Letters to the Editor

Preference is given to letters commenting on contributions published recently in the JRSM. *They should not exceed* **300 words** *and should be* **typed double-spaced.**

Adenocarcinoma of the gallbladder

We read with interest the 5-year review of outcome by Burgess *et al.* (February 1991 *JRSM*, p 84). The authors remind us of the dismal results of treatment of this uncommon disease. Publications concerning this neoplasm are few and it is likely that surgeons confronted with such a case would look to the above article for management guidance.

The authors state 'There is probably a place for routine adjuvant local radiotherapy in addition to surgery'. The statement is referenced but unfortunately, perhaps due to a typographical error, it is not printed at the end of the article with the other citations.

We do not think that routine postoperative radiotherapy for invasive or in-situ carcinoma of the gallbladder represents a statement of the conventional wisdom within the radiotherapeutic community. It would be useful if the authors could expand upon their statement and produce the missing reference.

J J BOLGER
S RAMAKRISHNAN
Department of Radiotherapy and Oncology
Weston Park Hospital
Whitham Road
Sheffield S10 2SJ

In their discussion, Burgess *et al.* (February 1991 *JRSM*, p 84) state that only premalignant in situ lesions can be treated by cholecystectomy alone. They quote an earlier report recommending radical resection in such cases where possible, including wedge resection of the gallbladder bed and portal vein reconstruction when dissection is necessary at the porta hepatis. Such further procedures are presumably carried out at a second operation as premalignant changes (other than adenoma) would be detected only on histological examination of the cholecystectomy specimen.

These particular radical procedures, however, do seem to represent 'overkill' for a non-invasive lesion. Theoretically subsequent carcinoma is much more likely to arise, not in the gallbladder bed, but in other in situ lesions remaining, or subsequently developing, in the hepatic or common bile ducts. But even this risk seems to be small. Areas of dysplasia are not infrequently found in cholecystectomy specimens but subsequent development of duct carcinoma appears not to be a cause for concern. In answer to my request for information about the incidence of such a sequel, Odeja *et al.*[1] did not refer to any such cases; follow-up of their own cases, however, had been short. They indicated that they did not recommend further surgery or investigation for these patients.

H G PENMAN
Crawley Hospital
West Green Drive
Crawley RH11 7DH

References

1 Ojeda VJ, Shilkin KB, Walters M. N-1 Premalignant epithelial lesions of the gall-bladder. *Pathology* 1987;**19**:103

We read with interest the article by Burgess *et al.* (February 1991 *JRSM*, p 84). We have recently reviewed 96 similar cases presenting in Wessex over the period 1982-1989[1]. We were able to confirm the poor prognosis of this tumour, with only three survivors beyond 2 years. We would argue that more aggressive treatment of established tumours, rather than the detection and removal of pre-malignant lesions, can most realistically be expected to improve this outlook. Even patients with intramucosal disease have a 5 year survival of only 64% following simple cholecystectomy, whilst aggressive surgery has yielded prolonged survival even in advanced cases[2]. We have proposed a modification to the staging system of Nevin[3], and would consider extended cholecystectomy for all patients with tumour limited to the gallbladder or directly invading other organs (stage I-III) and in selected cases with nodal spread (stage IV).

We feel that it will be necessary to confirm the relatively poor prognosis for 'early' disease treated by simple cholecystectomy before such an aggressive approach is likely to be widely embraced. Given the inherent inaccuracies in data gathered retrospectively, we have begun a prospective audit of gallbladder cancer in Wessex to resolve this and other issues.

N J CARTY
C D JOHNSON
University Surgical Unit
Southampton General Hospital,
Tremona Road, SO1 6HU

References

1 Carty NJ, Johnson CD. Carcinoma of the gallbladder: a survey of cases in Wessex 1982-1989. *J R Coll Surg Edinb* 1991; in press
2 Bergdahl L. Gallbladder carcinoma first diagnosed at microscopic examination of gallbladders removed for presumed benign disease. *Ann Surg* 1980;**191**:18-22
3 Nevin JE, Moran TJ, Kay R, King R. Carcinoma of the gallbladder: staging, treatment, and prognosis. *Cancer* 1976;**37**:141-8

Why do people have accidents?

With respect to 'Accident Prevention', (PAB Raffle, November 1990 *JRSM*, p 679) it is possible to state something which is a glimpse of the blindingly obvious, that accidents are always the result of human error. Whether that of the designer of the aircraft, the meteorologist, the operator, the pilot etc. In the experimental period, procedures are developed which ensure that with an acceptable level of performance (p in Figure 1) accidents will have an acceptable level of rarity, so that people will be happy to obey the rules imposed upon them. The Yerkes-Dodson Law

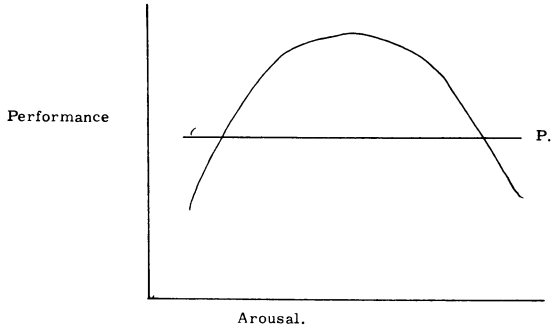

Figure 1. The curve of the Yerkes-Dodson Law for a task of average ability. Note that the performance falls below the acceptable level of safety when the level of arousal is high or low

propounded in 1907 but immediately forgotten, states that there is an optimum level of arousal (the important part) which is reduced as the level of difficulty is raised. Thus we cheer on oarsmen engaged in their mindless labour but remain silent at chess matches. The result is that there are two different kinds of human error leading to an accident, those corresponding to a level of arousal that is too low and too high respectively, and progress in accident prevention is unlikely until this distinction is made, since increasing the level of arousal to reduce the amount of one kind may increase the amount of the other. Progress in nephrology was not made until it was appreciated that there were two types of nephritis, the same principle applies in accident prevention.

