tomography (CT). From Bender's medically treated cases,2 it is clear that those in Grade 1 and, to a larger extent, in Grade 2 would be in a favorable

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**TABLE 2**

<table>
<thead>
<tr>
<th>Clinical Grade</th>
<th>No. of Cases</th>
<th>No. of Deaths</th>
<th>Serious Disability</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>59</td>
<td>0</td>
<td>5</td>
</tr>
<tr>
<td>2</td>
<td>39</td>
<td>0</td>
<td>8</td>
</tr>
<tr>
<td>3</td>
<td>15</td>
<td>1</td>
<td>4</td>
</tr>
<tr>
<td>4</td>
<td>20</td>
<td>1</td>
<td>3</td>
</tr>
<tr>
<td>total cases</td>
<td>133</td>
<td>2</td>
<td>20</td>
</tr>
</tbody>
</table>

* Mortality rate was 1.5% of all patients and 6.6% of Grade 3 and 4 patients; 15.0% suffered serious morbidity; and 85.0% enjoyed a full recovery. Clinical grade according to the Bender scale2,3 (see Table 1).

**TABLE 3**

| Major postoperative disabilities correlated with age and preoperative grading in 131 survivors* |
|-------------------------------------------------|-------------------------------------------------|-------------------------------------------------|
| Disability Variables                             | Variables                                       |                                                 |
| memory deficits                                  | age (yrs)                                      | 33 54 72 63 78                                  |
| grade                                           | 3 2 1 1 1                                      |                                                 |
| personality defects                              | age (yrs)                                      | 61 72 62 77 35                                  |
| grade                                           | 2 2 2 3 4                                      |                                                 |
| seizures                                        | age (yrs)                                      | 75 48 82                                       |
| grade                                           | 2 1 3                                          |                                                 |
| personality defects & hemiparesis               | age (yrs)                                      | 56 47 56 54                                     |
| grade                                           | 4 4 3 2                                        |                                                 |

* Late deaths included two in mental hospitals 6 months and 8 months after treatment and one alcoholic suicide 9 months postoperatively. For description of grading see Table 1.
class whatever was done. This has an important bearing on results. At present-day mortality rates, considerable numbers will be needed for the evaluation of any treatment. Our series of 133 patients had a mortality rate of 1.5%, but no deaths occurred among 107 patients with unilateral hematomas; these results compare well with any other large series before or after the introduction of CT (Table 4). 2,5,7,9,11,13,14,16,17,25,28,32,36,39,42,43 Retrospectively, only one of our two deaths could possibly have been prevented.

Over the last 25 years there has been a slow reduction in the mortality for the surgical treatment of chronic subdural hematoma. Much of this relates to the changing spectrum of presentation, with more patients being treated in a better clinical setting, and to the improved management of ill patients. A zero mortality rate in any large series is impossible as there will always be late cases or those with serious complicating illnesses, particularly in the elderly. The elderly do tend to do less well, 1,16,26,32,36 but advancing age is no bar to success. None of our patients aged 61 years and over died and our oldest patient was 84 years old. No elderly or comatose patient should be denied surgery, as the chances of recovery are great.

It is a basic assumption that the hematoma should be removed by simple means. A combination of twist-drill holes and closed drainage is at present favored. 16,23,25,39 This has received support from Tabaddor and Shulman 39 who compared several methods of treatment, but the comparisons are not valid as a new method of treatment was compared retrospectively with old treatments in different hands. Most series have been small (Table 4). Even if the eventual results were good the method has had its difficulties with delayed recovery and reaccumulation of the hematoma, and membranectomy has had to be resorted to. There is nothing to indicate that these complications have occurred any less frequently than with the traditional burr-hole method, in which one on each side may be enough. Our results were good without drainage which may increase the infection rate. 13 Sometimes a rapid removal of the hematoma has seemed to worsen the patient's condition, 16,26,36 but we had no such case. This might be an indication for urgent ventricular inflation.

The treatment advocated by LaLonde and Gardner 19 and used by us includes hematoma removal without drainage, active inflation of the brain at operation, and monitoring the CSF pressure intermittently so that a subnormal pressure could be corrected. The old view 6,27,31,39,41 that it may take some considerable time for the subdural space to close and for the brain to become symmetrical again has been confirmed by CT. 9,13,25,28,43 One detailed study has shown that the subdural space with closed drainage usually takes up to 21 days postoperatively to close, but that this could be delayed for as long as 40 days. 25 Furthermore, there was no close correlation between the persistence of the space and the patient's clinical state. Rapid obliteration was associated with speedy recovery and a thick subdural membrane tended to retard brain expansion.

