Muscle Strength in Type 2 Diabetes
Henning Andersen, Søren Nielsen, Carl E. Mogensen, and Johannes Jakobsen

Motor function in type 2 diabetes is largely unknown. In 36 type 2 diabetic patients and in 36 control subjects matched for sex, age, weight, height, and physical activity, strength of flexors and extensors at elbow, wrist, knee, and ankle was assessed at isokinetic dynamometry. The degree of neuropathy was determined by clinical scores, nerve conduction studies, and quantitative sensory testing. Eventually, all results were summed to obtain a neuropathy rank-sum score (NRSS). The degree of nephropathy and retinal condition were also evaluated. Diabetic patients had a 17 and 14% reduction of strength of ankle flexors ($P < 0.02$) and ankle extensors ($P < 0.03$), respectively. At the knee, strength of extensors and flexors was reduced by 7% (NS) and 14% ($P < 0.05$), respectively. At the elbow and wrist, muscle strength was preserved. The NRSS was related to the strength at the ankle ($r = -0.45$, $P < 0.01$) and knee ($r = -0.42$, $P < 0.02$). Following multiple regression analysis, the NRSS but not the degree of nephropathy or retinopathy was related to strength at the ankle and knee. In conclusion, type 2 diabetic patients may have muscle weakness at the ankle and knee related to presence and severity of peripheral neuropathy. Diabetes 53:1543–1548, 2004

Sensory symptoms and deficits are frequent in distal diabetic polyneuropathy. Motor symptoms are less dramatic, and motor deficits are more difficult to recognize. Not surprisingly, the literature on this manifestation of diabetic polyneuropathy is sparse. In patients with long-term type 1 diabetes, we have found impaired muscle strength at the ankle and knee closely related to the severity of neuropathy (2). Motor dysfunction is known to occur in type 2 diabetic patients; however, the severity and distribution of the weakness has not been reported (3).

In a population-based study from the U.K., a similar frequency of neuropathy was found in type 1 and 2 diabetic patients after age correction (4). Nevertheless, a considerably lower frequency of severe neuropathy, defined as an inability to walk on heels, was observed in type 2 diabetes in a population-based study from Minnesota (3). This observation may indicate less motor dysfunction in type 2 diabetes. However, the relation between inability to walk on heels and muscle strength has not been established, so there is a clear need for quantitative studies of motor function in type 2 diabetes.

In the present study, we evaluated muscular performance of lower and upper extremities quantitatively in type 2 diabetic patients, applying isokinetic dynamometry, which has high reliability in the determination of maximal strength in both neuropathic and healthy subjects (5). To study the relationship of muscle strength with the prevalence and severity of diabetic neuropathy, other diabetes complications, and metabolic control, patients were characterized clinically, biochemically, and with electrophysiological and sensory function tests.

RESEARCH DESIGN AND METHODS
All patients and control subjects gave informed consent for participation in the study, which was approved by the local ethic committee. Thirty-six type 2 diabetic patients (13 women and 23 men) aged <70 years with a diabetes duration >5 years participated in the study. Patients with severe cardiac or lung disease, acute or chronic musculo-skeletal disorders, acute metabolic dysregulation, other neurological or endocrine disorders, severe symptomatic macroangiopathy, or any previous or present lower-limb asymmetric proximal weakness were excluded, and case records were searched for other explanations of polyneuropathy. All patients were examined by a trained neurologist, who decided whether the findings were typical of diabetic neuropathy. Daily insulin injections were taken by 18 patients, among whom 7 had one injection a day, 8 injections twice a day, and 3 multiple injections. Fifteen patients were treated with oral agents, and 3 were on diet only. Seventeen patients received antihypertensive medication. Seven patients had symptoms of or a history indicating macroangiopathy. Four patients had mild to moderate intermittent claudication, but all could walk at least 500 m before symptoms occurred. Two of these patients had a history of myocardial infarction >1 year before the study, and one of these two patients also had mild stable cardiac angina. In addition, three other patients had mild stable cardiac angina, one of whom had a myocardial infarction 15 years before this study. Twelve patients were regular smokers. For motor control, 36 healthy age-, sex-, height-, and weight-matched subjects were recruited by advertising in the local press and among hospital employees. All subjects were interviewed about the intensity of their physical activity at work, during spare time, and during daily transportation to and from work with the classification by Saltin and Grimby (6) in a modified form, and care was taken to match control subjects and diabetic patients with respect to physical activity. To correct for the possible influence of intermittent claudication on motor function, four of the control subjects included had this symptom to a similar degree.

