Decreased driving ability in people with Parkinson's disease

V-M Heikkilä, J Turkka, J Korpelainen, T Kallanranta, H Summala

Abstract

Background—Driving is a complex form of activity involving especially cognitive and psychomotor functions. These functions may be impaired by Parkinson's disease. The relation between Parkinson's disease and driving ability is still obscure and clinicians have to make decisions concerning the driving ability of their patients based on insufficient information. Until now no studies have compared different methods for evaluating the driving ability of patients with Parkinson's disease.

Methods—The driving ability of 20 patients with idiopathic Parkinson's disease and 20 age and sex matched healthy control subjects was evaluated by a neurologist, psychologist, vocational rehabilitation counsellor, and driving instructor using a standard 10 point scale. The patients and controls also evaluated their own driving ability. Cognitive and psychomotor laboratory tests and a structured on road driving test were used for evaluating the subjects' driving ability.

Results—The patients with Parkinson's disease performed worse than the controls both in the laboratory tests and in the driving test. There was a high correlation between the laboratory tests and driving test both in the patient group and in the control group. Disease indices were not associated with the driving test. The neurologist overestimated the ability of patients with Parkinson's disease to drive compared with the driving ability evaluated by the structured on road driving test and with the driving related laboratory tests. Patients themselves were not capable of evaluating their own ability reliably. **Conclusion**—Driving ability is greatly decreased in patients with even mild to moderate Parkinson's disease. The evaluation of patients' driving ability is very difficult to carry out without psychological and psychomotor tests and/or a driving test.

tasks which in particular stress the visual and visuospatial cognitive and psychomotor functions, as well as attentive resources.¹⁻⁴

Parkinson's disease causes progressive motor dysfunction and possible impairment of cognitive and psychomotor functions.⁵⁻⁶ All these functions are essential to driving a car. The short term working memory and non-verbal recognitive memory are also impaired in Parkinson's disease⁷⁻⁹; this may be connected to dopaminergic stimulation.¹⁰ Although the existence of generalised visuospatial deficits caused by Parkinson's disease is disputed,¹¹⁻¹³ there is considerable evidence suggesting that the disease impairs visuoperceptual functions.¹⁴ Parkinsonian patients have problems in maintaining their attention,¹⁵ especially when a task is relatively complex.¹⁶⁻¹⁷ As regards the reaction times of patients with Parkinson's disease there are contradictory results: some investigators have found significantly slower reaction times in patients with Parkinson's disease than in healthy controls¹¹⁻¹⁸⁻¹⁹ whereas others have found no differences,²⁰ or if differences existed, they only appeared in complex situations.²¹ In addition the information processing abilities of patients with Parkinson's disease are weak,²²⁻²⁵ and the more complex the task undertaken, the more frequent the difficulties manifested.²⁶

Although it is well known that cognitive and psychomotor functions are impaired by Parkinson's disease, there are, still, only a few studies on the relation between Parkinson's disease and the ability to drive a car.⁶⁻²⁷⁻²⁹ McLay²⁹ studied the driving ability and the history of a small, heterogeneous Parkinson's disease group and found that some of their driving accidents were probably caused by the disease. Dubinsky *et al*²⁷ reported that accidents are more common among patients with more severe Parkinson's disease (Hoehn and Yahr III) than among patients with less severe disease (Hoehn and Yahr I) or healthy controls. However, their conclusion was that severity scales (The Northwestern University disability scale, the Schwab and England activities of daily living scale, and the Hoehn and Yahr scale) did not reliably predict inability to drive. Madeley *et al*⁶ discovered in their laboratory study a correlation between the severity of Parkinson's disease measured by Webster's rating scale and simulated driving reaction time and accuracy. Lings *et al*²⁸ showed in their simulator study that inpatients with Parkinson's disease, reactions are considerably slower, and they commit a greater number of errors than healthy people. However, to our knowledge there are no systematic studies considering the compatibility of different methods

Merikoski Research and Rehabilitation Centre, Finland
V-M Heikkilä

Department of Neurology, University of Oulu, Finland
J Turkka

Deaconess Institute of Oulu, Finland
J Korpelainen

Rehabilitation Research Unit, University of Oulu, Finland
T Kallanranta

Traffic Research Unit, Department of Psychology, University of Helsinki, Finland
H Summala

