Are sensorimotor strokes lacunar strokes? A case-control study of lacunar and non-lacunar infarcts

G Landi, N Anzalone, E Cella, E Boccardi, M Musicco

Abstract
To determine whether sensorimotor strokes should be considered as lacunar syndromes, 34 consecutive patients with first-ever ischaemic sensorimotor stroke were evaluated and compared with 103 patients with non-lacunar infarcts and another 88 patients with lacunar infarcts. Potential thromboembolic sources were more frequent in patients with non-lacunar infarcts (p = 0.003, versus sensorimotor strokes). Although the overall prevalence of hypodense lesions at CT scan was not significantly different among the three groups, lacunar lesions were found in 47-1% of sensorimotor strokes, compared with 6-8% of non-lacunar infarcts (p < 0.0001). In a mean follow-up period of 287 months, the incidence of stroke and myocardial infarction among sensorimotor strokes was similar to that of patients with lacunar infarct, but significantly lower than in non-lacunar infarcts (p < 0.05). These results demonstrate important differences between sensorimotor and non-lacunar infarcts, but quite similar findings in sensorimotor and lacunar strokes, and thus support the theory that sensorimotor strokes are commonly due to lacunar lesions.

Lacunar infarctions are a subgroup of ischaemic strokes characterised by well-defined clinical syndromes resulting from small infarcts in the deep structures of the brain. They are commonly attributed to small artery disease, as opposed to cardioembolic or large artery thromboembolic strokes. As summarised by a recent review, evidence appears to support this hypothesis. In addition to the classic lacunar syndromes (first described by Fischer), sensorimotor stroke has also been included among the lacunar strokes by some authors, but others point to the limited pathological confirmations available to support this opinion. Weisberg argued that the blood supply to the internal capsule and thalamus are usually separate, and attributed sensorimotor stroke to embolic or atherosclerotic disease, as opposed to lacunar strokes which he ascribed to arteriolar disease. In a recent population-based survey sensorimotor stroke accounted for 38-2% of all lacunar infarcts, and for 9-4% of all first-ever ischaemic strokes. It is thus important to establish whether sensorimotor strokes, as a group, are caused by small artery disease or whether they occur as a result of embolic or atherosclerotic mechanisms. We therefore conducted a prospective study on a series of patients with sensorimotor stroke and compared them to a group of definite lacunar infarcts, and to another group of non-lacunar infarcts.

Patients and methods
This prospective study included 225 consecutive patients with first-ever ischaemic stroke (symptoms lasting more than 24 hours) who were seen between 1 August 1983 and 31 January 1987 at the emergency room of the Policlinico Hospital in Milan within 72 hours of the onset of symptoms, and then admitted to hospital at the local neurological department. The clinical diagnosis was confirmed in all cases by CT scan which was performed on admission and repeated five to eight days. CT (performed in the first part of the study with an EMI 1010 and later with a GE 8800 scanner) was reviewed by a neuroradiologist (EB) who was unaware of the clinical picture. Hypodense areas were classified, according to their location, into superficial and deep; deep lesions were diagnosed as lacunar infarctions if their maximum diameter did not exceed 15 mm on more than two adjacent 10 mm tissue sections.

All patients had neurological and general physical examination on arrival at the emergency room; this was repeated daily during the first week and twice weekly until discharge. This enabled the study sample to be subdivided into three subgroups according to their neurological picture at the time of maximum deficit.

Patient types
Lacunar infarcts (LI) (n = 88), defined as producing: pure motor hemiparesis, pure sensory stroke, ataxic hemiparesis, dysarthria-clumsy hand syndrome, hemichorea/hemiballism.

Sensorimotor strokes (SMS) (n = 34) are defined as producing concomitant ipsilateral motor and objective sensory loss, both involving at least two of three areas (face, arm, leg).

Non-lacunar infarcts (NLI) (n = 103): this subgroup included all patients whose clinical picture did not conform to the preceding subgroups, but we also excluded all patients (n = 61) with impairment of consciousness on admission because of their poor short-term prognosis.

All patients included in the study were screened for the presence of cerebrovascular disease risk factors, such as hypertension (blood pressure higher than 160/90 mmHg or
regular use of antihypertensive drugs), smoking (10 or more cigarettes per day during the previous 6 months), diabetes (fasting blood sugar levels higher than 140 mg% or regular use of antidiabetic drugs), hyperlipidaemia (plasma cholesterol and/or triglycerides higher than 280 mg% and 200 mg%, respectively) and occurrence of previous transient ischaemic attacks (TIA).

