New Appliances

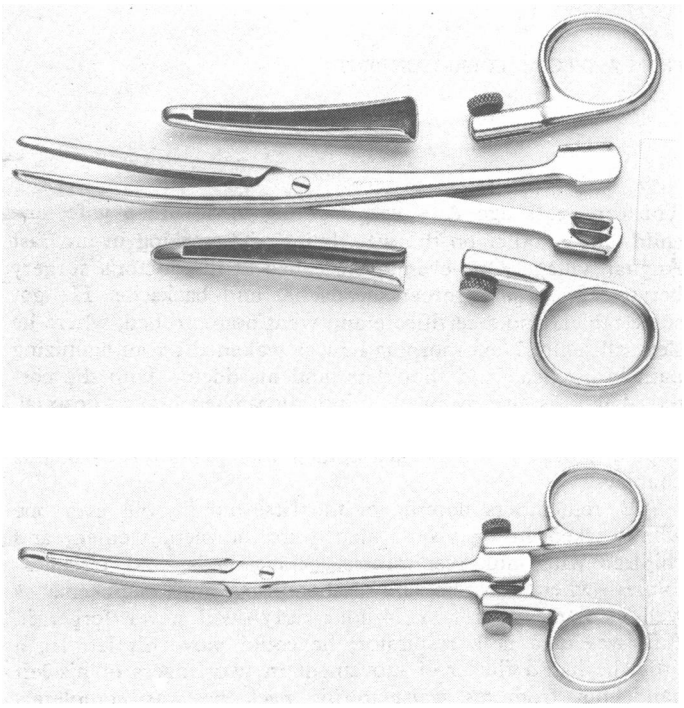
A Stomal Clamp

Mr. J. S. P. LUMLEY, lecturer in surgery, Mr. T. C. CRICHTON, of the department of physics, and Mr. J. DUCKIT, curator of instruments, St. Bartholomew's Hospital, London E.C.1, write: In fashioning a terminal colostomy or ileostomy the passage of the gut through the abdominal wall may precipitate faecal soiling of the peritoneal cavity or of the abdominal wall itself. Commonly this manoeuvre is achieved by passing a bowel clamp through the prepared stoma and applying it to the end of the bowel, a previously applied clamp requiring removal or a further segment of bowel requiring resection. Alternatively, a Cope's, or similar, clamp may be applied. This can be passed through the abdominal wall but cannot be easily sealed, and its application may be difficult at the exact site or angle required.

The clamp described below is a crushing clamp with removable handles, which can thus be drawn through the abdominal wall without reapplication to the bowel (see Figs.). The ribbed blades of the clamp are 6·5 cm long and have an accompanying pair of Parker-Kerr-type guards. The length of the shaft is 10 cm, this being short enough to allow passage through the abdominal wall without putting tension on a bowel mesentery. The disarticulated ends of the shaft are rounded to facilitate passage through the stoma.

The handles add length and purchase to the shaft, on to which they are tee slotted and fixed with milled screws. Each screw end is captive to prevent removal or loss during an operation. The clamp has proved comfortable to handle and has greatly facilitated this procedure.

The clamp is available commercially from the Seward Instrument Company, 6 Stamford Street, London S.E.1.



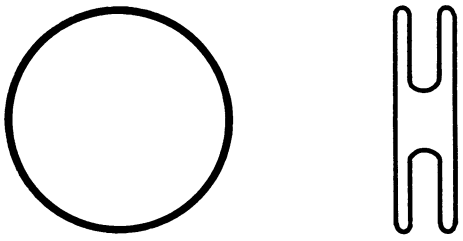
New Method of Femoral Hernia Repair Using a Silastic Stud

Mr. A. J. S. KNOX, surgical registrar, and Mr. K. P. S. CALDWELL, senior surgeon, Royal Devon and Exeter Hospital, Exeter, write: Femoral hernia is not uncommon (Koontz, 1958). Though Aird (1960) dismissed the possibility of recurrence after operation, others have reported recurrence rates of from 2% (Glassow 1966) to 10% (Koontz, 1958). Treatment of the hernia consists in excision of the sac, and, since this operation alone is followed by a high recurrence rate (McEvedy 1950), it is usually combined with some procedure to close the canal.

The operation is often done by relatively inexperienced surgeons, and though many methods have been described the placing of sutures in this area is not easy. Furthermore, since the femoral ring is bounded on three sides by the rigid inguinal, pectineal, and lacunar ligaments and on the fourth side by the femoral vein, it would be logical to occlude the canal with a plug rather than attempt to close it with sutures.

In 1959 Caldwell described a "collar-stud" operation in which the femoral canal was filled with a plug of polyvinyl sponge. The canal had to be exposed from above and below, however, and preparation of the sponge was tiresome. At this time no other suitable material was available, but in 1963 Silastic was developed (Dow Corning Corporation, 1963). Since then Silastic has been used successfully as replacement finger-joints (Swanson, 1968), mammary prostheses (Freeman, 1967), and implants (Caldwell *et al.*, 1968). A simple method of femoral hernia repair using a Silastic stud is described here. It overcomes the disadvantages of the polyvinyl sponge operation while retaining the advantages.

The stud (see Fig.) is made of Silastic (medical grade Silastic 382 Elastomer), which is well tolerated in the body, causes



no irritation to the tissues, and is highly resistant to the effects of body fluids, ageing, and oxidation (Dow Corning Corporation, 1963). Medical Silastic 382 Elastomer is supplied as a thick but easily pourable liquid. When a catalyst (catalyst M) is added the fluid sets to become a silicone rubber in about 10 minutes at room temperature. The stud is easily made by pouring the Silastic fluid mixed with the catalyst into a mould and allowing it to set. We use a Perspex mould as this needs no release factor once the Silastic has

dried. A supply of studs, which may be sterilized in the autoclave, is kept in the theatre.