Isokinetic muscle strength. Maximal isokinetic muscle strength (peak torque) of extension and flexion at the ankle, knee, elbow, and wrist was evaluated with an isokinetic dynamometer (Lido Active Multijoint II; Loredan Biomedical, Sacramento, CA). The nondominant leg and the dominant arm were tested. Hand dominance was determined by the preferred hand for hammering and leg dominance by the preferred leg to kick a ball. The tests were performed following standardized procedures as described elsewhere (2.5). In short, after a presession, the subjects were instructed to push and pull as hard and fast as possible through the full available range of motion. The velocity was 60°/s at the ankle and elbow and 80°/s at the wrist and knee. The range of motion was 45, 70, 55, and 110° at the ankle, knee, wrist, and elbow, respectively. Every test included eight reciprocal trials with a 10-s rest period after each trial. To exclude submaximal performance, data were only ac—

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Received for publication 7 January 2004 and accepted in revised form 15 March 2004.

CMAP, compound muscle action potential; MNCV, motor nerve conduction velocity; NDS, neurological disability score; NRSS, neuropathy rank-sum score; NSS, neuropathy symptom score; SNAP, sensory nerve action potential; SNCV, sensory nerve conduction velocity; VPT, vibration perception threshold.

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nerve. Sensory nerve conduction velocity (SNCV) and amplitude of the SNAP, normal values previously determined in age-matched control subjects are given in Table 1. The diabetic patients had a blood glucose of 8.7 mmol/l (2.2–25.6) (median, range), HbA1c 8.8% (6.7–14.4), C-peptide 146 pmol/l (23–1,270), serum insulin 34 pmol/l (17–920), and overnight urinary albumin excretion 20.1 μg/min (5.9–2,400.0). Biochemical parameters in relation to neuropathy are given in Table 2. The median NSS was 0. Eleven patients had symptomatic neuropathy, all of whom complained of sensory disturbances, whereas only 3 patients complained of muscle weakness. Three patients complained of paresthesias of the feet but did not fulfill the minimal criteria for diabetic neuropathy and, therefore, were classified as nonneuropathic subjects. The NDS for all patients was 9 (range 0–44). Scores for muscle weakness, sensory impairment, and impaired tendon reflexes were obtained in

**TABLE 1**

Clinical data of type 2 diabetic patients and control subjects

<table>
<thead>
<tr>
<th></th>
<th>n</th>
<th>Sex (M/F)</th>
<th>Age (years)</th>
<th>Weight (kg)</th>
<th>Height (cm)</th>
<th>Duration of diabetes (years)</th>
<th>Retinopathy (none, simplex, proliferative)</th>
<th>Nephropathy (none, incipient, overt)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Diabetic patients</td>
<td>36</td>
<td>23/13</td>
<td>59 (44–69)</td>
<td>85 (49–110)</td>
<td>174 (156–190)</td>
<td>11 (5–26)</td>
<td>(15, 19, 2)</td>
<td>(16, 15, 5)</td>
</tr>
<tr>
<td>Control subjects</td>
<td>36</td>
<td>23/13</td>
<td>58 (47–74)</td>
<td>83 (61–101)</td>
<td>174 (160–194)</td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

Data are median (range). Rejected if the coefficient of variation for torque values did not exceed 10%. In experiments where the coefficient of variation was >10%, the subject was retested once. If the coefficient of variation still exceeded 10% at the second test, data were excluded if no outlier torque curve could be identified.

**Electrophysiological studies.** Nerve conduction studies were performed using an electromyograph (Dantec Counterpoint, Skovlunde, Denmark) with standard methods as described elsewhere (7). Motor nerve conduction velocity (MNCV) and amplitude of the compound muscle action potential (CMAP) were measured in the dominant median nerve and the nondominant peroneal nerve. Sensory nerve conduction velocity (SNCV) and amplitude of the sensory nerve action potential (SNAP) were measured in the nondominant sural nerve (distance between stimulation and recording point: 140 mm) and the dominant median nerve, with skin temperatures in the range of 31–34°C. Z scores were calculated for MNCV and amplitude of the CMAP from values of healthy volunteers obtained with similar techniques (7). For SNCV and SNAP, normal values previously determined in age-matched control subjects were adopted.

