as incorporating hydrophobic membranes. This is not the case for many devices.

We always read published studies with proposals for new techniques with interest and enthusiasm, but we are disappointed that these points were not detected during the refereeing process for the paper. However, as the data and methods used differ markedly from those produced by independent experts in Working Group 4 of draft standard CEN/TC 215 and in view of the errors which we have identified, we felt that this paper should be questioned.

References


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M. H. Becquemin

Reply

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Sir: We were very interested in Dr. Peter Ball's comments on our paper and are pleased to have the opportunity to clarify some aspects of our study as follows:

1. Concerning the retention efficiency calculations: in each case, the total number of particles, although being as close as possible to the theoretical value of 1500, was not exactly equal to this number (impossible to obtain in experimental conditions). So this explains the small discrepancies between our retention efficiency values and the ones calculated by Dr Ball.

2. Figure 1 and Table 2 do not deal with the same results: Fig. 1 shows the particle sizes which are completely blocked by each filter. Table 2 indicates the sizes of particles that have passed through the filters. This is the reason why the Fig. 1 sizes are larger.

3. The microbial test method quoted by Dr. Ball is certainly an excellent method but it does not measure the same things: our method allows detection of aerosols with or without microbial charge but potentially toxic.

4. It is well known among users of devices such as the APS (but was not sufficiently specified in our paper) that their very high precision has a counterpart which is the observance of a limit to aerosol concentration in order to avoid coincidence errors. This limit we calibrated at 1500 per ml. Our counts were made during 20 s – that is, on 1 litre of aerosol. This brings the actual number of particles to 1500 x 1000 = 1.5 x 10^6.

5. Concerning the description of the filters, our objective was not to go into very technical details but to test their retention efficiency for practical clinical use. In conclusion, the laser velocimetry method, which allows many repeated reliable measurements, provided the opportunity to make a very complete comparison between 11 types of filters. The high initial aerosol concentrations used for the test pointed out very small levels of particles through all the filters (0.005 %). The important point is that the probability of particles passing through filters is very low in normal circumstances but increases at very high concentrations and after 24-h use. These observations might help clinicians for the patient's benefit.

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J. P. Bourdarias

Cardiac dysfunction in sepsis

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Sir: In their recent review on cardiac dys-
function associated with sepsis, Grocott-
Masson and Shah [1] present as a new the-
ory the concept developed by Parillo on
cardiac dysfunction in sepsis [2]. According
to this concept, cardiac enlargement occurs
during septic shock, and such an acute dilu-
tation allows survival by maintaining an
degate stroke output despite severely
depressed left ventricular ejection fraction
(LVEF). This acute adaptation is similar to
the chronic adaptation which is observed
in congestive heart failure and has been
known for a long time [3]. However, a surge
of echocardiographic data obtained since
then does not support this hypothesis [4-6].

Parillo's hypothesis mainly relies upon
measurements of LV dimensions using an
indirect method in 20 patients with septic
shock [7]. This indirect method combined
measurements of cardiac output by the
thermodilution method, and of LVEF by
radioisotope angiography to calculate LV
volumes. As a general rule, calculations
derived from the combination of two dif-
ferent methods, each with its own inaccu-
acy, is methodologically questionable. In
low-flow states, the thermodilution techni-
que overestimates cardiac output [8, 9]. A
recent experimental study showed that
overestimation of cardiac output could be
as high as 80 % when compared with the
dilution method, the gold standard for
indicator dilution techniques [10]. Thus,
the marked LV enlargement reported by
Parker et al. [7] might well be explained by
an overestimated cardiac output, which was
very likely since survivors had a greater
LVEF depression [7]. Thus, an artifactual
overestimation of LV end-diastolic volume
would be more frequent in this group, as il-
lustrated by Parker et al.'s results [7]. Ad-
ditionally, calculations performed in this
study appeared somewhat erroneous, as we
have previously noted [11]. Later, the same
indirect method applied to the right ventri-
cle by these authors ended in the descrip-
tion of acute biventricular dilatation during
septic shock [12], a concept inconsistent
with a close pericardium.