The initial steps of the operation need no description. Any approach which gives access to the canal from below may be used. Once the sac has been excised the stud, which plugs the femoral canal, is readily inserted from below, so that the flanges lie snugly on each side of the margins of the hernial orifice. Occasionally the stud has been fixed with a suture, but usually this has not been necessary.

Fifteen hernias have been repaired with the use of the stud, and there have been no recurrences. Though gangrenous bowel has been resected on 12 occasions and wound infection has occurred once, there have been no cases of persistent infection, sinus formation, or rejection of the stud. Fifteen patients have now been treated and the maximum period of follow-up is 36 months with no untoward effects. Occasionally the method has been abandoned when the hernial orifice was to large to hold the stud properly, but this has been an infrequent problem, and the stud has usually been easy to insert and has remained firmly in position. Strangulated hernia is not a contraindication to the use of the stud.

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Caldwell, K. P. S., Cook, P. J., Flack, F. C., and James, D. (1968). *British Journal of Urology*, **40**, 183.
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Any Questions?

We publish below a selection of questions and answers of general interest

Oxygen Dependence

Can a physical dependence on oxygen develop with frequent use in patients with, say, heart failure, and how can apparently unreasonable demands for oxygen be differentiated from genuine need?

A patient in extreme pulmonary failure may become physically dependent on oxygen in the sense that he would die without it,[1] but true addiction does not occur. However, once a patient with chronic heart or lung disease has experienced the benefits of oxygen he may become less willing to tolerate the discomforts of hypoxia. A patient can thus acquire psychological dependence on oxygen and use amounts far in excess of his physical needs.

The need for oxygen in patients with an *acute* cardiac or pulmonary illness (e.g., left ventricular failure or pneumonia) can usually be judged by simple clinical signs such as rapid, laboured breathing, tachycardia, and cyanosis. However, significant hypoxia can occur without cyanosis, and if in doubt it is safer to give oxygen in these acute situations than to withhold it. On the other hand, cyanosis due to certain *chronic* diseases is not necessarily an indication for oxygen therapy which may be useless in some instance (e.g., right to left intracardiac shunt) and positively harmful in others (bronchitic respiratory failure).

In general, domiciliary oxygen should be prescribed only for patients with chronic heart or lung disorders when it is otherwise difficult for them to perform such essential activities as defaecation, dressing, or moving about in bed. Portable oxygen equipment may be helpful in a few cases and permit a wider range of activities.[2] When the genuineness of the patient's need is in doubt he should be referred for a full assessment of cardiac and respiratory function at rest, on exercise, and during oxygen breathing. The relevant measurements include arterial oxygen and carbon dioxide tensions, alveolar ventilation, gas exchange, and cardiac output. Relatively simple bloodless methods for making these measurements are now available.[3]

Criteria for the selection of patients for long-term oxygen therapy have been provided by Cotes.[2] An accurate assessment of oxygen need is of particular importance in respiratory failure due to chronic bronchitis, since too much oxygen can induce CO_2-narcosis by abolishing the central hypoxic drive while too little may allow irreversible changes in vital organs.

[1] Comroe, J. H. *et al.*, *The Lung*, 2nd., edn. p. 309. Chicago Year Book Medical Publishers, 1962.
[2] Cotes, J. E., *Lung Function: Assessment and Application in Medicine*, 2nd edn. Oxford. Blackwell Scientific, 1968.
[3] Jones, N. L., *British Journal of Diseases of the Chest*, 1967, **61**, 169.

Laevadosin in Muscular Dystrophy

Has Laevadosin been found to be of value in treating muscular dystrophy? How does it act?

Laevadosin is a nucleotide nucleoside mixture which was considered to cause some improvement in muscular dystrophy as judged by increase in muscle power and by diminution in the activity of serum aldalase and transaminases.[1] A double-blind control trial in patients with the Duchenne type of muscular dystrophy carried out at Newcastle[2] did not show any observable benefits when compared with a placebo, and this has been confirmed elsewhere.[3]

[1] Thomson, W. H. S., and Guest, K. E., *Journal of Neurology, Neurosurgery and Psychiatry*, 1963, **26**, 111.
[2] Pearce, J. M. S., *et al.*, *British Medical Journal*, 1964, **2**, 915.
[3] Gordon, N. S., Epstein, B., Dixon, K., Forrester, R. M., and White, L., in *Research in Muscular Dystrophy: Proceedings of the Third Symposium*, 1965, p. 24. London, Pitman Medical, 1965.

U.T.P. and A.T.P.

Has any work been done on increasing the stability of uridine-5-triphosphoric acid (U.T.P.) and adenosine-5'-triphosphate (A.T.P.) in therapeutic mixtures?

There appears to be no published work on this subject. Uridine-5-triphosphoric acid (Uteplex) is available for oral administration in ampoules each containing 2 mg in 2 ml. Adenosine-5'-triphosphate (A.T.P. Adenotriphos) is very soluble in water and is said to be unstable in alkaline solution. It is available as 3 mg tablets of the disodium salt and as ampoules of 2 ml each containing 20 mg as the neutral sodium salt.[1]

Both U.T.P. and A.T.P. are present in Laevadosin, which is available as tablets and as an injection for intravenous infusion. Since both U.T.P. and A.T.P. are on the market in solution they must be reasonably stable, but the commercial solutions may, of course, be stabilized.

[1] *Extra Pharmacopoeia*, Martindale, 25th edn. London, Pharmaceutical Press, 1967.