All patients had ECG, chest X-ray and a cardiological examination. Further examinations were performed depending on specific diagnostic needs as follows: M-mode and B-mode echocardiograms were obtained in 61 patients: 31 non-lacunar infarcts (30-1%), 10 sensorimotor strokes (29-4%), and 20 lacunar infarcts (22-7%); and 24 hour ECG monitoring was performed on 13 patients: seven non-lacunar infarcts (6-8%), two sensorimotor strokes (5-9%), four lacunar infarcts (4-5%). Potential sources of cardiac emboli were diagnosed in patients with atrial fibrillation, endocarditis, mitral valve disease, and left ventricular thrombus. Ischaemic heart disease was diagnosed in patients with a typical history of angina or acute myocardial infarction, or with clear-cut ECG evidence of previous myocardial infarction. A continuous-wave Doppler study of the extracranial vessels was performed, as described by Büdingen et al., in all patients except three (all with non-lacunar infarcts) who died soon after admission to hospital. Sixty eight patients, considered for carotid endarterectomy, had cerebral angiography by transfemoral catheterisation. Their angiographic picture was correlated with the results of their previous ultrasound examination. Deaths occurring as a consequence of stroke were recorded, and residual disability at one month was classified as follows: "absent" (independent in self-care, with return to pre-stroke activities); "mild" (requiring help in some activities of daily living, but able to walk without assistance); "moderate" (requiring help in all activities of daily living, walks only with assistance); "severe" (bedridden or chair-bound).

All patients who survived their first stroke were followed for at least 12 months or until death. Antithrombotic treatments were recorded, but no attempt was made to unify prescription patterns of different physicians.

However, control of risk factors was pursued in all cases. Occurrence of new cerebrovascular events (which were subdivided according to duration of symptoms into TIA's and strokes) was reported. A CT scan was obtained whenever possible in patients with stroke recurrence, which was classified as: "mild" (no, or only mild persistent deterioration of the previous neurological deficit); "severe" (significant deterioration of the previous neurological deficit); and "fatal". The occurrence of myocardial infarction was also recorded: diagnosis was accepted in patients with typical chest pain who also had concordant ECG and enzymatic alterations. Finally, deaths from other causes were also recorded.

Statistical Methods

The patients with sensorimotor stroke were taken as the diagnostic reference group, and the strength of association between this group and the two diagnostic groups and the considered variables was calculated by means of odds ratios. Confidence intervals of odds ratios were calculated by Cornfield's method. The cumulative time-dependent probability of survival, free from major vascular events (stroke, myocardial infarction), was evaluated by survival analysis, according to the Kaplan-Meier method. The follow up times of patients who died from causes other than major vascular events were censored to the time of death.

Results

The age- and sex-distribution of patients in our three groups was relatively homogeneous as shown in table 1, which also reports the prevalence of cerebrovascular risk factors. There was no significant difference between the group with sensorimotor stroke and the other two groups (fig 1), although the frequency of hypertension, diabetes and hyperlipidaemia was rather similar among sensorimotor strokes and lacunar infarcts but lower in patients with non-lacunar infarcts.

Clinical findings are also summarised in table 1. A non-sudden onset of symptoms with a gradual, stepwise or stuttering course up to the time of maximum deficit was observed more often among patients with sensorimotor stroke than among those with non-lacunar infarcts, but the difference was only of borderline statistical significance (fig 1). There was no significant difference in the occurrence of head- ache among our three groups, although this information could not be reliably gathered from those patients with non-lacunar infarcts who had severe aphasia.