Correspondence to:
Dr Veli-Matti Heikkilä,
Merikoski Research and Rehabilitation Centre,
Nahkatehtaankatu 3, 90100
Oulu, Finland. Telephone
00358 8 8848690; fax 00358
8 8848650; email:
veli.heikkila@tiimi.merikoski.fi

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Driving a car is a highly complicated form of activity carried out in a constantly changing environment. It consists of perception, information processing, and decision making and requires the drivers to carry out simultaneous

Table 1 Clinical characteristics of patients with Parkinson's disease

	Mean (SD)	Range
Age (y)	59 (11)	35–73
Duration of PD (y)	5.6 (2.8)	2–12
Dose of levodopa (mg/day)	450 (188)	200–800
Hoehn and Yahr stage	1.9 (0.6)	1–3
MMSE score	28.6 (1.5)	25–30

MMSE = mini mental state examination.

for estimating the ability of patients with Parkinson's disease to drive.

To evaluate the driving ability of patients with Parkinson's disease, we performed a study using clinical evaluation, cognitive and psychomotor laboratory tests, and a standardised on road driving test.

Subjects and methods

SUBJECTS

Twenty consecutive patients with idiopathic Parkinson's disease (mean age 59 (SD 11) years) were recruited from the outpatient clinic of the Department of Neurology, University Hospital of Oulu, Finland. The criteria for inclusion were male sex, mild to moderate Parkinson's disease (Hoehn and Yahr stages 1–3), general good health, and regular car driving. Patients with other diseases as well as patients using any drugs (except those for the treatment of Parkinson's disease) known to affect driving ability were excluded. Similarly, patients with levodopa induced dyskinesiae and on-off phenomena were excluded. Twenty healthy age matched men (mean age 55 (SD 6) years) with similar driving habits served as controls. Tables 1 and 2 show the clinical and driving characteristics of the patients and controls. All the patients were being treated with selegiline (daily dose 10 mg) and levodopa (mean daily dose 450 (SD 188) mg). Additionally, five patients were taking bromocriptine, one pergolide, and one entacapone.

Apart from three traffic accidents that had occurred in the Parkinson's disease group during the past two years compared with none in the control group, there were no differences in the driving histories of the members of the two study groups.

This study was approved by the ethics committee of the Merikoski Rehabilitation and Research Centre, Oulu, Finland. Patient evaluations and all the laboratory and driving tests were performed after obtaining informed consent.

METHODS

Clinical evaluations of all the patients were carried out on an outpatient basis by the same neurologist (JT). The clinical evaluation com-

prised a neurological examination and the mini mental state examination (MMSE). To rule out any on-off effects, neuropsychological assessments and on road driving tests were performed when the drivers with Parkinson's disease considered that they were at their optimal level of performance.

Driving ability was assessed on a 10 point scale where 10 indicates an excellent ability, and 4 and below indicate that the subject is unable to drive safely. The ability of patients with Parkinson's disease to drive was estimated by a neurologist, by a psychologist using tests and an interview, by a driving instructor on the basis of a driving test, and by the patients themselves using the same 10 point scale. All the evaluations were done without any interaction between the specialists. The evaluations of the controls were carried out in the same way as the patients' evaluations, except that the neurologist's place was taken by a vocational rehabilitation counsellor.

Both the patients and controls underwent the computer aided laboratory tests³⁰ designed by the Austrian Road Safety Board³¹ and partially validated against driving measures.³² This test package comprises the following sections:

Visual short term memory—The material consists of 24 geometric figures. The subject has to memorise three consecutively presented figures at a time. The total time used for the test and the correct responses are registered.

Perceptual flexibility and decision making—Two hundred visual exercises are presented to the examinee. Processing time is unrestricted. The total time used for the test and the number of correct responses is taken into account.

Vigilance—Continuous vigilance is studied in low stimulus monitoring situations. The total number of test stimuli are 1000, of which 100 are critical. The stimuli appear at irregular intervals without advance warning. The number of correct and incorrect responses, as well as the mean value of reaction times are calculated.

Complex choice reaction time—This is used to measure the time required for a person to react to complex visual signals. The correct reactions and total number of errors are measured, together with the reaction time, which is subdivided into cognitive and motor components.