BRENNIG JAMES

Cherry Orchard
Marlow Common
Bucks SL7 2QP

Gastrointestinal complications of haemolytic uraemic syndrome

We concur with Crabbe and colleagues (December 1990 *JRSM*, p 773) that gastrointestinal complications are potentially serious problems in children with haemolytic uraemic syndrome (HUS). We recently reported our experience with gastrointestinal complications in 76 children with typical HUS[1]. Table 1 shows the incidence of the gastrointestinal complications we reported.

Table 1. The incidence of gastrointestinal complications in 76 children with typical HUS

Gastrointestinal complication	*Children studied*	*Children with complications*	
Haemorrhagic colitis	76	60	78.9%
Pancreatitis	29	6	20.7%
Rectal prolapse	76	10	13.1%
Diabetes mellitus	76	2	2.6%
Intestinal stricture	76	2	2.6%
Intestinal perforation	76	1	1.3%
Intussusception	76	1	1.3%

Many children with typical HUS have peritoneal signs. We routinely use peritoneal dialysis in children with oligo-anuric HUS and have not experienced the same frequency of technical problems reported by Crabbe and colleagues. We recommend the operative placement of a Tenckhoff catheter with omentectomy rather than the use of a percutaneous catheter. During the placement of the catheter, the urologist is able to visualize and assess the colon. Thereafter, the peritoneal dialysate can serve as a 'window' into the peritoneal cavity. Perforation may be evident by the presence of colonic contents in the dialysate. The blood that is present in the dialysate, subsequent to the catheter placement, usually clears within 24 h after the insertion of the catheter. In contrast to Crabbe and colleagues, we find that the dialysate white blood count is a reliable sign of peritonitis.

We do not agree that colonic resection, to remove the source of the verotoxin, is a logical step. There is evidence that the verotoxin is rapidly bound to Gb3 receptors on endothelial cells[2]. If the receptors are saturated early in the HUS process, removal of the source of the verotoxin would not be expected to improve the situation.

We agree with Crabbe and colleagues that H_2 receptor antagonists are helpful to minimize the risk of upper gastrointestinal bleeding. We prescribe these medications prophylactically to all children with severe HUS.

WM LANE M ROBSON
ALEXANDER K C LEUNG

Department of Pediatrics
The University of Calgary and
The Alberta Children's Hospital
1820 Richmond Road SW
Calgary, Alberta T2T 5C7
Canada

References

1 Grodinsky S, Telmesani A, Robson WLM, *et al.* Gastrointestinal manifestations of hemolytic uremic syndrome: recognition of pancreatitis. *J Pediatr Gastroenterol Nutr* 1990;11:518-24

2 Richardson SE, Petric M, Karmale MA. Uptake of 125 I-labeled verotoxin into rabbits. (Abstract) *Program Annu Meet Am Soc Microbiol* 1988;**B35**:35

Peripheral parenteral nutrition

We agree with Khawaja *et al.* that total parenteral nutrition (TPN) via the peripheral route (PPN) is a safer alternative than that via a central venous cannula (CVC) (February 1991 *JRSM*, p 69). It is worth emphasizing that the majority of TPN-related complications are CVC-related. Current first-line therapy in this unit for patients with intestinal failure needing TPN is peripheral administration of a within-hospital compounded formulation (690 mosmol) providing 1500 cal (glucose - 500 kcal/lipid - 1000 kcal), 12 g nitrogen with electrolytes and trace elements (Elamin 10%, Glucoplex 1000, Elolipid 20%; Oxford Nutrition Systems Ltd, Oxford OX4 3UH). Glyceryl trinitrate patches are used routinely. If nutritional support is needed for >10 days then conversion to central venous TPN is undertaken. Eighty-four per cent of patients receive TPN for <14 days in the UK, with 27% having TPN for <7 days[1]. Thus if incidences of peripheral vein thrombophlebitis can be reduced or onset delayed, many patients may not need CVCs. Sixty per cent of units in the UK use PPN[1], although this represents only 7% of TPN regimens administered. The advantages of PPN have been recognized in paediatric units for many years. Recently, a number of units in the UK (in addition to those referred to by Khawaja) have studied PPN. Intermittent PPN with alternation of infusion sites is a safe method of delivery[2]. Ultrafine-bore cannulas reduce thrombophlebitis[3,4]. We have examined the influence of cannula material[5]. The effects of heparin/hydrocortisone in the infusate, and osmolality on thrombophlebitis have also been reported[6]. The relevance of these studies is that more clinicians recognize the reductions in morbidity that increased use of PPN could bring. Additionally these studies are of importance to all hospital patients with peripheral venous cannulas *in situ* of whom at least 10% will develop thrombophlebitis[7]. Six million peripheral venous cannulas or more are sited in the UK annually so methods of reducing thrombophlebitis may be relevant to a large part of the hospital population.

J PAYNE-JAMES
G GRIMBLE
S KAPADIA
D SILK

Department of Gastroenterology & Nutrition
Central Middlesex Hospital
Acton Lane, London NW10 7NS