To the Editor:

We have a follow-up report from Dr. Kerkay, Professor of Biochemistry at Cleveland State University, who has been working with his group in conjunction with our clinical studies on polyvinyl chloride. He was co-author of the paper recently published in Chest. His comments follow:

"Dr. Stuart M. Brooks’ criticism of the first patient’s results and biochemical data is out of context. When he refers to published data (his references 2 and 6) of PVC breakdown products, he cites the thermal decomposition of bulk PVC rather than the plasticized final product. His reference 6 confirms our findings qualitatively (p 383 and 390) where they report the boiling off of phthalate plasticizers during thermal decomposition of certain (plastic E & G) PVC compounds. In his reference 1, Dr. Brooks cites an article by Vandevort and co-workers, which was published in 1977 and deals with the thermal decomposition of meat packaging films. There have been changes and improvements in the plasticization of meat wrapping films during the past 4 years. Our laboratory does have GC and HPLC tracings of the patient’s blood and urine indicating the presence of phthalate and not adipec plasticizers, as confirmed by mass spectroscopy. It is true that many commercial products contain phthalate plasticizers; however, these plasticizers are leached out of plastics only if the plastic comes in direct contact with blood (or lipid containing solutions) or when inhaled following volatilization.2 We have tested blood and urine specimens from over 100 normal individuals and not one of them showed the presence of phthalate or adipate plasticizers in greater amounts than the detection limit of our method of 40 ng/ml serum or urine.3 Therefore, we assume that the normal phthalate level is less than 40 ng/ml, which is undetectable.

“Our findings of DEHP, MEHP, phthalic acid, and phthalic anhydride in this patient resulted from the exposure to thermal degradation products of the plasticized PVC and from the metabolism of these phthalate plasticizers. The cytologic examination of lung tissues from the three cases presented in our paper are very similar to the changes observed in lung tissues of dogs following intravenous injection of chemically pure bis (2-ethylhexyl) phthalate (DEHP).”

Now in conjunction with the special vinyl chloride paint composition that one patient had been exposed to, this has been very difficult to follow-up because the dental equipment company, known as Weber of Canton, Ohio, has totally closed down, and I am in the process and have almost completed the process of finding additional information through the major dental equipment company in New Jersey. They have referred me to the dental school here and also through Dr. Carl Zenz of Milwaukee, Wisconsin. A special paint which is identical to the chemical B, is now being analyzed for total and specific contents.

The third case delineated the PVC occupational atmospheric level at 200-300 parts per million, which is what we had obtained from the industrial safety director of a specific plant. Our chemist co-author discussed this in detail with the Research Chemist Supervisor of B.F. Goodrich Company of Akron, Ohio (new plasticizer division), who stated that this pollutant can be reported in micrograms per cubic meter or in parts per million. We are fully aware of the fact that as a particular, this is appropriately reported in micrograms or milligrams per cubic feet of air. Therefore, we were in error as the occupational atmospheric reading should have been reported as micrograms per cubic feet.

We have seen many patients, in consultation with two chemical plants in New York State, with vinyl chloride and polyvinyl chloride exposures.4 We believe that the patient reported represented a case of subacute interstitial pneumonitis and not de-facto pneumocnosis.

If one reads the article carefully, on page 833, paragraph 2, we alluded to the fact that the relationship is highly presumptive and not conclusive as to cause-effect relationship in the development of interstitial fibrosing pneumonitis. We cautioned in our conclusions that the total clinical significance of the factors referable to the biochemical changes were still unknown and will be pursued further.

Consequently, we have felt that the published paper has left some unknown answers and in no way have implicated these agents to be direct causative factors of pneumoconioses in any of these three patients. We do feel that these histopathologic changes are probably related to the excessive exposures of the VC/PVC group of agents. Therefore, we disagree with Dr. Brooks that the authors have “fallen into the common trap of attributing occupational disease to be present because of potential toxic material in the work place.”

Edward M. Cordasco, M.D., F.C.C.P.,
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Cleveland Clinic, Cleveland

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CHEST, 81: 2, FEBRUARY, 1982

COMMUNICATIONS TO THE EDITOR 263
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ADDENDUM

The conclusion after analyzing the paint specimen and
the control paint is: (A) the paint specimen used in the
dental tools has a higher plasticizer than the control paint
(DuPont® Lucite); (B) from the toxicity studies on the
plasticizer1,2 we assume the chronic condition caused in
this patient may be due to plasticizer and not PFC or mono-
vinylchloride; (C) we have to emphasize “there is a gross
difference between composition of the plasticizer of the
paint used in the dental equipment and the paint used as
the control.”