Information processing capacity and reactive stress tolerance test³³—This test is designed to assess a person's reactive behaviour under changing conditions. Three test phases each consist of 180 signals with different signal presentation rates: (a) an "easy" phase—36 signals/min, (b) the "stress" phase—63 signals/min, and (c) the "recovery" phase—56 signals/min. Correct, correct and within the time, delayed, omitted, incorrect, and multiple responses are registered in all three phases.

The on road test was performed both in urban and rural surroundings on a standard and relatively difficult route. The time of the day and weather conditions did not vary noticeably. The time needed to cover the route was about 45 minutes. The route was designed to be sufficiently difficult. The evaluation was

Table 2 Driving characteristics of patients with Parkinson's disease (PD) and control subjects

	PD (n=20) Mean (SD)	Controls (n=20) Mean (SD)	p Value
Driving licence (y)	35 (9)	32 (6)	0.2511
Km travelled in past year*	2.6 (0.7)	2.9 (1.1)	0.2719
Total driving (1000 km)	984 (829)	1453 (1091)	0.3727

*1 ≤5000 km/y; 2 = 5000–10 000 km/y; 3 = 10 000–30 000 km/y; 4 = 30 000–100 000 km/y; 5 ≥100 000 km/y.

Table 3 Evaluation of driving ability carried out with a 10 point scale by the patients with Parkinson's disease (PD), control subjects, the neurologist, a psychologist, a vocational rehabilitation counsellor (control group), and the driving instructor; number 10 indicates an excellent ability to drive a car and the numbers 4 and below indicate inability to drive a car

Examinee	Parkinson's disease				Controls			
	Patients	Neurologist	Psychologist	Driving instructor	Controls	Vocational counsellor	Psychologist	Driving instructor
1	7	8	5	3	8	7	7	7
2	8	8	7	6	6	7	8	7
3	7	7	6	4	6	6	5	7
4	7	6	4	2	6	6	8	8
5	8	7	6	5	9	7	9	9
6	8	7	5	6	8	8	9	6
7	8	8	7	9	9	7	7	8
8	8	8	8	8	9	7	8	6
9	7	8	3	2	8	6	8	5
10	8	9	6	6	8	8	8	7
11	8	9	6	5	9	9	8	6
12	7	9	9	7	9	8	8	7
13	7	8	7	7	8	8	9	8
14	6	7	6	7	8	7	8	7
15	7	8	6	3	7	7	8	6
16	8	8	4	3	9	6	8	7
17	8	9	4	5	8	9	7	7
18	8	8	2	5	8	9	8	8
19	7	8	6	6	8	8	6	6
20	9	9	6	3	9	8	8	8
Mean	7.6	8.0	5.7	5.1	8.0	7.4	7.8	7.0
SD	0.69	0.83	1.66	2.00	1.03	0.99	0.97	0.97

Values ≤ 4 are in bold.

always performed by the same driving instructor in a manner similar to the obligatory Finnish driving test, using the official form. Two levels of errors were classified on the basis of their severity: (1) risky faults which could lead to danger, and (2) offences—that is, serious infringements of traffic regulations.

Comparisons of the study group with the control group were calculated by Mann-Whitney *U* test. Correlation analyses (Pearson's product moment correlation and Spearman's rank correlation coefficient) were used to measure the association between different variables. Stepwise regression analyses were used to determine which variables explain the variation in faults and offences in the driving test.

Results

Annual driving mileage and total driving experience were not related to faults or

offences in the driving test either in the patient group or in the control group.

The evaluation of driving ability in the Parkinson's disease group was a considerably more complicated task than the evaluation of a healthy person's ability to drive a car (table 3). The neurologist's evaluation of the driving ability of the patients with Parkinson's disease was much more optimistic than the traffic instructor's evaluation ($t=6.14$ $p<0.001$) or the one made by the psychologist ($t=5.69$ $p<0.001$). The evaluations of the psychologist and the driving instructor of the Parkinson's disease group's ability to drive correlated to a high degree (Spearman's rank correlation coefficient $r=0.745$ $p<0.001$), but there was no significant correlation between the assessments made by the neurologist and the instructor ($r=0.176$), nor between the neurologist's and the psychologist's evaluations ($r=0.220$). The

Table 4 Comparison of the results of the laboratory tests of the patients with Parkinson's disease (PD) ($n=20$) with those of the controls ($n=20$)