**Clinical evaluation and quantitative sensory testing.** All patients were evaluated according to a neuropathy symptom score (NSS) (8) and a neurological disability score (NDS) (9). The NSS indicates the number of motor, sensory, and autonomic complaints. The NDS is a combined score obtained from the neurologic examination of muscle strength, tendon reflexes, and sensation at the great toe and index finger. The retinal status of the patients was classified as “no”, nonproliferative, or proliferative retinopathy by a trained ophthalmologist.

Vibratory perception thresholds were evaluated at the dominant index finger pulp and nondominant dorsum of the great toe using forced-choice techniques (CASE IV; WR Medical Electronics, Stillwater, MN). Before the examination, patients were given written instructions and a demonstration of the technique. The thresholds were determined with the 4, 2, and 1 stepping algorithm (10). The perception thresholds for each patient were compared with results from a large group of healthy control subjects, and the corresponding percentiles were determined.

**Biochemical measurements.** Fasting blood samples were taken for the determination of blood glucose (normal range 3.5–5.5 mmol/l), HbA1c (4.4–6.0%), serum creatinine (55–110 μmol/l), serum C-peptide (174–960 pmol/l), and serum insulin (9–94 pmol/l) using standard laboratory methods. Urinary albumin excretion was measured by radioimmunoassay and assessed as the mean of three timed overnight collections (11). Overnight urinary albumin excretion was determined and categorized as normoalbuminuria (<30 μg/min), incipient nephropathy (between 20 and 200 μg/min), and overt nephropathy (>200 μg/min).

**Definitions and calculations.** The minimal criteria for diabetic neuropathy were adopted (4,12). Patients were defined as neuropathic if two or more of the following four categories were abnormal, one being an abnormality of nerve conduction or vibration perception threshold (VPT); NSS ≥1, NDS ≥2, abnormal MNCV or amplitude of the CMAP in at least two of four nerves, and abnormal VPT at the index finger and great toe (≥98th percentile). Patients fulfilling the minimal criteria were separated into patients with and without symptoms of neuropathy. Furthermore, for quantification of severity of neuropathy, an NSS was calculated for each patient. The NSS was a summation of the rank scores of the NSS, the NDS, the VPT, and the average of the rank scores of the MNCV, CMAP, SNCV, and SNAPs. To avoid bias of correlations by comparison of clinical and quantitative measures of motor dysfunction, scores due to symptoms or signs of muscular weakness were excluded from the NSS and NDS.

For each patient, a predicted strength value for the extensors and flexors at the ankle, knee, and wrist was calculated by multiple regression analysis, including results from a large group of healthy control subjects (>130 subjects), taking into account age, height, weight, and sex (2). Subsequently, the muscle strength of each patient was expressed in percentage of the predicted strength. Eventually, the muscle strength at the three joints was calculated as the average of the relative strength of the extensors and flexors.

**Statistical analyses.** The comparisons between strength of the diabetic patients and control subjects were performed with an unpaired t test. Comparisons of muscle strength and electrophysiological findings among nonneuropathic, asymptomatic, and symptomatic neuropathic patients were performed using ANOVA, including pairwise comparisons with protection for multiple comparisons. The biochemical findings in the three groups were compared using Kruskal-Wallis one-way ANOVA. To estimate the correlations between muscle strength and NSS and the various biochemical findings, linear regression analysis was applied. The influence of neuropathy, retinopathy, and nephropathy on muscle strength was evaluated with linear regression analysis and stepwise multiple regression analysis. No, nonproliferative, and proliferative retinopathy were scored 1, 2, and 3, respectively. Normoalbuminuria, incipient, and overt nephropathy were scored 1, 2, and 3, respectively. For all statistical analyses, a 5% limit of significance was applied.