Short-term prognosis of sensorimotor strokes appeared favourable, compared with that of lacunar infarcts (table 1): no death occurred as a direct consequence of stroke, and a high percentage of patients (82-4% with sensorimotor strokes, and 81-9% with lacunar infarct) were left at one month with no or only mild residual disability. In contrast, the short-term prognosis of non-lacunar infarcts was significantly worse (chi square for trend = 9-27, versus sensorimotor strokes; p = 0-002), as 14-6% of

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Table 1 Age, sex, prevalence of cerebrovascular risk factors and clinical features according to stroke subtype

<table>
<thead>
<tr>
<th>Non-lacunar infarcts (n = 103)</th>
<th>Sensorimotor strokes (n = 34)</th>
<th>Lacunar infarcts (n = 88)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Mean age (range)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Males</td>
<td></td>
<td></td>
</tr>
<tr>
<td>63 (61-2%)</td>
<td>63 (64-7%)</td>
<td>63 (62-5%)</td>
</tr>
<tr>
<td>Hypertension</td>
<td>54 (52-4%)</td>
<td>57 (54-6%)</td>
</tr>
<tr>
<td>18 (17-5%)</td>
<td>10 (9-2%)</td>
<td>10 (9-8%)</td>
</tr>
<tr>
<td>Smoking</td>
<td>18 (17-5%)</td>
<td>18 (16-5%)</td>
</tr>
<tr>
<td>Previous TIA</td>
<td>16 (15-5%)</td>
<td>3 (8-8%)</td>
</tr>
<tr>
<td>Sudden onset</td>
<td>85 (82-5%)</td>
<td>3 (8-8%)</td>
</tr>
<tr>
<td>Headache</td>
<td>18 (17-5%)</td>
<td>7 (7-0%)</td>
</tr>
<tr>
<td>Residual disability* (at one month)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>absent</td>
<td>31 (30-1%)</td>
<td>17 (16-5%)</td>
</tr>
<tr>
<td>mild</td>
<td>11 (10-3%)</td>
<td>21 (23-3%)</td>
</tr>
<tr>
<td>moderate</td>
<td>24 (23-3%)</td>
<td>3 (8-8%)</td>
</tr>
<tr>
<td>severe/dead</td>
<td>14 (15-2%)</td>
<td>30 (8-8%)</td>
</tr>
</tbody>
</table>

*Chi square for trend analysis: SMS versus NL: Chi square = 9-27; p = 0-002; SMS versus LI: Chi square = 0-28; p = ns.
prevalence of vascular lesions in our population. This was considered feasible as a correlation of Doppler results with 68 subsequent four-vessel angiographic findings revealed a sensitivity of 95-0% and a specificity of 97-9% in the diagnosis of stenoses > 50% or occlusion of the carotid and vertebral arteries. Doppler results, reported in table 2, were pathological in 26 2% of non-lacunar infarcts (0-05 < p < 0-06, versus sensorimotor strokes (fig 2)), and overall 54-4% of patients in this subgroup had stenosis or occlusion of the symptomatic arterial district, embolic heart disease, or both. The prevalence of potential thromboembolic sources was significantly lower among sensorimotor strokes (23-5%; p = 0-003), whereas no significant difference was found between sensorimotor strokes and lacunar infarcts.

The results of the CT scans are reported in table 3. The examination disclosed a congruous infarct (that is, appropriate to the side of symptoms) in 19 sensorimotor strokes (55-9%); 16 of them (84-2%) had a lacunar lesion, whereas the other three had either a cortical infarct or a larger subcortical lesion. In all patients with lacunar infarcts, CT scan was either normal or showed evidence of a small deep lesion in the appropriate side of the brain. By contrast, out of the 71 patients with non-lacunar infarcts and a congruous hypodense area (68-9%), 64 had evidence of a cortical or large subcortical infarct (p < 0-0001 versus sensorimotor strokes), and only seven had evidence of an appropriate lacunar infarct (p < 0-0001 versus sensorimotor strokes) (fig 2).

All 210 patients who survived after their first stroke were followed up for an average of 28-7 months. New vascular events occurred in 23-5% of sensorimotor strokes and in 21-6% of lacunar infarcts, as opposed to 30-7% of non-lacunar infarcts (table 4). This difference became even more marked when only major vascular events (that is, stroke and myocardial infarction) were considered, as they occurred in 14-7% of sensorimotor strokes and 13-6% of lacunar infarcts, but in as many as 27-3% of non-lacunar infarcts. Severity of stroke recurrence was not appreciably different among the three groups. Survival analysis for major vascular events (fig 3) confirmed a significantly different long-term prognosis among the three groups (chi square = 10-04, p = 0-007), with a worse outcome for non-lacunar infarcts compared with sensorimotor strokes (chi square = 3-87, p < 0-05), whose prognosis appeared strikingly similar to that of lacunar infarcts (chi square = 0-03, p > 0-85).