	PD		Controls		Mann-Whitney <i>U</i> test <i>p</i> Value
	Mean	SD	Mean	SD	
Visual memory:					
Correct responses	14.9	4.6	18.6	2.3	0.0071
Total time (s)	177	81	118	35	0.0041
Perception:					
Correct responses	182	10	187	9	0.0783
Total time (min)	13:54	04:30	12:07	02:33	0.3090
Vigilance:					
Correct responses	94	14	98	4	0.2418
Wrong responses	8	9	3	5	0.0741
Choice reactions:					
Correct reactions	31.6	16.1	48.0	7.8	0.0004
Omitted stimuli	19.0	16.9	5.6	5.5	0.0065
Incompleted reactions	17.1	10.5	14.8	5.5	0.7763
Wrong decision	0.5	0.8	0.1	0.3	0.1516
Motor speed (ms)	240	37	221	34	0.5221
Cognitive speed (ms)	896	96	863	76	0.1062
Information processing:					
Correct responses	239	105	393	65	0.0000
Correct and within the time responses	110	78	216	81	0.0003
Omitted responses	262	92	124	60	0.0000
Wrong responses	39	25	24	19	0.0365
Multiple responses	25	18	22	14	0.8037

Table 5 On road test records of patients with Parkinson's disease (PD) (n=20) and of control subjects (n=20)

	PD		Controls		Mann-Whitney U test p Value
	Mean (SD)	Range	Mean (SD)	Range	
Faults	13.2 (6.0)	3–25	9.0 (3.4)	2–16	0.0340
Offences	4.3 (3.7)	0–13	1.9 (2.0)	0–9	0.0119

patients' own evaluations differed significantly from those of the instructor ($t=5.42$ $p<0.001$) and of the psychologist ($t=4.85$ $p<0.001$). There was no disagreement between the three evaluations of the controls' general driving ability, and the controls were able to evaluate their driving ability adequately by themselves. According to every assessment all the controls were able to drive a car.

The patients with Parkinson's disease did worse than the controls in all the laboratory tests, the differences being most pronounced in the visual memory test, in the choice reaction time test, and in the test for information processing capacity in a complex situation (table 4). Both cognitive and psychomotor impairments were evident even in the mild to moderate stages of Parkinson's disease.

The groups differed in the number of classified faults in the driving test. The patients committed significantly more risky faults and offences than the controls. The individual variation was more pronounced in the Parkinson's disease group than in the control group (table 5). In terms of faults, driving in a traffic flow was a considerably more difficult task for the patients with Parkinson's disease than for the controls (mean (SD) of Parkinson's disease group 3.9 (SD 2.4), controls 1.6 (SD 1.4); $p=0.018$), as well as turning to the left (Parkinson's disease group, mean 1.7 (SD 2.1), controls, mean 0.6 (SD 0.6); $p=0.0461$). The Parkinson's disease group's problems in driving appeared mostly in urban conditions, and the groups did not differ from each other in highway driving. The driving instructor's overall evaluation correlated with the faults (Par-

kinson's disease group $r=-0.740$ $p<0.001$; controls $r=-0.532$ $p<0.05$) and the offences (Parkinson's disease group $r=-0.716$ $p<0.001$; controls $r=-0.593$ $p<0.01$) that occurred during the on road test, indicating that his evaluation was based on the recorded observation during the driving test.

The number of classified faults in the on road driving test correlated with laboratory tests (except for the visual memory test) in both of the groups (table 6), supporting previous findings^{31–33} that this test package measures the essential cognitive and psychomotor functions needed for driving a car.

Disease indices (such as duration of the disease, the Hoehn and Yahr scale, and the MMSE scale) did not show significant correlations with the results of the driving test. The dose of levodopa was slightly, but not significantly, associated with the driving test, and there was a slight positive correlation ($r=0.479$ $p<0.05$) between age and the faults in the driving test and between age and offences ($r=0.504$ $p<0.05$).

According to the stepwise regression model, slowness of visual processing, a dose of levodopa and age explained 67% of the variation in the faults in the driving test (total number of faults and offences) of the Parkinson's disease group. If age was excluded, then the model explained 56% of the variation. When only the laboratory variables were included, slowness of visual processing, errors in perception, and slowness in recalling visual material explained 62% of the variation in faults. Correspondingly, the wrong decisions in the choice reaction time test and slowness in information processing explained 46% of the variation in faults in the driving test in the control group.