*Sample of dental equipment paint obtained from Case
Western Reserve University Dental School.

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Improperly Assembled Expiratory Flow
Tube/Sensor

To the Editor:

The incident reported by Dr. S. Carson and Mr. P.
Holl in the March, 1981 issue of Chest is a fairly com-
mon problem. I would like to share this additional infor-
mation with your readers, if I may.

When the expiratory flow tube/sensor is improperly as-
sembled and connected to the Bourns Bear respirator it
causes a total obstruction of the tidal volume that is to
be delivered to the patient and this results in the follow-
ing: 1) the pressure alarm should activate since peak pressure
is reached due to this obstruction; 2) the low volume alarm
should activate since there is no flow through the flow
sensor. This also causes volume read out to read zero.

These are fairly common occurrences with any respira-
tor and are indicative of physiologic obstruction such as the
patient trying to cough or “fighting” the machine. If no
obvious effort is noted on the patient’s part, the attention
should be diverted and focused on the mechanical aspect.

Shashi M. Dani, C.R.R.T.,
Director, Respiratory and Pulmonary Department,
Helene Fluid Medical Center, Trenton

To the Editor:

Mr. Dani’s comments regarding the incident which oc-
curred in our ICU and his assertion that similar accidents
occur frequently in other ICUs are both appreciated.

Although we agree with Mr. Dani that expiratory ventila-
tor flow obstruction will produce a zero expired volume
read-out and will activate the low exhale volume and pres-
sure limit alarms, we disagree with his contention that
mechanical malfunction can be suspected by the absence
of respiratory effort on the part of the patient. As described
in our report, the patient was extremely agitated during
the period of ventilator malfunction and was making active
(but ineffectual) respiratory efforts until she lost conscious-
ness.

We wish to emphasize that the purpose of our letter was
to alert readers to one of the many potentially fatal hazards
which exist in ICUs which can be prevented. If the equip-
ment modifications which we described are utilized, acci-
dental misassembly of the Bourns Bear I external flow
transducer producing an expiratory obstruction can be
totally prevented in the future.

Sanford A. Carson, M.D.,
Pulmonary Disease Section;
A. Peter Holm, R.R.T., M.A.,
City of Hope National Medical Center,
Duarte, CA

Evulsion Biopsy of Pleura Concomitant with
Insertion of Closed Thoracostomy Tube

To the Editor:

Needle biopsy of the pleura at the time of thoracocentesis
is an acceptable procedure for pleuropneumonic diseases
causing pleura effusion. In addition to the risk of causing
pneumothorax, the concave configuration of the pleural
cavity does not lend itself to a satisfactory pleural biopsy
in many cases. Since most massive, as well as intractable
pleural effusions do require a closed thoracostomy chest
tube, it is logical to obtain an adequate piece of parietal
pleura for histologic and bacteriologic studies at the time
of tube insertion.

I have utilized the concomitant evulsion biopsy of pleura
at the time of closed thoracostomy tube for the past year
and find this combination to be safe and productive.

TECHNIQUE

1. With the patient lying on the normal side, the lateral
chest wall is prepped and draped. Appropriate level of
insertion of chest tube is selected and a small skin in-
cision is made under local anesthesia.

2. An oblique track is made by gentle dissection with a
curved hemostat from the skin incision leading to the
pleural cavity.

3. Once the pleural cavity is entered, the hemostats are
rotated in such a way that the tip of the hemostat comes
in contact with the parietal pleura of the chest wall.

4. The hemostat is open and the pleura is grasped.

5. With a quick withdrawing motion, a piece of pleura is
evulsed. The biopsy specimen is submitted for appro-
riate studies.

6. Through the same track the chest tube is inserted and
anchored to the skin with appropriate nonabsorbable
suture material.

Multiple biopsies of the pleura can be done with this
 technique. The size of the biopsy specimen is considerably
larger than can be retrieved with pleural biopsy needle.
The procedure does not add any discomfort, and to date,
no untoward complication has been encountered in 15
cases.

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43616