**RESULTS**

Clinical evaluation, biochemical measurements, and quantitative sensory evaluation. Clinical data for the diabetic patients and their matched control subjects are given in Table 1. The diabetic patients had a blood glucose of 8.7 mmol/l (2.2–25.6) (median, range), HbA1c 8.8% (6.7–14.4), C-peptide 146 pmol/l (23–1,270), serum insulin 34 pmol/l (17–920), and overnight urinary albumin excretion 20.1 μg/min (5.9–2,400.0). Biochemical parameters in relation to neuropathy are given in Table 2.

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**TABLE 2**

Laboratory findings in type 2 diabetic patients in relation to neuropathy

<table>
<thead>
<tr>
<th></th>
<th>n</th>
<th>Serum creatinine (μmol/l)</th>
<th>Blood glucose (mmol/l)</th>
<th>HbA1c (%)</th>
<th>Serum insulin (pmol/l)</th>
<th>C-peptide (pmol/l)</th>
<th>Albumin excretion rate (μg/min)</th>
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</thead>
<tbody>
<tr>
<td>No neuropathy</td>
<td>14</td>
<td>81 (51–114)</td>
<td>9.8 (5.9–14.5)</td>
<td>8.9 (6.7–11.3)</td>
<td>66 (21–308)</td>
<td>127 (35–1,270)</td>
<td>20 (6–1,562)</td>
</tr>
<tr>
<td>Asymptomatic neuropathy</td>
<td>11</td>
<td>79 (59–104)</td>
<td>8.3 (6.0–20.4)</td>
<td>8.5 (7.0–10.6)</td>
<td>40 (17–920)</td>
<td>88 (35–745)</td>
<td>15 (8–41)</td>
</tr>
<tr>
<td>Symptomatic neuropathy</td>
<td>11</td>
<td>80 (51–153)</td>
<td>9.2 (2.2–25.6)</td>
<td>9.6 (6.9–14.4)</td>
<td>56 (28–197)</td>
<td>150 (29–784)</td>
<td>63 (8–2,400)</td>
</tr>
</tbody>
</table>

Data are median (range). *P < 0.05.*
6, 23, and 25 patients, respectively. Twelve patients had abnormal VPT at both the index finger and great toe.

**Isokinetic muscle strength.** Maximal isokinetic muscle strength in all patients was reduced by 14% for the ankle extensors \((P < 0.03)\) and by 17% for the ankle flexors \((P < 0.02)\) (Fig. 1). Knee flexor strength was reduced by 14% \((P < 0.05)\), whereas a statistically insignificant reduction of 7% was found for the knee extensors \((P = 0.26)\). No reduction in muscle strength was found at the wrist or elbow (Fig. 1).

At the ankle, muscle strength was \(79 \pm 19.6\%\) for men and \(80 \pm 18.2\%\) for women (NS). Muscle strength at the knee was \(81 \pm 17.7\%\) and \(86 \pm 18.1\%\) in men and women, respectively (NS).

Comparing muscle strength in nonneuropathic, asymptomatic neuropathic, and symptomatic neuropathic patients, there was a statistically significant difference at the ankle and knee (Fig. 2). In pairwise comparisons, asymptomatic neuropathic patients had decreased muscle strength at the knee compared with nonneuropathic patients (Fig. 2). Symptomatic neuropathic patients had decreased strength at the ankle and knee compared with nonneuropathic patients (Fig. 2).

A relationship could be established between the NRSS and the strength at the ankle and knee (Fig. 3). The correlation coefficients for the NRSS and the strength of ankle extension and ankle flexion were \(-0.47 (P < 0.005)\) and \(-0.35 (P < 0.05)\), respectively. For extensors and flexors at the knee and wrist, the coefficients were \(-0.44 (P < 0.02), -0.38 (P < 0.05), -0.05 (NS), and -0.19 (NS), respectively. No statistically significant difference in muscle strength at the ankle, knee, or wrist were found between patients with normoalbuminuria and patients with nephropathy (incipient and overt). Furthermore, there were no relations between degree of nephropathy and the

**FIG. 1.** Isokinetic muscle strength of extensors and flexors at the ankle, knee, wrist, and elbow in type 2 diabetic patients (■) and control subjects (□). Values are mean ± SD. *\(P < 0.03\); †\(P < 0.05\).