Discussion

Our results show that Parkinson's disease significantly influences driving ability even in the mild and moderate stages although most patients in this sample seemed sufficiently competent to drive. Secondly, the evaluation of the driving ability of patients with Parkinson's disease seems to be a highly complicated task which even an experienced clinician cannot accomplish without the support of other specialists and driving related tests. The neurologist who took part in this study overestimated the driving ability his patients with Parkinson's disease, despite his extensive contact with them, and the patients themselves were not at all capable of evaluating their own driving ability reliably. Seven patients out of 20 (35%), whom the neurologist approved to drive were evaluated as being unable to drive on the basis of the driving test; they still drove actively. Two of them, both ranked very low by the driving instructor (2 and 3 on the 10 point scale) and by the psychologist (3 and 5), had each caused a traffic accident in the past two years. A third patient who had also caused an accident was ranked fairly high by all three specialists (7, 8, and 7). The controls had not caused any accidents during the previous two years.

Table 6 Pearson's correlation coefficient between laboratory variables and the number of faults and offences recorded in the on road driving test in the patients with Parkinson's disease (PD) (n=20) and the controls (n=20)

	PD		Controls	
	Faults	Offences	Faults	Offences
Visual memory:				
Correct responses	0.069	-0.069	-0.236	-0.059
Total time	0.023	-0.178	0.034	0.110
Visual perception:				
Correct responses	-0.804***	-0.168	-0.054	-0.045
Total time	0.595**	0.508*	0.590**	0.341
Vigilance and concentration:			(n=11)	(n=11)
Correct responses	-0.609**	-0.266	-0.341	-0.027
Wrong responses	0.232	0.077	0.724**	0.481
Reaction time/response	0.179	-0.045	0.590*	0.356
Choice reactions:				
Correct reactions	-0.561**	-0.376	0.335	-0.138
Wrong decisions	0.036	0.034	0.523*	0.175
Decision time	0.520*	0.691***	0.286	-0.291
Motor time	0.433*	-0.272	-0.250	-0.075
Information processing:				
Correct responses	-0.624**	-0.859***	-0.468*	0.010
Correct and within time responses	-0.449*	-0.748***	-0.503*	-0.192
Omitted stimuli	0.690***	0.888***	0.519*	-0.063
Wrong responses	0.265	0.511*	-0.063	0.194
Multiple responses	-0.139	0.220	-0.281	0.230

* $p<0.05$; ** $p<0.01$; *** $p<0.001$.

It should be noted that the range of evaluations differed substantially between the specialists. The driving instructor gave low scores, indicating disability, for seven patients with Parkinson's disease and his range for the controls extended from five to nine. The psychologist also used almost the whole range, but the neurologist was fairly conservative, avoiding extreme values. Our results might simply arise from this scaling difference as the two specialists, aware of the fact that half of the subjects had Parkinson's disease, may have indicated lower values whenever they noted any symptom of the disease. However, this is not a plausible explanation as there was no correlation between the evaluations of the neurologist and those of the driving instructor and the psychologist, and as the estimations of the last two (mutually independent) were based on structured observations in real traffic or on objective cognitive and psychomotor tests.

Who is right, then, in evaluations of driving ability? Our joint opinion is that we should place more trust on the driving instructor's and the psychologist's results, as their estimates are directly based on traffic specific information. In this study all the specialists were highly experienced, each of them having had at least five years of active work in this area, but only one specialist was used in each category. Therefore, to confirm the results larger patients groups, more evaluators in each category, and new evaluators (for example, general practitioners) should be included in further research.

A further point should be raised. The major problems experienced by the patients with Parkinson's disease in the on road test occurred in fairly heavy traffic in an unfamiliar city. Drivers generally adapt their behaviour to the requirements set by traffic conditions, but they also tend to compensate for their impaired skills by avoiding difficult environments or situations.⁴ It is possible, therefore, that we put our subjects into too demanding conditions considering their driving needs. On the other hand, sufficient demands are necessary to create critical situations and variety which make a proper assessment of driving ability possible. However, the patients with Parkinson's disease who were ranked by the driving instructor as being unable to drive evaluated their own skills as satisfactory or even good. Although they might have been pretending just to keep their driving licence, the lack of correlation between their self evaluation and the driving instructor's evaluation suggests that their continued driving is likely to result in risks to themselves and to other road users.