Active brain expansion in the small series of Moussa and Joshy 28 using CT did not seem to increase the speed of closure of the subdural space. From what is now known of the prognosis 2 there would be no justification for its use in Grade 1 and 2 cases. Our impression that the clinical state of patients in Grades 3 and 4, with bilateral hematomas, and in the elderly, was improved with brain expansion confirms what others have reported. 5,12,34–36 This technique has not become generally used and it is said to be risky or of no value. 1,20,22,23,40 Apart from the problem of brain needle placement this is not borne out by our series. If ventricular puncture is difficult then lumbar puncture is an acceptable alternative. 5,12,34,36

The persistence of the subdural space postoperatively does carry a potential for reaccumulation of the hematoma. 9,13,25,28,43 which drainage may not overcome. 13,14,16,30 Our experience was that, if the hematoma was washed out completely and the subdural space not injured again, then actual recurrence was exceptional. Possibly, brain expansion may have had some tamponading effect. Such fluid as was present was space-filling rather than space-occupying. With CT visualization of the subdural space any decision to reoperate is essentially based on clinical deterioration. 9,13,25 The part that membranectomy plays in treating further bleeding is controversial 23,42 Two of our cases had a secondary bone flap but, in retrospect, it is doubtful if anything useful was achieved for the patient.

Postoperative collapse of the brain and intracranial

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**TABLE 4**

**Surgical treatment of chronic subdural hematoma in series reported since 1972**

<table>
<thead>
<tr>
<th>Authors, Year</th>
<th>No. of Cases</th>
<th>Treatment</th>
<th>Mortality Rate (%)</th>
</tr>
</thead>
<tbody>
<tr>
<td>without computerized tomography</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Raskind, et al., 1972</td>
<td>52</td>
<td>2 burr-holes</td>
<td>0</td>
</tr>
<tr>
<td>Bender, 1976</td>
<td>110</td>
<td>2 burr-holes</td>
<td>12.7</td>
</tr>
<tr>
<td>Conomy, et al., 1976</td>
<td>115</td>
<td>2 burr-holes</td>
<td>20.0</td>
</tr>
<tr>
<td>So, 1976</td>
<td>20</td>
<td>2 burr-holes</td>
<td>10.0</td>
</tr>
<tr>
<td>Tabaddor &amp; Shulman, 1977</td>
<td>21</td>
<td>twist drill &amp; drainage</td>
<td>9.5</td>
</tr>
<tr>
<td>Cameron, 1978</td>
<td>114</td>
<td>burr-holes</td>
<td>4.5</td>
</tr>
<tr>
<td>Kaste, et al., 1979</td>
<td>29*</td>
<td>burr-holes</td>
<td>10.3</td>
</tr>
<tr>
<td>Hubschmann, 1980</td>
<td>22</td>
<td>twist drill &amp; drainage</td>
<td>22.7</td>
</tr>
<tr>
<td>Tyson, et al., 1980</td>
<td>48</td>
<td>burr-holes</td>
<td>0</td>
</tr>
<tr>
<td>Harders, et al., 1981</td>
<td>50</td>
<td>burr-holes &amp; drainage</td>
<td>2.0</td>
</tr>
<tr>
<td>with computerized tomography</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Dolinskas, et al., 1979</td>
<td>37</td>
<td>burr-holes</td>
<td>10.8</td>
</tr>
<tr>
<td>Markwalder, et al., 1981</td>
<td>32</td>
<td>twist drill &amp; drainage</td>
<td>0</td>
</tr>
<tr>
<td>Victoratos &amp; Bligh, 1981</td>
<td>17</td>
<td>burr-holes</td>
<td>0</td>
</tr>
<tr>
<td>Galbraith, 1982</td>
<td>30</td>
<td>2 burr-holes</td>
<td>16.6</td>
</tr>
<tr>
<td>Harders, et al., 1982</td>
<td>100</td>
<td>burr-holes &amp; drainage</td>
<td>2.0</td>
</tr>
<tr>
<td>Moussa &amp; Joshy, 1982</td>
<td>24</td>
<td>burr-holes</td>
<td>0</td>
</tr>
</tbody>
</table>