### Table 2. Cardiological findings and prevalence of thromboembolic sources

<table>
<thead>
<tr>
<th></th>
<th>Non-lacunar infarcts (n = 103)</th>
<th>Sensorimotor strokes (n = 34)</th>
<th>Lacunar infarcts (n = 88)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Normal, or irrelevant ECG abnormalities</td>
<td>52 (50.5%)</td>
<td>22 (64.7%)</td>
<td>65 (73.8%)</td>
</tr>
<tr>
<td>Ischaemic heart disease</td>
<td>13 (12.6%)</td>
<td>5 (14.7%)</td>
<td>8 (9.0%)</td>
</tr>
<tr>
<td>Atrial fibrillation</td>
<td>11 (10.6%)</td>
<td>1 (2.9%)</td>
<td>5 (5.6%)</td>
</tr>
<tr>
<td>Other embolic heart disease</td>
<td>18 (17.4%)</td>
<td>3 (8.8%)</td>
<td>4 (4.5%)</td>
</tr>
<tr>
<td>Ischaemic + embolic heart disease</td>
<td>3 (3.0%)</td>
<td>1 (2.9%)</td>
<td>2 (2.2%)</td>
</tr>
<tr>
<td>Other cardiopathies</td>
<td>4 (5.6%)</td>
<td>2 (5.9%)</td>
<td>4 (4.5%)</td>
</tr>
<tr>
<td>Stenosis/occlusion of symptomatic vessel (Doppler findings)</td>
<td>27 (26.2%)</td>
<td>3 (8.8%)</td>
<td>16 (18.2%)</td>
</tr>
<tr>
<td>Total with ascertainment thromboembolic source</td>
<td>56* (54.4%)</td>
<td>8 (23.5%)</td>
<td>27 (30.7%)</td>
</tr>
</tbody>
</table>

*Four patients had comorbidity of embolic heart disease and stenosis/occlusion of the symptomatic vessel.
Cardiological findings:
- Normal, or irrelevant ECG abnormalities
- Ischaemic heart disease
- Potential cardiac embolic source
- Doppler findings:
  - Stenosis/occlusion of symptomatic vessel
  - Aspectended thromboembolic source (overall)
- CT findings:
  - Normal, or incongruous lacunar infarct
  - Congruous lacunar infarct
  - Congruous non-lacunar infarct

![Figure 2](image)

**Comparison of the prevalence of cardiac disease, Doppler findings and CT scan results in sensorimotor strokes (SMS) versus lacunar infarcts (LI) (right) and non-lacunar infarcts (NLI) (left). For explanation, see fig 1.**

**Discussion**

We included in our hospital-based study only patients admitted to a neurological department. While this may have biased the relative proportions of patients in our three subgroups, it enabled us to study them more extensively than is usually possible in population-based studies. All our patients received a homogeneous evaluation for analysed items, with the exception of cerebral angiography, and of Doppler ultrasounds (not available in only three patients).

We subdivided our patients into one of the three groups on the basis of their neurological findings, a simple and widely applicable method in all clinical and research settings. Conversely, extensive attempts to classify cerebral infarcts according to their aetio- pathogenesis would leave as many as 40% of cases with a final diagnosis of "infarct of undetermined cause". 18

To avoid misdiagnosis of lacunar stroke in patients recovering from more extensive clinical deficits, we included only patients visited within three days of their first-ever stroke (89% of the patients were seen within 24 hours). These were classified according to their neurological features at the time of maximal deficit, as determined by repeated examination during their hospital stay.

The results of our study demonstrate a striking similarity between sensorimotor strokes and lacunar infarcts, but several impor-