**FIG. 2.** The averaged muscle strength of the extensors and the flexors given as the percentage of the expected value in type 2 diabetic patients in relation to severity of neuropathy. Muscle strength at the ankle \((F = 4.6, P < 0.02)\) and at the knee \((F = 9.1, P < 0.002)\) in the three groups is different. In addition, symptomatic (□) and asymptomatic neuropathic (■) patients are compared with nonneuropathic (■) patients. Values are mean ± SD. *\(P < 0.02\); †\(P < 0.005\).

**FIG. 3.** Averaged isokinetic muscle strength of the extensors and flexors at ankle \((r = -0.45, P < 0.005)\) and knee \((r = -0.42, P < 0.02)\) in relation to the NRSS in type 2 diabetic patients.
maximal muscle strength at the ankle ($r = -0.15$, NS) or knee ($r = -0.18$, NS), but the degree of retinopathy was related to the combined strength at the ankle ($r = -0.36$, $P < 0.05$) and knee ($r = -0.40$, $P < 0.02$). However, including the NRSS and the scores for retinopathy and nephropathy in a multiple regression analysis, a statistically significant relation was found only between the NRSS and the muscle strength at the ankle and knee. Eventually, no relationship could be established between the strength at the knee and ankle and the biochemical variables blood glucose, HbA1c, serum creatinine, serum C-peptide, and serum insulin.

**Electrophysiological testing.** For all patients, the MNCV and amplitude of the CMAP of the median nerve were 49.3 m/s (range 33.1–63.6) and 5.2 mV (1.8–10.8), respectively. For the peroneal nerve, the corresponding values were 39.7 m/s (23.7–49.1) and 2.9 mV (0.5–8.7). The SNCV of the median sensory nerve was 46.6 m/s (27.2–62.2), and the SNAP was 6.9 μV (1.0–17.3). For the sural nerve, SNCV and SNAP were 42.2 m/s (28.3–51.6) and 3.3 μV (0.9–8.1), respectively.

**DISCUSSION**

The present study found that type 2 diabetic patients have muscle weakness at the ankle and knee, whereas the strength at the elbow and wrist is preserved. As to the underlying etiology, in this study muscle strength at the ankle and knee was related to the severity of neuropathy rather than to the degree of nephropathy, the degree of retinopathy, or the metabolic variables.

Diabetic polyneuropathy is frequently encountered in type 2 diabetic patients. The prevalence of diabetic polyneuropathy increases from 8% in newly diagnosed patients to >40% after 10 years of diabetes (13). Based on clinical experiences, diabetic polyneuropathy is usually categorized as a sensory neuropathy associated with an increased risk of foot ulcers and amputation (14). In contrast to sensory abnormalities, motor dysfunction is considered to seldom occur. However, motor function has not previously been studied with appropriate quantitative techniques. In previous clinical studies of diabetic polyneuropathy, motor function has been evaluated by manual muscle testing (12,15), applying the Medical Research Council Scale (16) or the NDS (9). All existing rating systems for manual muscle testing use ordinal scales with low sensitivity, especially in the range of slight to moderate muscle weakness, as is usually seen in diabetic patients (17). The presence of muscle weakness suggests more severe diabetic polyneuropathy. Previously, muscle performance has been evaluated with easily performed function tests such as the ability to walk on heels (4,18). The ability to stand on heels is used because isolated inability to stand on toes indicates intraspinous rather than peripheral nerve pathology (19). According to the classification of diabetic polyneuropathy developed by Dyck et al. (18), inability to walk on heels is the determinant for the presence of severe symptomatic diabetic polyneuropathy. Functional tests are potentially of great practical value, as they are performed quickly without any technical equipment. However, if not validated, it will remain unclear what the results represent. Additional study is required to determine the degree of muscle weakness at which inability to walk on heels occurs and how age, weight, and sex are involved. Furthermore, in addition to muscle weakness, loss of proprioception, decreased joint mobility and impaired vision may all contribute to this functional shortcoming.

