which agents were used, the doses, or when the last morning
dose was given. This may be relevant for drugs with a long
half-life, or if slow-release preparations have been adminis-
tered. Furthermore, there is little discussion on possible
effects of these agents on the results obtained. There is
insufficient information to allow us to see how conclusions
regarding the pharmacological receptors involved in changes
in renal and cardiovascular haemodynamics were derived.

The authors state in the discussion that myocardial is-
chemia has been reported with dopexamine, and incorrectly
cite the work of Dawson and colleagues [2], who showed no
alteration in myocardial oxygen demand or supply with
increased rate-pressure products. Although two patients
reported mild chest pain with 6 μg kg⁻¹ min⁻¹, there was no
ECG evidence of myocardial ischaemia or a greater increase
in myocardial oxygen consumption than the group mean. The
absence of adverse effects of dopexamine on the myocardium
has been observed by others [3]. However, the ST segment
depression in this study, shown to occur in patients receiving
dopamine [1] and dopexamine [2], emphasizes that caution
should be exercised when using inotropic stimulation in
patients with coronary artery disease.

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Sirs.—We are aware of the fact that equipotent doses of
dopexamine and dopamine are difficult to define because of
differing receptor activities. This is the reason why we did not
perform a statistical comparison between the groups. As we
were more interested in renal than in cardiovascular effects of
both drugs, we started with dopexamine 1 μg kg⁻¹ min⁻¹ and
dopamine 2.5 μg kg⁻¹ min⁻¹ because these doses resulted in
comparable increases in renal blood flow. Then we looked at
the effects of twice these doses. Our results show clearly that,
in contrast with dopamine, dopexamine increased renal blood
flow mainly by an increase in cardiac index. Nevertheless, we
admit that (as with any other clinical study) we can only
speculate on the different receptor potencies of both agents.

We observed that all patients were receiving maintenance
doses of calcium channel blocking drugs and that two patients
in the dopexamine group and three patients in the dopamine
group were treated additionally with β-receptor antagonists.
We stated that the last doses of all drugs were administered in
the morning of the operation, but omitted to note that these
drugs were nifedipine and atenolol. We did not comment on
the doses, because oral doses do not allow any conclusions to
be drawn on plasma concentrations.

We do not think that we cited the work of Dawson and
colleagues incorrectly, because the common surface ECG is not
always a sensitive marker of myocardial ischaemia. Berry and
colleagues [1] examined the effects of selective regional
myocardial ischaemia by occluding vessels during percu-
taneous coronary angioplasty. While ST elevation occurred in
84% of patients during occlusion of the left anterior
descending artery and in 92% of patients during occlusion of
the right coronary artery, only 32% of patients undergoing
occlusion of the left coronary artery demonstrated ST
elevation in the routine surface ECG; four of 19 patients of the
latter group had only precordial ST depression, and nine
(47%) patients had no ECG changes. Moreover, unaltered
global myocardial oxygen consumption does not exclude
regional myocardial ischaemia, which can be detected bio-
chemically only by an impaired lactate balance, or even better,
by sensitive markers of ischaemia, such as hypoxanthine [2].

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MEMORY MECHANISMS

Sirs.—I read with interest the Editorial in the November issue
of the Journal, "Awareness and memory in anaesthetized
patients". It contains a short review of memory mechanisms to
which I wish to add the following comments.

First, the anatomical basis for memory formation was
proposed long before Ramon y Cajal. There is, for example,
an account in 1780 by the Italian anatomist Malacarne [1] of an
impressive experiment in which he trained one of two siblings
of various species (dogs and several birds) and left the other
sibling untrained. After a long period, he killed all the animals,
examined the brains and reported that there were more folds in
the cerebellum of the trained animals than in the untrained
ones.

My second comment refers to the absence of any reference
to cholinergic mechanisms within the hippocampus (and
elsewhere in the forebrain) which mediate memory processes.
As anaesthetists, we are very familiar with the effects of
hyoscine on memory.

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