tant differences between the former group and non-lacunar infarcts. Embolic heart disease was found in almost one third of patients with non-lacunar infarcts, but only in 14-7% of our patients with sensorimotor strokes and in 12.5% of patients with lacunar infarcts. This agrees with the 10% prevalence among patients with lacunar infarcts in the Harvard Cooperative Stroke Registry, as well as with the 12.3-13.0% frequency in two other reports on lacunar infarcts.25 Scant data are available on angiographic findings in sensorimotor strokes, as either they were reported together with those of other lacunar strokes, or the number of patients was too small to account for any conclusion. The frequency of angiographic abnormalities among patients with lacunar infarcts ranges from 0-75% according to several studies.26-23 This high variability may reflect some bias in the selection of patients who are submitted to invasive examination, as well as the impact of angiographic results on the final diagnostic classification. For example, in the Harvard study22 13 diagnoses of lacunae were changed to thrombosis on the basis of angiography. Some studies have employed Doppler ultrasound to detect haemodynamically significant vascular lesions in patients with lacunar infarcts, reporting a prevalence of 17-27%.14 24 25 Loeb et al25 observed one case of carotid stenosis among 11 patients with sensorimotor strokes, compared with 18 cases out of 45 patients with non-lacunar infarcts.

We found symptomatic arterial stenosis or occlusion in 23.0% of patients with non-lacunar infarcts, but only in 8.8% of patients with sensorimotor stroke. Overall, potential thromboembolic sources of cardiac or arterial origin were present in 23.5% of our patients with sensorimotor stroke, but in more than twice as many patients with non-lacunar infarct. This is a highly significant difference that shows the different pathogenetic mechanisms behind the non-lacunar infarcts as compared with sensorimotor stroke and lacunar infarcts. It is interesting that 40-9% of lacunar infarcts and 33.5% of sensorimotor strokes in our study experienced a gradual, fluctuating or stuttering course, as opposed to only 17.5% of non-lacunar infarcts. A leisurely mode of onset characterises many lacunar infarcts, in contrast to the sudden onset of most cases of major atheromatous or embolic strokes.21 Thus although it did not reach conventional statistical significance (p = 0.11), this difference in mode of onset between sensorimotor strokes and non-lacunar infarcts points indirectly to a difference in the mechanisms underlying cerebral ischaemia in these two groups.

Additional evidence supporting the theory that sensorimotor strokes are similar to lacunar

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**Table 3 CT findings according to stroke subtype**

<table>
<thead>
<tr>
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<th>Lacunar infarcts (n = 88)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Normal, or incongruous infarct</td>
<td>32 (32.1%)</td>
<td>15 (44.1%)</td>
<td>51 (56.0%)</td>
</tr>
<tr>
<td>Congruous lacunar infarct</td>
<td>7 (6.5%)</td>
<td>16 (47.1%)</td>
<td>37 (42.0%)</td>
</tr>
<tr>
<td>Congruous non-lacunar infarct</td>
<td>64 (62.1%)</td>
<td>3 (8.8%)</td>
<td>0</td>
</tr>
</tbody>
</table>
infarcts is provided by CT findings. Although none of our patients with typical lacunar syndrome had CT evidence of a superficial or > 15 mm diameter infarct, such lesions have been reported in previous papers, with a frequency ranging from 1-6%–16-2%. Since the 42.0% detection rate of appropriate lesions among the lacunar infarcts was comparable to that of other large series, we attribute the absence of large or superficial lesions in our lacunar population to chance. Although significant from a statistical point of view, we do not feel the difference from sensorimotor strokes to be of clinical importance. Large or superficial lesions were actually found in a minority of our patients with sensorimotor strokes (8-8%), a slightly higher proportion than reported by Bamford (4.9%)20, but lower than the 21.6% observed by Huang et al28 among their ischaemic patients with sensorimotor strokes. Although the frequency of appropriate ischaemic lesions was higher among non-lacunar infarcts than among the sensorimotor stroke patients, this difference was not statistically significant. Highly significant differences, however, were apparent if the type of lesion is considered, as 84-2% of infarcts among patients with sensorimotor strokes were of the lacunar type, as opposed to only 9-9% of ischaemic lesions in the subgroup with non-lacunar infarcts.