Some hospital-based studies of diabetic patients report a higher frequency of neuropathy and more abnormal test results in type 2 than in type 1 diabetic patients (20–22). In a population-based study from the U.K., the frequency of neuropathy was higher in type 2 diabetes (4), but following correction for age, the difference between type 1 and 2 diabetic patients disappeared. In comparison, a population-based study from Rochester, Minnesota, reported that severe neuropathy was six times more frequent in type 1 diabetes (3). The findings in the present study do not necessarily reflect the presence of neuropathic complications, including muscle weakness in the general type 2 diabetic population, because a higher frequency of complications can be expected in patients attending a hospital outpatient clinic. In addition, the quality of metabolic control was considerably lower in this study (median HbA1c 8.8%) compared with an unselected group of type 2 diabetic patients attending a primary health care center in Sweden, who reported a mean HbA1c of 6.3% (23). Furthermore, willingness to participate in this study might be higher in symptomatic neuropathic patients, thereby introducing bias if the prevalence and severity of neuropathic complications in type 2 diabetes are sought.

In type 1 diabetic patients with a diabetes duration of ∼30 years, we observed weakness at the ankle and knee (2). Ankle flexor and extensor strength was reduced by 21%. At the knee, a weakness of 17 and 16% for the extensors and flexors, respectively, was found. A comparison with the present study is hampered by differences in diabetes duration and age. However, since the diabetes duration was only 11 years in the present study, the degree of muscle weakness in type 1 and 2 diabetic patients seems similar. It is noteworthy that a distal-proximal gradient of muscle weakness was found for both types of patients. Also, in accordance with the present findings, correlations could be established between strength at the ankle and knee and the severity of neuropathy in type 1 diabetic patients. In a previous study using a quantitative electromyographic technique (macroEMG), we evaluated a mixed group of type 1 and type 2 diabetic patients and found that there were signs of compensatory reinnervation in distal muscles (24). This further supports the notion that the motor impairment observed is due to neurogenic weakness.

In a group of aged diabetic patients, including primarily type 2 diabetic patients, Lord et al. (25) found impaired muscle strength of knee extension in women but not men. The strength at the ankle was not measured, no correction for weight was made, and the relation to presence and severity of neuropathy was not studied. In two studies evaluating gait and posture in diabetic patients with and without neuropathy, quantitative assessment of muscle strength at the ankle in diabetic patients was used, but the type of diabetes was not stated and relations to severity of neuropathy were not sought (26,27). To our knowledge, this is the first study applying quantitative assessment of motor performance in type 2 diabetes to severity of neuropathy and other diabetes complications. Following
moderate strength training, type 2 diabetic patients can improve their muscle strength (28); however, whether this applies to neurogenic-weakened muscles is unknown and should be addressed in future studies.

Four of our patients had slight to moderate intermittent claudication reflecting peripheral vascular disease, which may affect muscular performance (29–31). The impaired oxygen supply could affect striated muscle directly or indirectly via the peripheral nerves (29). Concerning the relation between motor function and polyneuropathy, it may be questioned whether patients with macroangiopathy should be included. However, already at the time of diagnosis, reduced blood flow can be found with ultrasound Doppler in one-third of type 2 diabetic patients, even after exclusion of patients with advanced atherosclerotic diseases (32). Since many newly diagnosed patients do not have intermittent claudication, it is difficult to identify patients with reduced blood supply simply by the presence of symptoms. However, to exclude any major influence of peripheral vascular disease in the diabetic patients on motor performance, we included the same number of subjects with intermittent claudication in the control group. Future studies combining assessment of blood flow and peripheral nerve function could provide further information about the effect of reduction in blood flow on motor function.

In conclusion, type 2 diabetic patients may have weakness of the extensors and flexors at the ankle and of knee flexors with preservation of strength of the knee extensors and of muscles at the wrist and elbow. The distribution of muscular weakness indicates that a distal neuropathic process underlies the impaired motor performance. This assumption is supported by the present observation that muscular strength at the ankle and knee is related to severity of neuropathy and not to degree of nephropathy, retinopathy, or the metabolic abnormalities associated with diabetes.

ACKNOWLEDGMENTS

The Karen Elise Jensen Foundation, the Novo Nordisk Foundation, the Research Initiative of Aarhus University Hospital, and the Danish Diabetes Association are acknowledged for financial support.

M. Møller, E. Hornemann, B. Holmboe, P. Kousgaard, T. Thillermann, and J. Tjoernholm are acknowledged for their excellent technical assistance.

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