Our results show that the cognitive and psychomotor impairment noticeable even in the early stages of Parkinson's disease can be assessed through specific laboratory tests and on road tests. The results support the findings of Dubinsky *et al.*²⁷ concerning the unreliability of questionnaires and severity scales in evaluations of driving ability. Similarly, we found no significant correlations between Hoehn and Yahr, MMSE, and the driving test. Disease indices such as duration of the disease and the

dose of levodopa medication were not directly linked to performance in the driving test.

The laboratory tests used in the present study correlated to a high degree with the driving test both in the patient group and in the control group. The tests also distinguished patients with Parkinson's disease from controls. Slowness of cognitive processing in Parkinson's disease was the key factor in the test variables.^{13 24 26} According to our study an evaluation of the driving ability of patients with Parkinson's disease should be based on a test package which includes at least the following tests: (1) vigilance and concentration, (2) visual perception, (3) choice reaction times, (4) information processing in a complex situation.

In conclusion, the driving ability of patients with even mild to moderate Parkinson's disease is clearly impaired. The highly complex task of evaluating the driving ability of patients with Parkinson's disease requires both psychological and psychomotor tests, and/or an on road driving test.

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NEUROLOGICAL STAMP

Louis Pasteur (1822-95)

Pasteur, the son of a tanner, was not a physician but became the most important medical scientist working in the 19th century. His earlier work which led to the discovery of the molecular asymmetry of tartaric and racemic acid had a profound consequence for structural chemistry. The crystals of tartaric and racemic acid had the same chemical structure but had different effects on polarised light. By 1856 he had begun his work on fermentation beginning with fermentation of milk into lactic acid. He reported the presence of micro-organisms which continued to bud and multiply. He was able to declare that the multiplication of the micro-organism resulted in true fermentations and caused wine and milk to become sour. Heating or "pasteurisation" as it was called prevented this occurring. This early work on fermentation and the demonstration that if heated, wines no longer went sour saved the French wine industry. Pasteur disproved spontaneous generation and demonstrated that life floated in the air as countless bacteria. Pasteur's proof of the existence of atmospheric germs led Lister to apply the principle to surgery with amazing results. In 1865 he began investigating a disease devastating silk worms in southern France. Despite a stroke in 1868 (left sided) and considerable confusion caused by two independent infections, he was able to provide a comprehensive analysis of the disease and its prevention. In 1877, turning to human disease he pioneered effective methods of treatment against virulent infections. The breakthrough came in 1880, as a result of a batch of chicken cholera standing in the laboratory over the long hot summer. Injection of this chicken cholera into healthy chickens produced only mild transient disease and

then when the chickens were injected with fresh bacillus they survived unscathed. Pasteur had accidentally discovered an attenuated vaccine. By May 1882 he had produced a comparable vaccine against anthrax and in 1885 he used rabies vaccine, recently developed by him, in a badly bitten 9 year old Alsatian boy, Joseph Meister. (Meister committed suicide 55 years later in 1940 when, as a caretaker of the Pasteur Institute, he preferred to die rather than open the tomb of Pasteur to the invading Nazi forces.) A few months later Pasteur successfully inoculated a shepherd from Jura named Jupille who had been bitten by a rabid dog while grappling with it in an effort to save his comrades. Pasteur had not only administered the first protective treatment for rabies in humans on 6 July 1885, but was also responsible for launching the science of immunology and protective vaccination. Pasteur concluded that the agent causing rabies had its seat in the nervous system. In 1903 Adelchi Negri (1876-1912), an Italian physician and pathologist, described the hallmark of the infection, small round oval occlusions—Negri bodies—in the protoplasm and the processes of the nerve cells, but especially in the hippocampus of rabid animals. Agriculture, industry, medicine, and humanity are indebted to this remarkable scientist. Pasteur is shown here on a stamp issued in 1936 (Stanley Gibbons 566, Scott B53) Surtax was used for the relief of unemployed intellectuals. Alongside, another stamp issued in 1985 commemorates the centenary of antirabies vaccination (Stanley Gibbons 2684, Scott 1979). Pasteur is shown at the inoculation of the patient.

L F HAAS