Published reports have stressed the favourable short-term prognosis of patients with lacunar strokes, who have a very low mortality10 and a less severe neurological impairment8 than patients with other stroke subtypes. These conclusions agree with the results in our series, where mortality was absent among both lacunar infarct sensorimotor strokes and patients, and more than 80% of these patients experienced a good recovery. This compares closely to the findings of Bamford et al30, who reported that 23-1% of sensorimotor stroke and 19-5% of lacunar infarct patients had lost their previous capability of independent existence one month after stroke. By contrast, the outcome of non-lacunar infarcts was significantly worse than sensorimotor strokes for both mortality and residual deficit. Two studies reported on the long-term outcome of patients with lacunar infarcts, and both incorporated sensorimotor strokes into this definition. Gandolfo et al30 found that the average recurrence rate for new cerebrovascular episodes among their lacunar strokes was 4.74% per 100 patient-years, much lower than previously reported in survivors from cerebral infarction. On the other hand, Bamford et al10 observed a one year recurrence rate of 11.8% among lacunar infarcts, which they considered similar to that of other groups of patients with stroke. The younger age of the patients may account for the lower recurrence rate observed in this and other30 hospital-based studies. By comparing the survival curves of three subgroups of patients recruited uniformly during the same time period, our study provides evidence that major vascular events were more frequent among non-lacunar infarct than among sensorimotor stroke patients, whose prognosis was rather similar to lacunar infarcts.

In conclusion, our study demonstrates several important differences between sensorimotor strokes and non-lacunar infarcts, but quite similar findings in sensorimotor strokes and lacunar infarcts. While careful investigation is essential to identify the minority of patients in whom sensorimotor stroke is caused by thromboembolism, our results support the theory that sensorimotor stroke is commonly due to lacunar infarcts resulting from small vessel disease. In clinical and therapeutic studies they should therefore be included in this distinct subgroup of patients as they share the same pathogenesis and favourable short and long term prognosis.

We thank Mr Luigi Grilli for his help in the preparation of the manuscript.


Table 4 Incidence and type of events during follow up according to stroke subtype

<table>
<thead>
<tr>
<th></th>
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<th>Sensorimotor strokes (n = 34)</th>
<th>Lacunar infarcts (n = 88)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Mean follow up (months)</td>
<td>25.1 (9-7%)</td>
<td>31.5 (9-7%)</td>
<td>31-1 (21-6%)</td>
</tr>
<tr>
<td>Total vascular events</td>
<td>27 (9-7%)</td>
<td>19 (21-6%)</td>
<td>9 (21-6%)</td>
</tr>
<tr>
<td>TIA</td>
<td>3 (9-7%)</td>
<td>2 (9-7%)</td>
<td>3 (9-7%)</td>
</tr>
<tr>
<td>Stroke mild</td>
<td>5 (9-7%)</td>
<td>1 (9-7%)</td>
<td>0 (9-7%)</td>
</tr>
<tr>
<td>Stroke severe</td>
<td>11</td>
<td>11</td>
<td>11</td>
</tr>
<tr>
<td>Myocardial infarct (fatal)</td>
<td>6 (9-7%)</td>
<td>2 (5-9%)</td>
<td>3 (9-7%)</td>
</tr>
<tr>
<td>Non vascular deaths</td>
<td>6 (9-7%)</td>
<td>2 (5-9%)</td>
<td>3 (9-7%)</td>
</tr>
</tbody>
</table>

Figure 3 Cumulative probability of survival, free from stroke and myocardial infarction, according to stroke subtype. Numbers are those of patients at risk at the beginning of each six month time interval.
Neurological stamp

Ambrose Paré 1510–90

The influence of Paré on sixteenth century surgery was as
great as Vesalius' on anatomy and like Vesalius he based his
work on original observation. As a result surgical treatment
became more humane. Among his many contributions,
Paré showed that wounds need not be cauterised by
applying boiling oil and that haemorrhage after amputation
could be controlled by ligature instead of cautery. "Je le
panse, Dieu le guartit" ("I treated him, God healed him")
was a favourite expression of his.

Paré introduced new instruments for opening the skull,
and wrote on wounds of the head and nerves. He was
surgeon to four kings of France, becoming in 1552 surgeon
to King Henry II. In 1559 Henry, whilst celebrating the
marriage of his daughter Elizabeth to Philip of Spain, was
struck in the eye by a lance at a jousting tournament. Great
physicians, including Paré, were sent for. To discover the
nature of the injury an experiment was performed in which
the stump of a lance was applied to the heads of four
executed criminals. When Vesalius arrived soon after, he
correctly forecast that the King would die. Post-mortem
showed this was due to cerebral compression from a contrecoupe
injury and subdural haemorrhage.

This stamp was one of a series issued in 1943 to honour
famous sixteenth century Frenchmen. (Stanley Gibbons
No 793, Scott No B163).

LF HAAS
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