In a study to be published, using daily
two-dimensional echocardiography, we
measured LV dimensons in 90 successive
patients hospitalized for an episode of sep-
tic shock. Despite fluid administration of 4
to 5 liters per day, we observed that LV re-
main unchanged: mean end-diastolic
volume was 69 ± 24 cm³/m² at day 1, 68 ± 23
day 2, 70 ± 21 at day 3, and 66 ± 23 at
day 5 (normal value for our laboratory:
69 ± 15 cm³/m²). In these patients with se-
verely depressed LV ejection fraction
(47 ± 16 % at day 1, normal value:
69 ± 6 %), cardiac output could be main-
tained only through increased heart rate,
and circulatory status improvement, when
it occurred, resulted from a progressive in-
crease of LVEF (52 ± 15 % at day 5).

In the near future, routine use of bed-
side echocardiography in septic patients
will put matters in their true light by taking
into account two well-established physiolo-
gical principles: the restraining action of
pericardium and the fact that all clinical
settings involving myocardial depression
are associated with slow and incomplete
relaxation, inconsistent with increased
myocardial compliance.

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A. M. Shah
R. M. Grocott-Mason

Reply

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Sir: We thank Drs. Jardin and Bourdarias
for their interest in our recent article [1].
Their letter, however, suggests that, in pur-
suing their long-standing disagreement
with the work of Parillo and colleagues,
they have missed the main point of our ar-
ticle. This was to review potential cellular
mechanisms responsible for the myocardial
depression, which they agree is a feature of
septic shock. Their letter also contains a
number of inaccuracies.

We state that cardiac output is usually
normal or high in patients with septic
shock, mainly due to the presence of tachy-
cardia and a reduced afterload. Many stu-
dies (not just those of Parillo and
colleagues) have reported the occurrence
of acute left ventricular dilatation in septic
shock in both animals and humans, al-
though it may only be revealed by volume
loading [2–7]. We did not present utilisa-
tion of “preload reserve” by the Frank–
Starling mechanism as a “new” mechanism
for the maintenance of cardiac output in
the face of impaired contractility. This is a
basic principle of cardiac adaptation, and
even in the context of septic shock has been
published for nearly a decade [8]. We out-
line the difficulties in clinical assessment of
cardiac inotropic state during septic shock.
The profound changes in both preload and
afterload make interpretation of loud-de-
pendent indices, such as LVEF, impossible
in isolation.

The paper of Parillo’s group on serial
LV function [8] actually includes 54 pa-
tients with septic shock, which is larger
than the patient numbers in either of the quoted
papers by Drs. Jardin and Bourdarias [9,
10]. The cardiac outputs of patients with
septic shock were equal to or higher than in
the control patients, in keeping with the
typical haemodynamic pattern of septic
shock [8]. Thus, the criticisms of the ther-
modilution technique in low-flow states are
unlikely to be relevant.

The role of the pericardial constraint in
acute septic shock is not known. The nor-
mal pericardium is not rigid, but is compli-
ant and will respond to volume-loading by
distending, along with an increase in intra-
cavitary and intrapericardial pressures. The
only condition where this is not true is con-
strictive pericarditis. An intact pericardium
is therefore not incompatible with acute
biventricular dilatation.

We look forward to reading Drs. Jardin
and Bourdarias’ paper on serial echocar-
diographic studies in patients with septic
shock when it is published. They do not
specify their method for measuring LV vol-
umes but all echocardiographic methods
for measurement of LV volume have po-
tential errors and tend to underestimate
true ventricular volumes [11]. Excellent
delineation of the endocardial border is
crucial and this may be extremely difficult
in patients with septic shock on mechanical
ventilators who often have suboptimal
transthoracic windows. Such factors are
likely to add to the inaccuracies of serial
echocardiographic volume measurements.
We are not aware of any data which have
validated serial echocardiographic measure-
ments of LV function in such patients.
In their previous studies they used a single-
plane area–length method to calculate LV
volumes [9, 10]. Even in non-ventilated pa-
tients the correlation between LV volume
measured by an area–length method and
other methods is, at best, fair, and single-
plane is inferior to the biplane method [11].

The final comments by Drs. Jardin
and Bourdarias suggest an incomplete under-
standing of the factors that influence ven-
tricular diastolic properties. The left
ventricular diastolic pressure–volume rela-
tion may be influenced by several factors,
including myocardial viscoelasticity, the
time-course and extent of myocardial re-
laxation, ventricular interaction, the peri-
cardium, diastolic suction and coronary
vascular engorgement [12, 13]. The rate of