* Bilateral cases only.
hypotension have often gone hand-in-hand, for in itself intracranial hypotension is associated with lowered states of consciousness, reversible when the pressure rises. Probably the intracranial pressure (ICP) must be below 50 mm H2O to cause symptoms, and commonly it is around zero. While spontaneous resolution can occur under suitable conditions, this may take several days if the patient is in an obtunded state. The syndrome of intracranial hypotension was featured in the older European literature but latterly has tended to be neglected. McKissock, et al., reported decreased ICP in seven of 212 patients with hematomas and recently it was noted in 10 of 100 surgically treated cases. Markwalder, et al., studied CSF pressures postoperatively and there were four patients with pressures from zero to 80 mm H2O; the brain was slow to expand in all of these cases. The patient with zero CSF pressure had a pressure of 70 mm H2O at 10 days. The CSF pressure of the patient who underwent meningeal was unfortunately not recorded. It was hoped that, with a high index of suspicion, early preventive treatment might be effective, but no conclusions can be reached from our data. The marked low ICP syndrome occurred in 10.7% of the 121 patients studied for this phenomenon, and was more prone to occur in cases with bilateral hematomas and in the elderly. These are the high-risk patients and our treatment seemed of value; there was no doubt about its immediate effectiveness, and the patients were less liable to intercurrent illnesses. Adverse effects or lack of value for such treatment have been forecast but never categorized; no ill effects were noted in our patients. All patients needed ongoing treatment for some days and it was never expected that intracranial dynamics would be restored quickly. All standard methods to prevent or combat intracranial hypotension were used. Now that it is common to monitor ICP, and with the use of CT, it should be possible to explore further the problems of slow brain expansion and intracranial hypotension in relation to the clinical state.

Computerized tomography has shown that additional lesions such as cerebral contusion, hemorrhage, infarction, swelling, or atrophy are not uncommon and may be responsible for poor clinical progress. Raised ICP and brain swelling have been considered adverse features. None of our patients had a CSF pressure above 300 mm H2O, and even at that level it was without clinical significance. This was the finding of others. Tyson, et al., found cerebral swelling in three of seven patients with chronic cerebral hematoma who were treated by a decompressive craniectomy and meningeal. It is far from clear what provoked this unusual train of events. None of our full cerebrospinal fluid studies resulted in brain swelling, rather the reverse and difficulties were experienced with so much dead space.

Spontaneous resolution of chronic subdural hematoma is well known and led Bender to manage 75 of 185 patients medically; he began to treat another 22 with that intent, but had to perform surgery. Under this regimen, hospitalization was needed for 3 weeks although ambulation was allowed. Another series treated medically had an average stay in the hospital of 41 days, and one case of bilateral hematoma took 42 days to resolve. By the end of 3 weeks, 77% of our patients had been discharged home cured after operation. There is a report of 51 patients of whom 14 were managed medically with the aid of CT. It may be expected that CT will permit more cases to be treated nonsurgically, but this can only be risk-free outside the hospital if the patient is adequately supervised by relatives or friends for a week or two. Unexpected deterioration is a notorious feature of the condition so that surgeons have preferred to operate in marginal cases.

Few reports give the duration of hospital stay, and clearly the patients of Markwalder, et al., were in the hospital 10 days postoperatively. One CT-scanned series of 24 patients had an average hospital stay of 7 days (range 3 to 13 days), and another of 20 patients had an average stay of 10 days. Computerized tomography should help shorten hospitalization.

Up to 90% of survivors may be expected to do well. Some 16% of McKissock's large series of 389 patients were wholly or partly disabled and there are reports of 7.3% and 5.2% of patients having disabilities. Fifteen percent of our survivors were permanently disabled (Table 2). This was particularly a problem with the aged and for those who were in Grades 3 and 4 preoperatively. None of our patients were in a chronic vegetative state. There is a long-term toll in quality of survival that cannot be ignored and which continues after leaving the hospital.

The single most important factor in the outcome of chronic subdural hematoma treatment is the extent of the preoperative neurological dysfunction, which in turn depends upon delay in diagnosis. Diagnosis has been improved materially by CT which has also helped in the identification of some postoperative problems. But, how far this has been reflected in results is less clear, if our pre-CT results are a valid group for comparison. Most patients do recover but there are some who have difficulties because of the reluctance of the compressed brain to return to its normal form and function. The role that intracranial hypotension plays in this is in need of reappraisal. Any future improvements in treatment will be best shown in Grade 3 and 4 patients, and in those with bilateral hematomas; it was here that we believe that our methods were of greatest value.

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References


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Address reprint requests to: Richard G. Robinson, Ch.M., F.R.C.S., 32 Burwood Avenue, Dunedin, New Zealand.